Third Month To Birth: The Fetus and Placenta

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Fetal Period (9th Week to Birth)

Definition

- Time frame: Beginning of 9th week → Birth.
- Key features:
  - Maturation of tissues and organs.
  - Rapid growth of body.

Measurement of Fetal Size

- Crown-Rump Length (CRL): Sitting height (vertex → buttocks).
- Crown-Heel Length (CHL): Standing height (vertex → heel).
- Measurements in cm correlate with fetal age in weeks/months.
- Growth pattern:
  - Length: Rapid in 3rd–5th months.
  - Weight: Most rapid in last 2 months.

Pregnancy Duration

- From LNMP: ~280 days (40 weeks).
- From Fertilization: ~266 days (38 weeks).
  (Exam Tip: Age is calculated from fertilization in developmental discussions.)

Monthly Developmental Changes

Third Month (Weeks 9–12)

- Head proportion:
  - Start of month: ~½ CRL
  - End of month: Growth of body accelerates, head growth slows.
- Facial changes:
  - Eyes move from lateral → ventral position.
  - Ears move to definitive position at side of head.
  - Face becomes more human-like.
- Limbs:
  - Reach relative proportions, but lower limbs slightly shorter & less developed than upper

limbs.

- Ossification:
    - Primary ossification centers in long bones & skull appear by week 12.
- External genitalia:
    - Distinct enough to determine sex via ultrasound.
- Gut development:
    - Herniated intestinal loops (present at week 6) retract into abdominal cavity by week 12.
- Reflex activity:
    - Present in aborted fetuses — shows muscle activity.

Fourth & Fifth Months (Weeks 13–20)

- Length: Rapid increase — CRL ~15 cm by end of 5th month (~½ newborn's length).
- Weight: Minimal gain (<500 g).
- Hair:

- Fine hair (lanugo) covers body.
- Eyebrows & scalp hair visible.

- Fetal movements:
  - Felt by mother during 5th month (quickening).

Second Half of Pregnancy (Weeks 21–40)

- Weight gain:
  - Significant; last 2.5 months → 50% of term weight (~3200 g) added.

- Sixth Month:
  - Skin: Red & wrinkled (little connective tissue).
  - Survival difficult if born early — respiratory & CNS not fully coordinated.

- 6.5–7 Months:
  - CRL ~25 cm, weight ~1100 g.
  - Survival chance ~90%.

- Last 2 Months:
  - Fat deposition → rounded contours.

- Skin covered by vernix caseosa (whitish, fatty; sebaceous gland secretions).

End of Ninth Month (Full Term)

- Head: Largest circumference (important for birth canal passage).
- Measurements:
  - Weight: 3000–3400 g.
  - CRL: ~36 cm.
  - CHL: ~50 cm.
- Sexual characteristics: Pronounced; testes should be in scrotum.

Time of Birth & Gestational Age

Precise Timing

- Actual duration from fertilization: 266 days / 38 weeks.
- Obstetric calculation (LNMP): 280 days / 40 weeks from 1st day of Last Normal Menstrual Period (LNMP).
- Fertilization window:

    - Oocyte fertilized within ~12 hours of ovulation.
    - Sperm can survive up to 6 days before ovulation.
    - Most pregnancies occur when intercourse happens within 6 days before ovulation → day of ovulation.

Challenges in Dating Pregnancy

- Patient recall: Fertilization day hard to determine — often seen by doctor after 2 missed periods.
- Implantation bleeding: May occur ~14 days after fertilization → can be mistaken for menstruation.
- Cycle irregularity: Can lead to major miscalculations.
- Normal delivery window: Within ±10–14 days of calculated date.
- Premature: Born before 37 weeks.
- Postmature: Born significantly after calculated date.

Fetal Age Determination Methods

- Early pregnancy (7th–14th week):
    - CRL (Crown-Rump Length) via ultrasound →

accuracy: ±1–2 days.

- Mid–late pregnancy (16th–30th week):
    - Biparietal diameter (BPD).
    - Head circumference.
    - Abdominal circumference.
    - Femur length.
- Importance: Helps plan delivery, detect growth restrictions, anticipate complications (e.g., small pelvis, birth defects).

Clinical Correlates: Low Birth Weight & IUGR

Normal Birth Size

- Weight: 2,500–4,000 g (average ~3,200 g).
- Length: ~51 cm (20 in).

Low Birth Weight (LBW)

- Definition: Weight < 2,500 g, regardless of gestational age.
- Main causes:
    - Preterm birth (<37 weeks).

- Intrauterine Growth Restriction (IUGR).

Intrauterine Growth Restriction (IUGR)

- Definition: Failure to reach optimal intrauterine growth → pathologically small.

- Small for Gestational Age (SGA):

  - Birth weight < 10th percentile for gestational age.
  - May be pathologic (IUGR) or constitutional (healthy small).
  - Important to differentiate — to avoid unnecessary high-risk interventions.

Epidemiology & Risks

- Incidence: ~10% of newborns.

- Risks:

  - Neurological problems.
  - Congenital malformations.
  - Meconium aspiration.
  - Hypoglycemia, hypocalcemia.
  - Respiratory distress syndrome (RDS).

- Long-term risks (Barker's Hypothesis):

    - Adult obesity.
    - Hypertension.
    - Hypercholesterolemia.
    - Cardiovascular disease.
    - Type 2 diabetes.

Causes of IUGR

- Genetic: Chromosomal abnormalities.
- Maternal:
    - Poor health (hypertension, renal, cardiac disease).
    - Malnutrition, low socioeconomic status.
    - Smoking, alcohol, drug use.
- Infections: Rubella, CMV, toxoplasmosis, syphilis.
- Placental: Placental insufficiency.
- Multiple births: Twins, triplets.
- Environmental teratogens.
- Ethnicity: Higher in blacks than whites.

Growth-Promoting Factors

Prenatal Growth

- Insulin-Like Growth Factor I (IGF-I):
    - Mitogenic + anabolic effects.
    - Expressed by fetal tissues; serum levels correlate with growth.
    - IGF-I gene mutations → IUGR persisting after birth.

Postnatal Growth

- Growth Hormone (GH):
    - Acts via GH receptor (GHR) → stimulates IGF-I synthesis.
    - GHR mutations → Laron's Dwarfism:
        - Marked short stature.
        - Sometimes blue sclera.
        - Little/no IUGR (because fetal growth does not depend on GH).

Fetal Membranes & Placenta – Trophoblast Changes

Placental Origin

- Fetal component:
    - Trophoblast + extraembryonic mesoderm → form chorionic plate.

- Maternal component:
    - Uterine endometrium (decidua basalis).

Development by Beginning of 2nd Month

- Trophoblast features:
    - Numerous secondary and tertiary villi → radial appearance.
- Stem (anchoring) villi:
    - Extend from chorionic plate mesoderm → cytotrophoblast shell.
- Villous structure:
    - Outer layer: Syncytiotrophoblast.
    - Beneath: Cytotrophoblast layer.
    - Core: Vascular mesoderm (with developing capillaries).

Extraembryonic Vascular System Formation

1. Capillaries form inside villous core.
2. Connect with capillaries in chorionic plate and connecting stalk.

3. Together form extraembryonic vascular system — links fetus to placenta.

Maternal Blood Supply to Placenta

- Source: Uterine spiral arteries.
- Mechanism of blood entry into intervillous spaces:
    - Endovascular invasion by cytotrophoblast cells.
    - Origin: Ends of anchoring villi.
- Process:
    1. Cytotrophoblasts replace maternal endothelial cells in spiral arteries.
    2. Undergo epithelial-to-endothelial transition.
    3. Spiral arteries transform:
        - Small-diameter, high-resistance → large-diameter, low-resistance.
        - Increases maternal blood flow to intervillous spaces.

Villous Maturation

- 3rd-4th Month:

- Small free villi grow from stem villi into intervillous spaces.
- Initially primitive: multiple layers (syncytium, cytotrophoblast, connective tissue, endothelium).

- By early 4th Month:

  - Cytotrophoblast cells & some connective tissue cells disappear from most villi.
  - Exchange barrier reduced to:
    1. Syncytiotrophoblast
    2. Endothelial wall of fetal capillaries
  - Barrier becomes thinner → more efficient exchange.

Syncytial Knots

- Definition: Large pieces of syncytium (with multiple nuclei) that break off into maternal intervillous blood.

- Fate: Enter maternal circulation → degenerate without symptoms.

- Importance: Sign of normal villous turnover.

Cytotrophoblast Regression

- Progresses from smaller villi → larger villi.

- Some cytotrophoblast always persists in large villi (structural support), but not involved in exchange.

Clinical Correlate – Preeclampsia

Definition

- Disorder of pregnancy characterized by:
    - Maternal hypertension
    - Proteinuria
    - Due to reduced organ perfusion.

Epidemiology

- Occurs in ~5% of pregnancies.

- Can progress to eclampsia (preeclampsia + seizures).

- Leading cause of maternal mortality in USA.

Onset & Risks

- Sudden onset: 20 weeks gestation → term.

- Risk factors:

1. Previous preeclampsia
2. Nulliparity (first pregnancy)
3. Obesity
4. Family history
5. Multiple gestation (twins or more)
6. Medical conditions (HTN, diabetes)
7. Hydatidiform mole → tends to occur early in pregnancy

Consequences

- Fetal growth restriction.
- Fetal death.
- Maternal death.

Pathophysiology (Key Exam Point)

- Probable trophoblastic disorder.
- Cytotrophoblast cells fail to undergo normal epithelial-to-endothelial transformation.
- Result:
    - Poor invasion of maternal spiral arteries.
    - Arteries remain high-resistance, narrow-diameter.

    - Reduced uteroplacental blood flow.

- Exact mechanism linking to HTN & proteinuria still unclear.

Treatment

- Only definitive cure = Delivery of baby.

- Challenge: Balancing maternal safety with fetal maturity to avoid complications of preterm birth.

Exam Tip:

Preeclampsia vs Eclampsia –

- Preeclampsia = HTN + proteinuria

- Eclampsia = Preeclampsia + seizures

Chorion Frondosum & Decidua Basalis

Early Chorion (First Weeks)

- Villi cover entire chorionic surface.

Later Development

- Embryonic pole:

- Villi persist, branch, and expand → Chorion frondosum ("bushy chorion").

- Abembryonic pole:
  - Villi degenerate → smooth surface = Chorion laeve.

Decidua (Functional Endometrium in Pregnancy)

- Decidua basalis:
  - Located over chorion frondosum.
  - Decidual plate: Compact layer of large decidual cells rich in lipids & glycogen.
  - Firmly attached to chorion.

- Decidua capsularis:
  - Covers abembryonic pole.
  - Becomes stretched & degenerates as pregnancy progresses.

- Decidua parietalis:
  - Lines remainder of uterine wall.

Fusion Events

- Chorion laeve contacts decidua parietalis → fusion → obliteration of uterine lumen.

- Only chorion frondosum + decidua basalis form functional placenta.

- Amnion + chorion fuse → amniochorionic membrane.

    - Obliterates chorionic cavity.
    - Ruptures at labor → "water breaking".

Structure of the Placenta

By Beginning of 4th Month

- Two components:

    1. Fetal portion → Chorion frondosum
    2. Maternal portion → Decidua basalis

- Fetal side: Bordered by chorionic plate.

- Maternal side: Bordered by decidual plate (part of decidua basalis).

- Junctional zone:

    - Trophoblast + decidual cells intermingle.
    - Contains decidual cells + syncytial giant cells.

- Rich in amorphous extracellular material.
- Most cytotrophoblast cells have degenerated by this stage.

Intervillous Spaces

- Located between chorionic plate & decidual plate.
- Filled with maternal blood.
- Origin: Derived from lacunae in syncytiotrophoblast.
- Lining: Syncytium (fetal origin).
- Villous trees extend into maternal blood lakes.

Decidual Septa

- Formed during 4th–5th months.
- Project into intervillous spaces but do not reach chorionic plate → spaces remain interconnected.
- Core: Maternal tissue.
- Surface: Syncytial layer → always separates maternal blood from fetal tissue.
- Function: Divide placenta into 15–20 compartments =

Cotyledons.

Placental Growth

- Enlarges in proportion to uterus.
- Covers 15–30% of internal uterine surface.
- Thickening due to arborization of villi, not deeper invasion into maternal tissue.

Full-Term Placenta

- Shape: Discoid.
- Diameter: 15–25 cm.
- Thickness: ~3 cm.
- Weight: 500–600 g.
- Expulsion: ~30 min after birth (afterbirth).
- Maternal surface:
    - 15–20 cotyledons (bulging areas).
    - Grooves between cotyledons = decidual septa.
- Fetal surface:

- Covered by chorionic plate → amnion externally.
- Chorionic vessels converge toward umbilical cord.
- Cord attachment: Usually eccentric, sometimes marginal, rarely velamentous (insertion into membranes outside placenta).

Circulation of the Placenta

Maternal Circulation

- 80–100 spiral arteries:
  - Pierce decidual plate.
  - Open into intervillous spaces → maternal blood under pressure bathes villi with oxygenated blood.

- Return flow:

  - Blood drains toward decidua → endometrial veins → maternal circulation.

Intervillous Spaces

- Mature placenta → ~150 mL maternal blood at a time.

- Replenished: 3–4× per minute.

- Blood bathes chorionic villi → exchange of gases, nutrients, waste.

Surface Area for Exchange

- Chorionic villi surface area: 4–14 m^2.
- Not all villi participate → only those with fetal vessels in close contact with syncytium.
- Brush border (microvilli) on syncytium → ↑ surface area → ↑ exchange rate.

Placental Barrier

Definition & Type

- Placental membrane: separates maternal blood in intervillous spaces from fetal blood in chorionic villi.
- Not a *true* barrier → many substances pass freely.
- Human placenta = Hemochorial type → maternal blood in direct contact with chorionic tissue.
- Normally no mixing of maternal & fetal blood, but microscopic defects allow occasional fetal RBC escape.

Initial 4 layers (early pregnancy):

1. Endothelial lining of fetal vessels.
2. Connective tissue in villus core.
3. Cytotrophoblastic layer.
4. Syncytiotrophoblast.

From 4th month onward:

- Membrane thins → endothelial lining directly contacts syncytium.

- Purpose: ↑ efficiency of maternal–fetal exchange.

Clinical Correlation – Hemolytic Disease of the Fetus & Newborn (HDFN)

Cause

- Some fetal RBCs cross placenta → enter maternal circulation.

- Maternal immune system may recognize foreign RBC antigens → isoimmunization.

- Antibodies attack fetal RBCs → hemolysis → anemia.

Terminology

- Old term: Erythroblastosis fetalis → used when severe anemia caused ↑ erythroblast production.

- Preferred term: Hemolytic disease of the fetus and newborn (HDFN).

Severe Complication

- Fetal hydrops:

    - Generalized edema + effusions into body cavities.
    - May cause fetal death if untreated.

Rh (CDE) Group – Most Dangerous

- Rh (D antigen) → high immunogenicity.

- Mechanism:

    - Mother Rh-negative, fetus Rh-positive.
    - Fetal blood enters maternal circulation (e.g., microbleeds in placenta, childbirth).
    - First exposure → sensitization.
    - Subsequent pregnancies → earlier & more severe hemolysis.

Prevention

- Screen all pregnant women early for:

    - Rh type.
    - Anti-D antibodies.

- In Rh-negative women without antibodies:

    - Rh immunoglobulin (RhIg) at:
        - 28 weeks gestation.
        - After potential feto-maternal bleed (amniocentesis, miscarriage).
        - Post-delivery (if newborn Rh-positive).

- Since RhIg introduction (1968) → HDFN from Rh almost eliminated in the USA.

ABO Incompatibility

- Can also cause HDFN, but milder.

- ~20% pregnancies → maternal ABO incompatibility.

- Only ~5% clinically affected.

- Postnatal treatment effective.

Functions of the Placenta

1. Exchange of Metabolic & Gaseous Products

- Gases:

- O_2, CO_2, CO → simple diffusion.
- Term fetus extracts 20–30 mL O_2/min from maternal circulation.
- Oxygen delivery depends mainly on placental blood flow, not just diffusion capacity.

- Nutrients & Electrolytes:

  - Amino acids, free fatty acids, carbohydrates, vitamins → rapid transfer.
  - Transfer ↑ as pregnancy advances.

- Maternal Antibody Transmission:

  - IgG transport begins ~14 weeks.
  - Passive immunity against maternal infections.
  - Neonate makes own IgG, but adult levels reached by ~3 years.
  - Complement system components made by late 1st trimester.

2. Hormone Production *(all in syncytiotrophoblast)*

Hormone	Timing	Function
Progesterone	From 4th month	Maintains pregnancy; corpus luteum no

		longer essential.
Estrogens (esp. estriol)	Gradually ↑, peaks near term	Uterine growth, mammary gland development.
hCG	First 2 months	Maintains corpus luteum; detectable in urine → pregnancy test.
Somatomammotropin *(placental lactogen)*	Throughout pregnancy	Gives fetus priority for glucose; diabetogenic effect in mother; promotes breast development for lactation.

Clinical Correlates – Placental Barrier Limitations

Hormonal Transfer

- Freely crosses: most maternal steroid hormones.
- Slowly crosses: thyroxine.
- Synthetic hormones:
    - Some progestins → may masculinize female fetus.

- Diethylstilbestrol (DES) → crosses easily; causes:
  - Clear-cell carcinoma of vagina.
  - Cervix & uterus malformations in females.
  - Testicular abnormalities in males.

Infections Crossing Placenta

- Rubella, Cytomegalovirus, Coxsackie, Variola, Varicella, Measles, Poliovirus.

- May cause fetal infections → cell death, birth defects.

Drugs Crossing Placenta

- Most drugs & metabolites → cross freely; many are teratogenic.

- Heroin & cocaine: cause fetal addiction.

Amnion & Umbilical Cord Development

- Primitive Umbilical Ring (5th week):

  - Contains:
    1. Connecting stalk → allantois + umbilical vessels (2 arteries, 1 vein)
    2. Yolk stalk (vitelline duct + vessels)

3. Canal between intra- and extraembryonic cavities

- Amnion Growth → Envelops connecting & yolk stalks → primitive umbilical cord

- Early cord contents: yolk stalk, umbilical vessels, intestinal loops, allantois remnant

- By 3rd month:

    - Amnion contacts chorion → chorionic cavity obliterated
    - Yolk sac shrinks/obliterates
    - Intestinal loops return to abdomen → cavity in cord gone
    - Final cord: 2 arteries + 1 vein in Wharton's jelly

- Wharton's jelly: Proteoglycan-rich; protects vessels

- Artery walls: Muscular + elastic → rapid constriction after cord clamping

Placental Changes Near Term

Signs of ↓ exchange capacity:

1. ↑ Fibrous tissue in villus core

2. Thickened fetal capillary basement membranes

3. Obliterated villous capillaries

4. Fibrinoid deposition → infarction of intervillous lake or whole cotyledon

Amniotic Fluid

- Source: Mostly maternal blood; partly from amniotic cells

- Volume:

    - 10 wks → 30 mL
    - 20 wks → 450 mL
    - Term → 800–1000 mL

- Functions:

    1. Cushion from trauma

    2. Prevent adhesion to amnion

    3. Allow movement

- Turnover: Replaced every ~3 hrs

- By 5th month:
    - Fetus swallows ~400 mL/day
    - Fetal urine (mostly water) contributes
    - Placenta handles waste removal
- During labor: Amniochorionic membrane → hydrostatic wedge for cervical dilation

Clinical Correlates

Umbilical Cord Abnormalities

- Normal: 2 arteries, 1 vein; 50–60 cm long; 1–2 cm diameter
- Length:
    - Short → may cause placental detachment during delivery
    - Long → may wrap around neck (usually harmless)
- Single umbilical artery (~0.5% births):
    - 20% risk of cardiac/vascular defects
    - Cause: agenesis or early degeneration

Amniotic Bands

- Tear in amnion → fibrous bands

- May wrap around fetal parts → amputations, constrictions, craniofacial defects

Amniotic Fluid Disorders

- Polyhydramnios (>1.5–2 L)
    - Causes: idiopathic (35%), maternal diabetes (25%), fetal CNS defects (↓ swallowing; e.g., anencephaly), GI atresia (e.g., esophageal)

- Oligohydramnios (<400 mL)
    - Causes: renal agenesis (↓ urine), amniotic leak
    - Complications: fetal compression (clubfoot), lung hypoplasia

- PROM (Premature rupture of membranes)
    - Before labor; ~10% pregnancies
    - Preterm PROM (<37 wks): ~3% pregnancies → major cause of preterm labor
    - Risks: prior PROM, prematurity, Black race, smoking, infection, severe polyhydramnios

Fetal Membranes in Twins

Multiple Gestations – General Facts

- Current frequency: >3% of all live births (USA)
- Twin rate in USA (2008): 32.6 per 1,000 births
- Increase due to:
    1. ↑ Maternal age at childbirth
    2. ↑ Fertility treatments (ART, ovulation induction)

1. Dizygotic (Fraternal) Twins

- Incidence: ~90% of twins
- Mechanism:
    - 2 oocytes ovulated → fertilized by 2 different sperm
    - Genetically distinct, like regular siblings → may be same or opposite sex
- Membranes & Placenta:
    - Usually: separate placenta, chorion, and amnion (dichorionic diamniotic)
    - Sometimes: placentas/chorions fuse → possible erythrocyte mosaicism (RBC exchange via

vascular anastomoses)

- Risk Factors:
    - Maternal age (↑ incidence; doubles at ~35 years)
    - Fertility procedures (ART, gonadotropins)

2. Monozygotic (Identical) Twins

- Incidence: 3–4 per 1,000 births (constant worldwide)
- Mechanism: Single zygote → splits at various stages

Timing of Splitting & Resulting Membranes

Stage of splitting	Placenta	Chorion	Amnion	Example
2-cell stage (very early)	Separate	Separate	Separate	Dichorionic diamniotic
Early blastocyst (inner cell mass splits)	Common	Common	Separate	Monochorionic diamniotic

Bilaminar germ disc (before primitive streak)	Common	Common	Common	Monochorionic monoamniotic

- Blood supply: Usually balanced even if placenta is shared
- Rare: Monoamniotic → ↑ risk of cord accidents

3. Higher-Order Multiples

- Triplets: ~1 per 7,600 pregnancies
- Quadruplets, quintuplets: rarer, ↑ with fertility drugs (gonadotropins) and ART

Clinical Correlates in Twins

A. General Risks

- ↑ Perinatal mortality & morbidity
- ~60% born preterm
- ↑ Low birth weight incidence
- Infant mortality = ~3× higher than singletons

B. Vanishing Twin Syndrome

- One fetus dies in utero (first trimester/early second)
- Mechanisms:
  - Resorption
  - Fetus papyraceus (compressed remains)

C. Twin-Twin Transfusion Syndrome (TTTS)

- Occurs in ~15% of monochorionic monozygotic twins
- Cause: Unbalanced placental vascular anastomoses →
  - Donor twin: reduced blood flow → smaller, anemia
  - Recipient twin: excess blood → larger, polycythemia
- Prognosis: Poor → 50–70% risk of death of both twins

D. Conjoined Twins

- Due to partial splitting of primitive node/streak at later stage

- Classification: by nature & degree of union (thoracopagus, pygopagus, craniopagus, etc.)
- May be joined by skin bridge or liver bridge
- Possible cause: misexpression of Goosecoid gene
- Famous example: *Chang and Eng* (joined at abdomen; lived normal adult lives)

Dizygotic Twins – Hormonal Influence

- Brother–sister pairs:
    - Testosterone transfer from male twin to female twin (via shared maternal environment → amniotic fluid/placental circulation)
    - Effects on female twin:
        - Square jaw
        - Larger teeth
        - Better spatial ability & ball skills
        - ↓ Marriage likelihood (–15%)
        - ↓ Fertility (~25% fewer children)

Parturition (Birth)

I. Pre-labor Uterine Changes

- Gestational weeks 34–38: Myometrium unresponsive to birth signals

- Last 2–4 weeks:

    - Upper uterus → myometrium thickens
    - Lower uterus & cervix → softens & thins (cervical ripening)

2. Stages of Labor

Stage	Events	Main Forces
Stage 1	Effacement (thinning) + dilatation of cervix to full	Uterine contractions → amniotic sac or fetal head presses on cervix
Stage 2	Delivery of fetus	Uterine contractions + intra-abdominal pressure from abdominal muscles
Stage 3	Delivery of placenta & fetal	Uterine

membranes	contractions + intra-abdominal pressure

3. Mechanism of Uterine Contractions

- Upper uterus: Retracts → lumen progressively smaller
- Lower uterus: Expands → directs fetal movement downward
- Frequency:
  - Early: ~10 min apart
  - Late (Stage 2): <1 min apart, lasting 30–90 sec
- Pulsatile pattern essential → prevents prolonged interruption of uteroplacental blood flow

Clinical Correlates – Preterm Birth

Definition: Birth before 37 completed weeks

- Incidence (USA): ~12% of births
- Significance:

- Leading cause of infant mortality
- Major contributor to morbidity

Causes:

1. Preterm PROM (Premature rupture of membranes)
2. Premature onset of labor
3. Pregnancy complications → indicated early delivery

Risk Factors:

- Previous preterm birth
- Black race
- Multiple gestations
- Maternal infections:
  - Periodontal disease
  - Bacterial vaginosis
- Low maternal BMI

Proposed Mechanisms of Labor Initiation

- Retreat from maintenance: Withdrawal of pregnancy-supporting factors (e.g., hormones)

- Active induction: Stimulatory factors acting on uterus

- Likely combination of both

Here's how to tackle these problems step-by-step using embryology reasoning and clinical application.

Exercise Problems

Problem 1

Q: Ultrasound at 7 months → excess fluid in amniotic cavity.

A: Polyhydramnios

Definition:

- Polyhydramnios = Excess amniotic fluid

- Criteria: Amniotic fluid index (AFI) > 24 cm OR single deepest vertical pocket > 8 cm

Causes:

1. Fetal swallowing defect (commonest cause)

- Anencephaly → no swallowing reflex
- Esophageal/intestinal atresia → fluid cannot pass to stomach/intestines

2. Maternal diabetes mellitus → fetal polyuria from osmotic diuresis

3. Multiple gestations

4. Fetal anemia → ↑ cardiac output → ↑ urine production

5. Idiopathic (~50% cases)

Mechanism:

- Normally → amniotic fluid is swallowed by fetus → absorbed in GIT → transferred to maternal circulation via placenta.

- In swallowing defect or blockage, fluid accumulates → polyhydramnios.

Problem 2

Q: Exposure to toluene at 3rd week gestation → "placenta protects against toxins" — Is this correct?

A: Incorrect

Why?

- Placenta is not an absolute barrier → many toxins, drugs, viruses cross.
- 3rd week = early embryonic period (gastrulation, early organ primordia) → high teratogenic vulnerability.
- Toluene = lipophilic organic solvent → easily crosses placenta by diffusion → can cause fetal toxicity & malformations.

Key points about placenta as barrier:

- Effective for: Some bacteria, large molecules (e.g., most proteins)
- Ineffective for:
    - Lipid-soluble toxins (e.g., alcohol, organic solvents, some pesticides)
    - Viruses (rubella, CMV, HIV)
    - Many drugs (thalidomide, warfarin)
- In first trimester, teratogen exposure can cause major structural malformations.

✅ Final Answers:

1. Polyhydramnios – often due to fetal swallowing defects (e.g., anencephaly, esophageal atresia), maternal diabetes, multiple gestations, or idiopathic causes.

2. No – placenta is not an absolute barrier; toluene can cross and harm the developing embryo, especially during the critical early weeks.