CLINICAL OBSTETRICS NOTES

PRE-SUMMARIZED
READY-TO-STUDY
HIGH-YIELD NOTES

FOR THE TIME-POOR
MEDICAL, PRE-MED,
USMLE OR PA STUDENT

REGENT STUDIES

MEDICAL NOTES
(MBBS, MD, MBChB, USMLE, PA, & Nursing)
Anatomy, Physiology, Pathophysiology, Pathology, Histology & Treatments

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What's included: Ready-to-study anatomy, physiology and pathology summaries of the female reproductive system presented in succinct, intuitive and richly illustrated downloadable PDF documents. Once downloaded, you may choose to either print and bind them, or make annotations digitally on your iPad or tablet PC.

Free Bonus: 'Obstetrics' chapter of Toronto Notes for reference and further detailed reading.

File List:

- Female Reproductive System Overview
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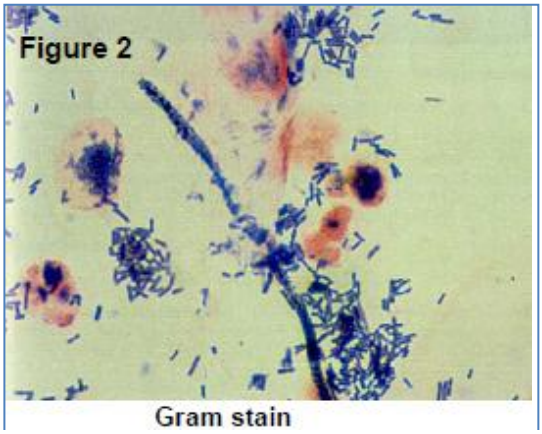
System: FEMALE REPRODUCTIVE SYSTEM

Pelvic Cavity:

- **Male:**
  - Urinary Bladder
  - Rectum
- **Female:**
  - Urinary Bladder
  - Uterus
  - Rectum

Normal Flora of the Genital Tract

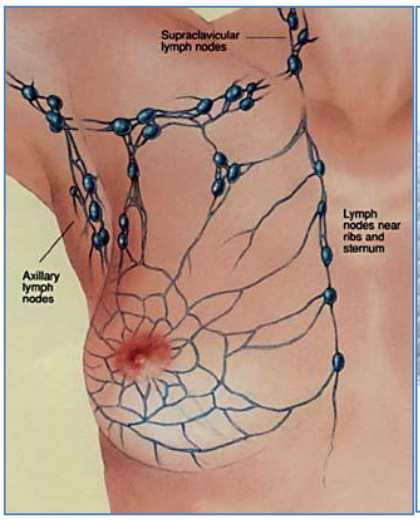
- **Male:**
  - **Urethra** – Few Organisms - (*Staph. epidermidis*, Streptococci, *Uroplasma urealyticum*)
- **Female:**
  - **Vagina** – High Numbers of Bacteria – (***Lactobacillus*** - Blue Gram Positive Rods, + Some Anaerobes)
    - → Produce lactic acid
    - → Protects against Bacterial Vaginosis & Yeast Infections.

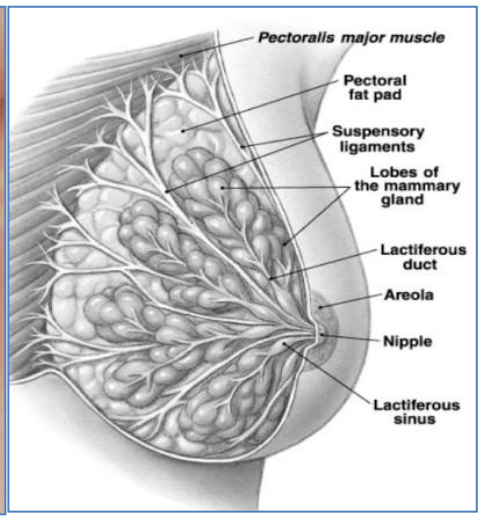


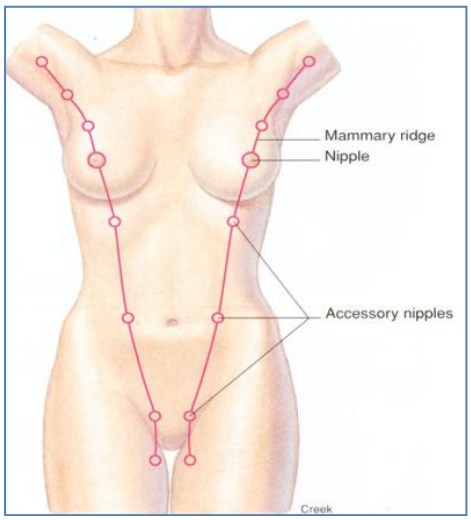
Gram stain

Revision of The Breast:

- **Anatomy:**
  - Attached to Pec-Major by Suspensory Ligaments
  - **Glandular Breast Tissue:**
    - Approx 20 lobes/lobules → Converge to Lactiferous Ducts → Lactiferous Sinuses → Nipple
  - **Lymphatic Drainage:**
    - Supraclavicular, Infraclavicular, Parasternal, Pectoral, Axillary, Central, Subscapular

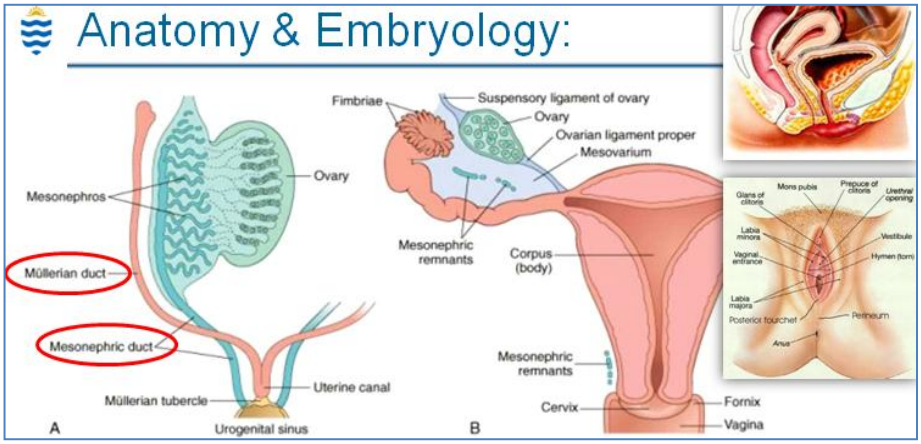




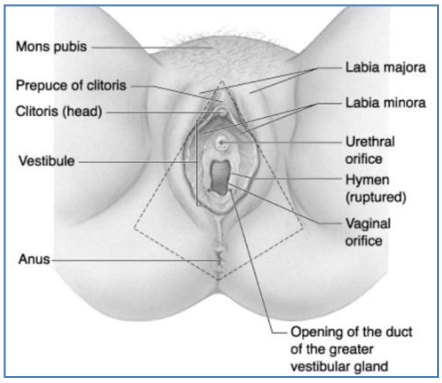


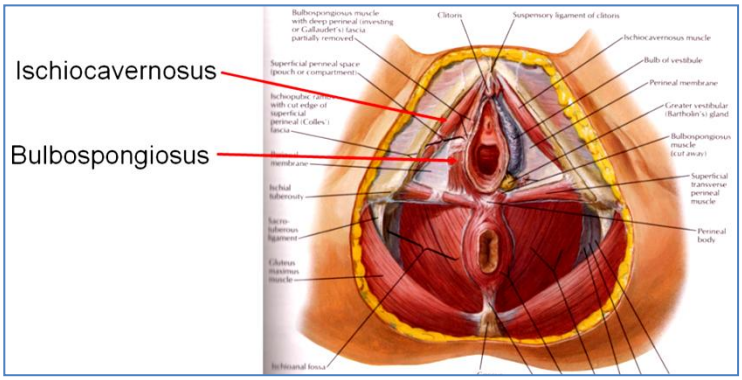
Review of Normal Female Reproductive Anatomy:

- **Embryology:**
  - **Female = The Default Sex** - (NB: The SRY Gene on the Y-Chromosome = the Male Determining Gene)

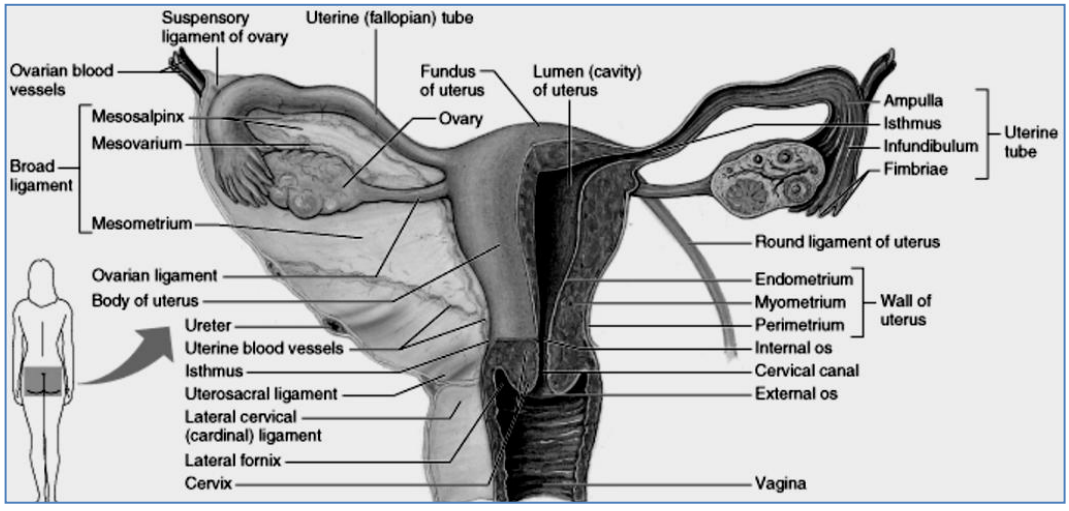


- **Anatomy:**
  - **Vagina/Vulva:**
    - Labia Majora & Minora
    - Clitoris & prepuce of clitoris
    - Urethral orifice

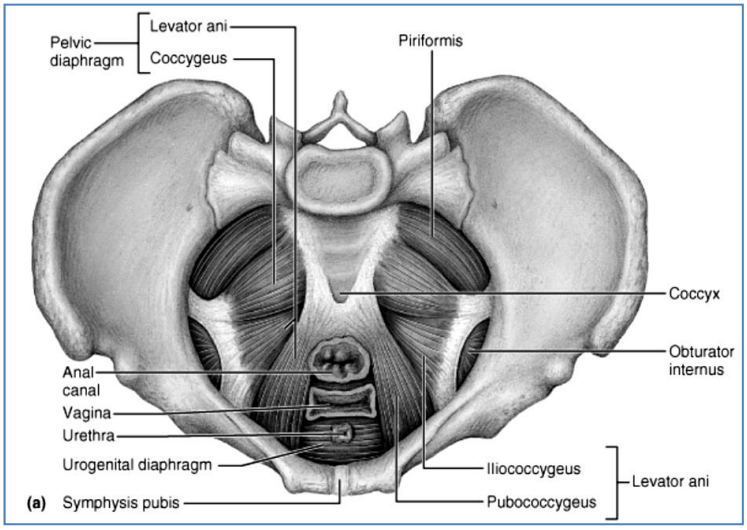




  - **Uterus -** Fundus (top / head), Body, Cervix (external os, canal, internal os), Lumen (internal cavity)
    - **Perimetrium** – Outer wall
    - **Myometrium** – Middle of wall
    - **Endometrium** – Inner wall
  - **Uterine (fallopian) Tubes**
    - Common site of fertilisation
    - Infundibulum – projections = fimbriae (closest to ovary) → Receives oocyte
  - **Ovaries (gonads)**
    - Produce female gametes (oocytes)
    - Secrete female sex hormones – (Oestrogen & Progesterone)
    - Held in place by ligaments & muscles

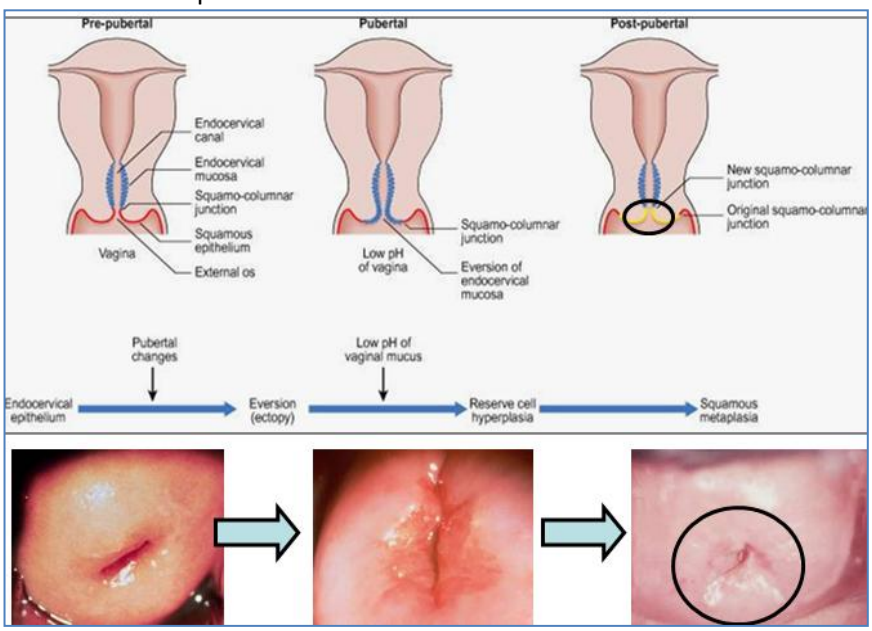


- **Blood Supply:**
  - **Internal iliac artery:**
    - Branches from common iliac artery.
    - Uterine Artery
    - Vaginal Artery
    - To external genitalia
  - **Ovarian Artery:**
    - To ovaries, uterine tubes and uterus
- **Pelvic Diaphragm:**
  - **Levator Ani (anterior half)**
    - Iliococcygeus
    - Pubococcygeus
  - **(posterior) Coccygeus** (ischiococcygeus)
  - **(posterior) Piriformis**



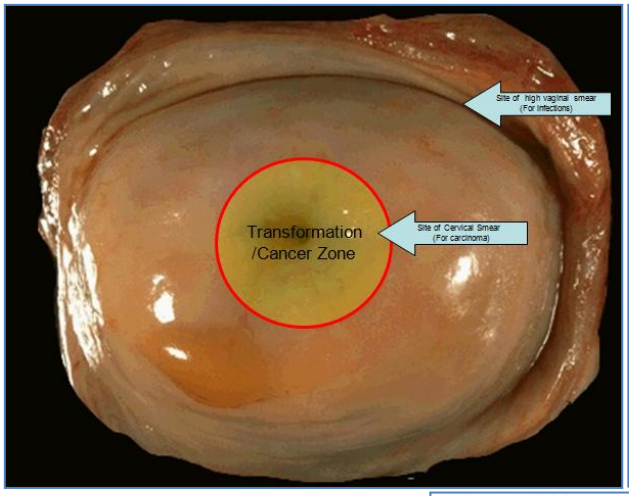
Background Information on the Cervix:

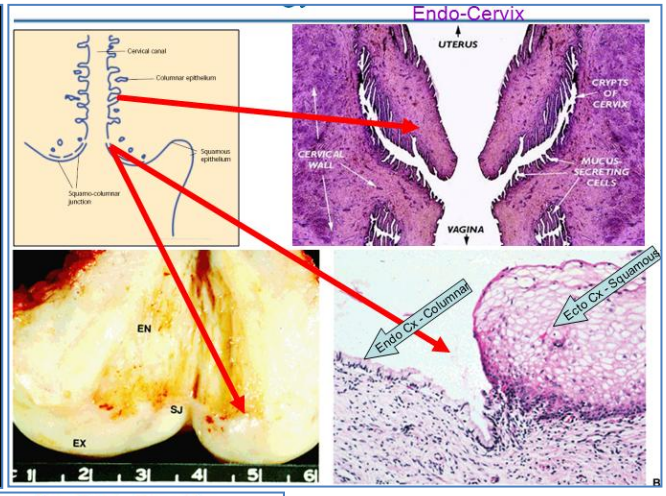
- **NB: The Transformation Zone – Commonest location of Cervical Cancer.**
  - **TZ =** The location of Transition from Squamous to Columnar Epithelium.
  - **NB:** During puberty, Columnar Epithelium Migrates out of the os → Exposed to Vaginal Acidity → Metaplasia to Squamous Epithelium
  - This is the area Predisposed to Cancer.

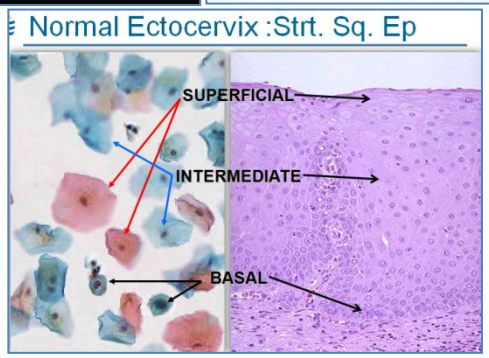


(Red = Squamous) (Blue = Columnar)

- **NB: The Normal Cervix – Anatomy & Histology:**
  - **Endocervix =** *Simple Columnar Epithelium*
  - **Ectocervix =** *Stratified Squamous Epithelium*

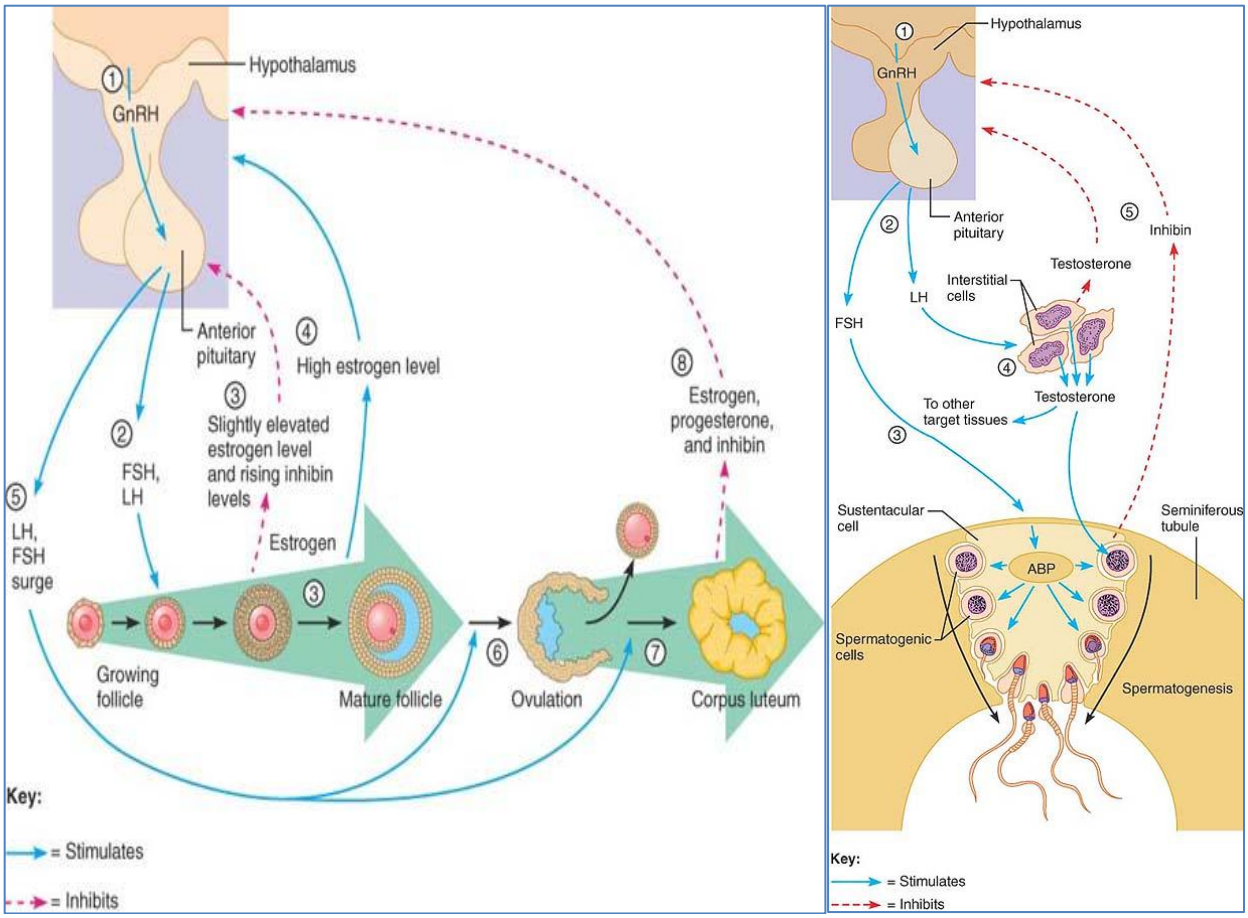






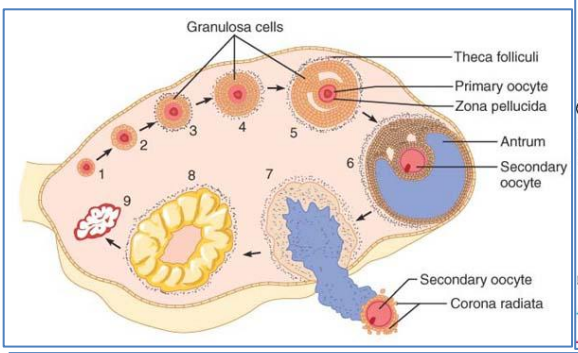
Review of Female Reproductive Physiology:

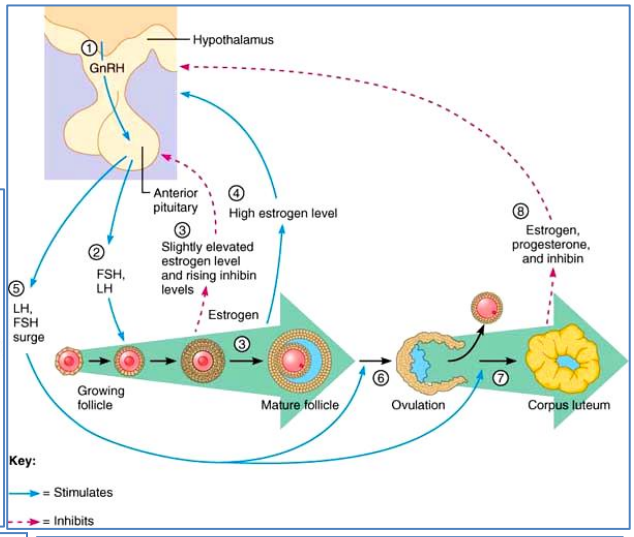
- **Puberty:**
  - A gradual series of events that transform a child into a sexually mature adult.
  - **Female:** Marked by first menstrual period (average age 13)
  - **(Male:** Marked by physical development of Male Sex Characteristics)
- **Initiation:**
  - Activation of **Hypothalamo-Pituitary-Gonadal Axis** → establishes regulation of gonadal function.
    - **At puberty** → ↓ Sensitivity of the hypothalamus to Inhibitory Steroid Hormones →↑GnRH →↑FSH & LH → ↑Gonadal Testosterone/Oestrogen/Progesterone → Sexual Maturation.

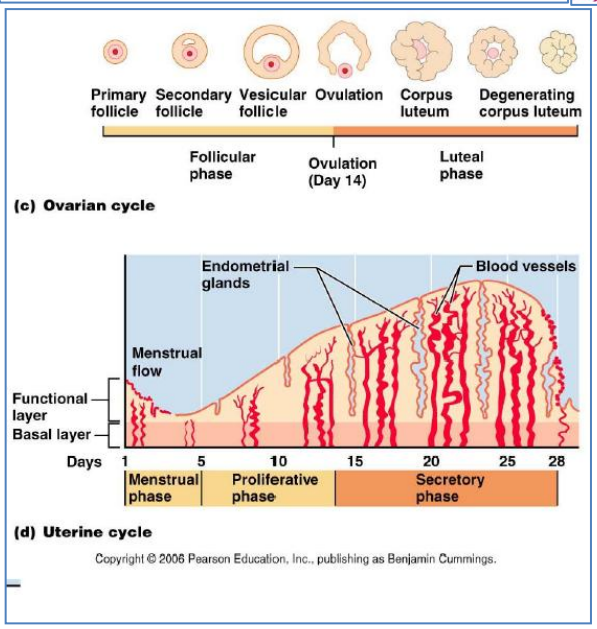


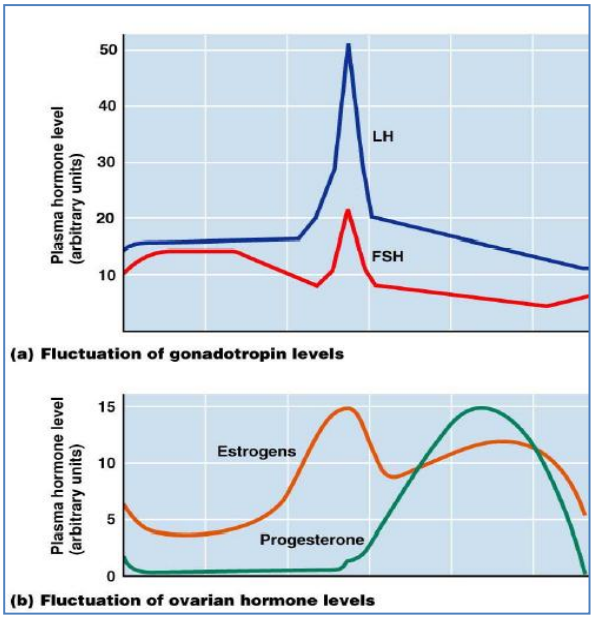
- **Menopause:**
  - **Menopause "occurs" when it has been a year since the last menstruation.**
  - Gradual process over 3-5yrs (between ages 46-54)
  - **Mechanism:** ↓Follicle Sensitivity to FSH → ↓Follicles Recruited →↓Oestrogen Levels Production → Symptoms:
    - ↓Ovulation
    - Irregular, Lighter Periods
    - Hot flushes
    - Palpitations
    - Insomnia, Depression
    - Breast Atrophy
    - Vaginal Dryness
    - **Osteoporosis**

- **The Female Reproductive Cycle:**
  - The monthly series of events associated with the maturation of an egg.
  - Typically 28 days long.
  - **Days 1-5: *Menstruation*:**
    - **Shedding of the Endometrium**
    - Low levels of all hormones (FSH, LH, Oest. & Prog).
  - **Days 5-14: The Follicular/Proliferative Phase:**
    - **Follicular Recruitment & Growth**
    - **+ Endometrial Proliferation**
    - Rising levels of Oestrogen as Follicle/s get larger.
  - **Day 14 (Mid-Cycle): *Ovulation*:**
    - Surge of FSH & LH → Ovulation into peritoneal cavity → Oocyte enters Fallopian Tubes.
    - **FERTILE**
  - **Days 14-28: The Luteal Phase:**
    - **Transformation of Follicle → Corpus Luteum**
    - Corpus Luteum Secretes Mainly Progesterone (& Some Oestrogen)
      - Degenerates (Unless pregnancy occurs →C.L. persists until the placenta can take over).
    - **FERTILE**
  - **Day 28: End of Cycle:**
    - **Corpus Luteum Degenerates** → No Oestrogen/Progesterone to sustain Thick Endometrium →Endometrial Arteries become Spastic & Tortuous → Menstruation.









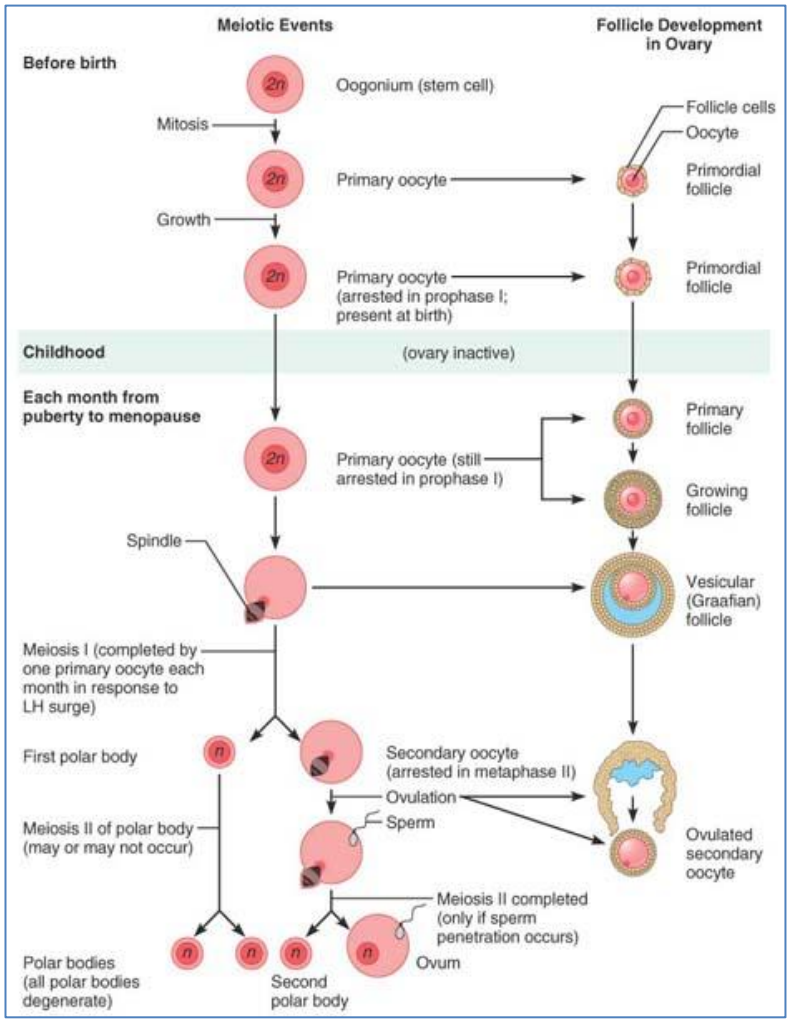
Meiosis (Female) – Oogenesis:

- It is thought that in general, the total number of eggs in a female is predetermined at birth.
- **Female gamete production = Oogenesis.**
    - Done through **meiosis**
        - Specialized cell division
        - Usually produces 4 haploid cells.

1) **Foetal period** - the, **Oogonia** (diploid ovarian stem cells) multiply rapidly by mitosis, then enter a growth phase and lay in nutrient reserves as **Primary Oocytes**.
2) These **Primary Oocytes** then become **surrounded by** a single layer of **Follicle Cells** forming a **Primordial Follicle.**
3) **Primary Oocytes** (of the primordial follicles) then begin the **first meiotic division.** However, they are **arrested in prophase I.**
4) Female is born with approx. 2million primary oocytes. By puberty, 250000 primary oocytes are left.
5) **Puberty—Menopause:** Each month, a small number of **primary oocytes** are recruited in response to the LH surge midway through the menstrual cycle. (Luteinising Hormone) As these **primary oocytes** prepare to divide, a spindle forms on its edge, creating a small "nipple" where half of the chromosomes will be cast during division.
6) Only **one of the primary oocytes** is selected to **continue meiosis I.** Produces **2 haploid cells** (23 chromosomes each) **dissimilar in size**. The smaller cell is the "**first polar body"** (little->no cytoplasm) and the larger cell is the **secondary oocyte**. → The **secondary oocyte** is then arrested in **metaphase II** and **OVULATED.** (unequal Cytoplasmic divisions ensure that a fertilised egg has ample nutrients for its week-journey to the uterus.)
7) The **ovulated secondary oocyte MUST be penetrated by a SPERM for it to complete MEIOSIS II,** yielding one large **OVUM** and a **"Second polar body"**

***Note: -** The potential products of oogenesis are 3 small polar bodys and one large ovum. (3 polar bodies aren't always formed – first polar body often perishes before meiosis II)

-Only the **OVUM** is a ***functional gamete.***



SPECIFIC OBSTETRIC NOTES:
MATERNAL CHANGES IN PREGNANCY

Effects of Pregnancy on Mother:

- **Anatomical Changes**
  - **Reproductive Organs:**
    - - Become increasingly vascular and engorged with blood.
    - Vagina develops purplish hue (Chadwick's sign)
    - Breasts engorge with blood → enlarge + darken.
    - Uterus enlarges – pushes higher into abdominal cavity – abdominal organs intrude into thoracic cavity causing ribs to flare.
    - Placental release of **relaxin** relaxes pelvic ligaments + pubic symphysis
- **Metabolic Changes**
  - Placenta secretes **human chorionic somatomammotropin (hCS)** →cooperatively with oestrogen & progesterone to stimulate maturation of breasts for lactation.
  - **hCS** exerts glucose-sparing in mother-cells → sparing glucose for foetus. (mother-cells increase fatty acid metabolism, decrease glycolysis) [can cause gestational diabetes]
  - **human Chorionic Thyrotropin (hCT)** causes hypermetabolism and increase Ca^{+} concentration→ensuring mineralisation of foetus' bones.
- **Physiological**
  - **Gastrointestinal System**
    - Morning sickness – due to increased oestrogen & progesterone
    - Heartburn/reflux – increased pressure on stomach by foetus
    - Constipation – increased fluid uptake + decreased bowel motility.
  - **Urinary System**
    - Kidneys produce more urine – extra fetal metabolic wastes + increased pressure on bladder.
  - **Respiratory System**
    - Nasal mucosa becomes congested – nasal stiffness.
    - Tidal volume & respiratory rate increases.
  - **Cardiovascular System**
    - Total body water rises → blood volume increases 25-40% to accommodate foetus. Also compensates for blood loss during birth.
    - Blood pressure rises
    - Pulse rises
    - Cardiac output increases 20-40% → propels greater circulatory volume.
    - Uterus may press on pelvic blood vessels, impairing blood return from lower limbs → varicose veins.

Maternal Adaptations to Pregnancy

Reproductive System:

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Urinary System:

- **Anatomical Changes:**
  - Compression of the bladder
  - Relaxation of the sphincters (progesterone)
  - Dilation & Lengthening of the ureters (progesterone → SM relaxation)
- **Physiological Changes:**
  - Urinary Incontenance (SM relaxation of sphincters)
  - Decreased capacity of the bladder to store urine → ↑frequency of urination.
  - Increased plasma volume → Increased GFR.
    - ↑ Urine Production – (To remove the extra foetal metabolic wastes)
    - → Decreased reabsorption of ions in nephron.
    - NB: an increase in aldosterone compensates for the above. (In the distal nephron)
  - Increased risk of UTIs due to the decreased tone in sphincter & therefore bugs can get in.

Respiratory System:

- **Anatomical:**
  - Diaphragm can't flatten fully → "Splitting of the Diaphragm"
    - Diaphragm also rises up to 4cm.
  - Ribs flare out → Diameter of thorax increases by 6-10cm
  - Use of more accessory muscles
  - Pregnant women snore due to hyperaemia → Nasal & Tracheal oedema/congestion. (Can make it hard to intubate a pregnant lady – Endotracheal tube is smaller)
- **Physiological:**
  - More oxygen is needed
  - Increases respiration (↑Tidal Volume & ↑Minute Ventilation Rate)
  - Minimises residual volume
  - No changes in partial oxygen pressure in blood.
  - No changes in partial CO_2 pressure in blood.
  - Hyperventilation accounts to the above 2.
  - Progesterone increases the sensitivity of the respiratory centre to CO_2.
  - Increases capacity for inspiration & expiration → decreases residual volume.

Cardiovascular System:

- ↑Plasma Volume → Hypertension
- ↑HR
- ↑Cardiac output required to perfuse the extra body tissues (placenta, breasts, fat tissues)
- Uterus may press on pelvic blood vessels, impairing blood return form lower limbs → Varicose Veins.

GI System:

- **Anatomical:**
  - **GI tract is displaced superiorly & laterally.**
    - Important to know in case of appendicitis as the appendix will be higher than normal (usually experience pain in right flank)
  - **Mouth:**
    - Saliva increases (keeps the mouth alkaline – keeps the pH up to prevent mouth ulcers & tooth decay)
  - **Cardioeosophageal sphincter is more relaxed (due to progesterone) & has to resist more pressure due to ↑intraabdominal pressure.**
    - Increased risk of aspiration if under anaesthetic or unconscious. → Pregnant women must fast for longer than normal before anaesthetic.
  - **Stomach:**
    - Hydrochloric acid production decreases.
    - Decreased gastric motility (due to progesterone) allows more time for digestion of stomach contents.
  - **Bowel:**
    - Decreased bowel motility (less peristalsis due to progesterone) → more time to absorb nutrition & water → Constipation.
    - Constipation also caused by mechanical obstruction of the rectum by the uterus.
  - **Liver:**
    - No change in size
    - Works harder to produce enzymes but they don't increase. (eg. ALT). Raised liver enzymes are a sign of disorder.
      - One enzyme will be increased = "Alkaline Phosphatase" – double/triple in pregnancy – Produced by the placenta & liver.
  - **Gallbladder:**
    - Progesterone → less contraction of the gallbladder → Flow of bile becomes slower → more time for the metabolilsm of fat/cholesterol.
      - If Bile-flow is too slow → Gallstones risk is higher.
- **Physiological:**
  - Morning Sickness – due to ↑Oestrogen & ↑Progesterone.
  - Heartburn/Reflux – Increased Pressure on stomach by foetus.
  - Constipation – Increased fluid uptake + ↓Bowel Motility (Due to Progesterone)

Endocrine:

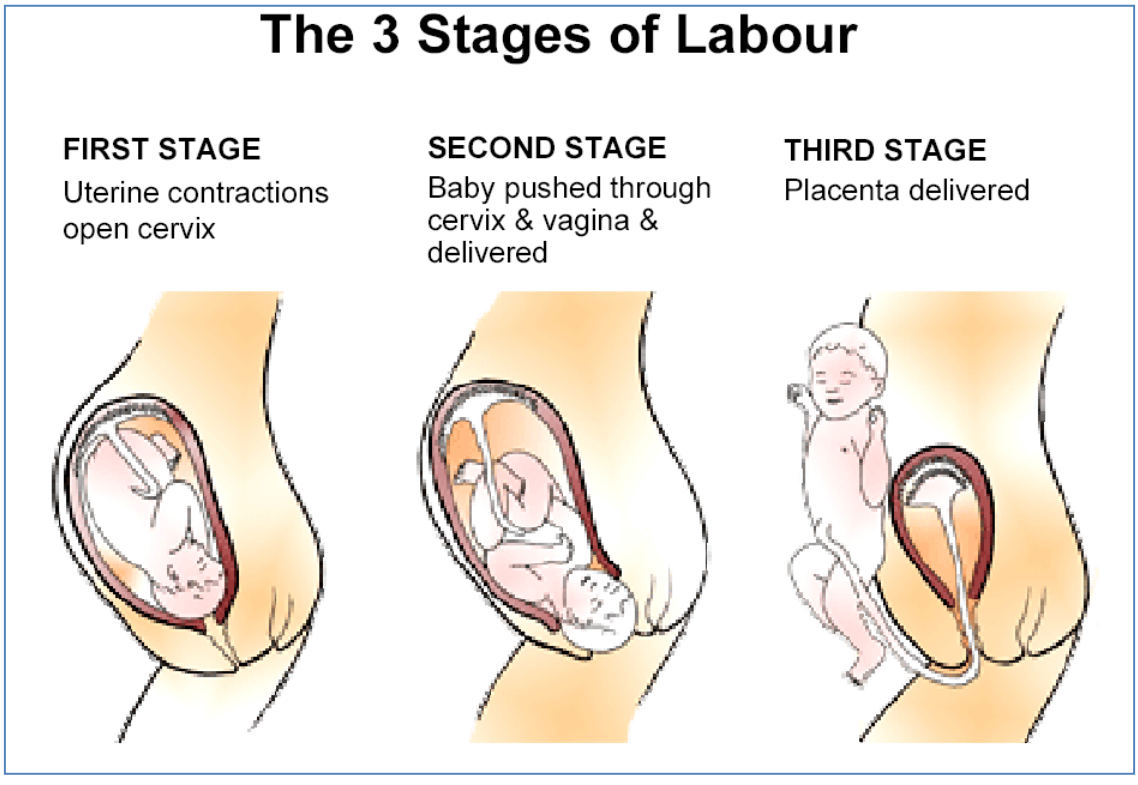
- **Pancreas:**
  - Secretes lots of insulin → to facilitate glucose uptake in the cells.
  - But we still get hyperglycaemia. Why?:
    - Pregnancy induces insulin resistance to maintain the high blood sugar.
    - Human placental lactogen & cortisone/cortisol & Progesteron competes with the insulin for the receptors. → Insulin 'resistance'. (Competetive inhibition)
- **Thyroid Gland:**
  - Increases in size
  - Increases vascularity
  - Increases production of Thyroid Hormones – but may not have hyperthyroidism. Why? – Oestrogen increases thyroid binding globulin to decreases the 'free thyroxin' in the blood doesn't increase.
  - Human Chorionic Gonadotropin is what stimulates the thyroid & causes hypertrophy (not Thyroid Stimulating Hormone)
- **Adrenals:**
  - Oedema – due to ACTH production → ↑Aldosterone & Cortisol → Water retention.

Parturition / Labour / Childbirth

Time of birth:
280 Days after last menstrual period +/- 15 days

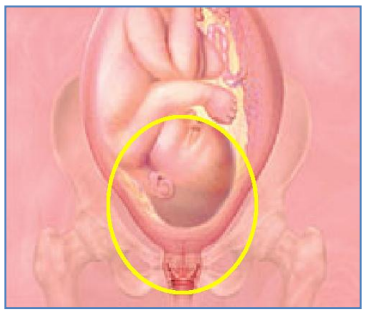
Foetal Maturation of Organ Systems:
- Essential systems for extrauterine life:
  - Interface organs – lungs, gut, immune system
  - Homeostatic organs – endocrine (hypothalo-pituitary-adrenal axis), kidneys, liver, pancreas
- Promoted by maternal adrenal **glucocorticoids** (steroid hormones). **Causes**:
  - Surfactant production in lungs
  - Activity of enzyme systems in gut, retina, pancreas, thyroid gland, brain.
  - Deposition of glycogen in liver.

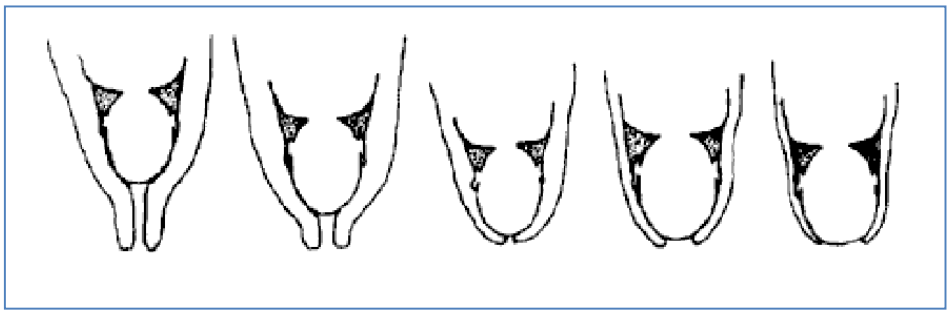
Initiation of Labour / Parturition
- Triggered by a combination of hormonal changes.
- Oestrogen levels peak in late pregnancy, resulting in:
  - Increased uterotonin receptors in uterus – oxytocins + prostaglandins.
  - Decreased effect of progesterone → Myometrium becomes irritable → weak, irregular contractions = **false labour**
- Shortly before parturition (**true labour**), foetal cells produce **oxytocin** – cause placenta to release **prostaglandins**.
  - Both hormones (**oxytocin + prostaglandins**) are powerful **uterine muscle stimulants**.
  - The highly sensitive uterus responds – contractions become more frequent & vigorous.
- Then, increasing stresses activate mother's **hypothalamus** – causes **Post. Pituitary** to release **oxytocin** – causing a positive feedback loop of stress & oxytocin release.



Stages of Labour:

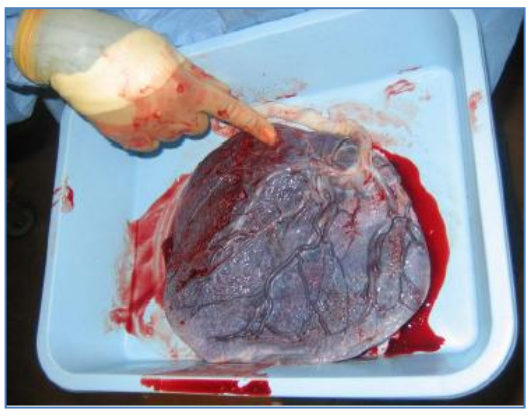
1. **Dilation Stage**
   - Time between initiation of labour and full cervical dilation. (10 cm)
   - Amniotic sac usually breaks just before this stage.
     - At first, contractions begin in the fundus, then spread down towards vagina.
     - As the infant's head is forced against cervix, the cervix softens and thins – begins to dilate.
     - Takes approx 6-12 hrs
     - Baby's head rotates to minimise its profile.





2. **Expulsion Stage**
   - Time between full dilation to delivery of infant.
     - By the time cervix is fully dilated, contractions are strong and regular – 2-3 min intervals.
     - Takes on average 1hr.
   - **Vertex position (usual – head first)**
     - **Crowning** occurs – baby's head is halfway distended from the vulva.
     - Baby's head is delivered, then the rest of the body.
     - Umbilical cord is clamped and cut.
   - **Breech position (unusual – bum first)**
     - Much more difficult
     - Often requires use of forceps or c-section.

3. **Placental Stage**
   - Delivery of the placenta (approx 30 mins)
     - Baby should be breast fed directly after 2nd stage → stimulates oxytocin release → causes uterine contractions:
       - Uterine contractions compress uterine blood vessels, sheering placenta from uterine wall.
       - Also prevents *postpartum bleeding*



Adjusting to Extrauterine Life:

Breathing

- Baby must be able to breathe.
- Once placenta detatches, CO_2 builds up in baby's blood, causing acidosis.
    - This excites respiratory control centres in brain – triggers first inspiration.
    - Breathing aided by surfactant in alveolar fluid – reduces surface tension in aveoli.

Breast Feeding

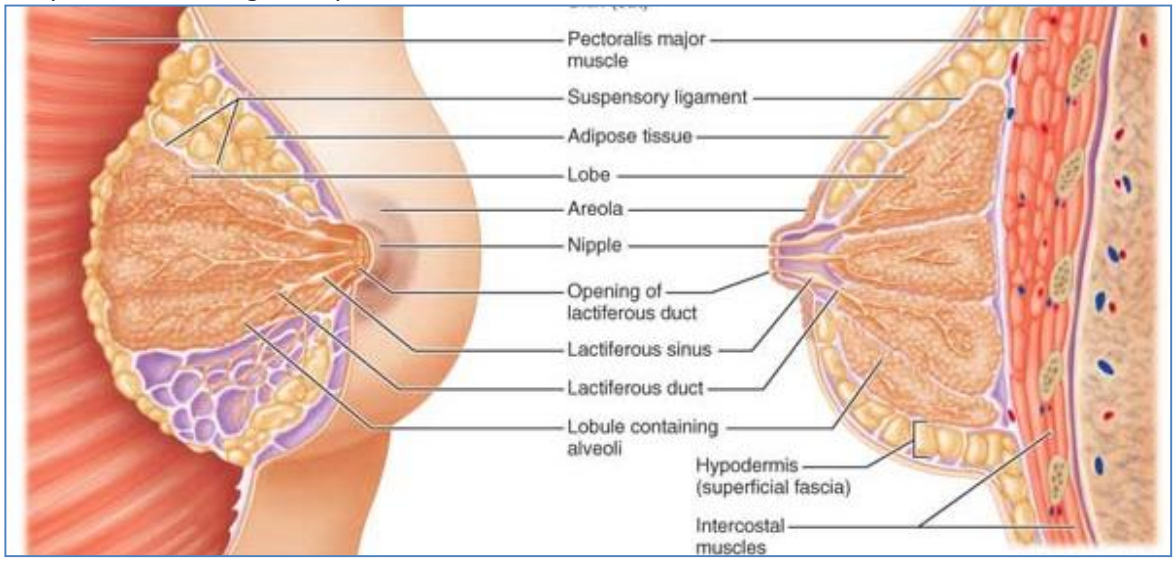
- Lactation = production of milk by mammary glands.
- During late pregnancy, high levels of estrogens, progesterone & lactogen → stimulates hypothalamus – secrete **prolactin-releasing hormone (PRH)** → Ant.Pituitary secretes **prolactin.**
- For first 3 days – colostrum is secreted (less lactose than milk & minimal fat – but contains more protein, vit.A & minerals.) – Rich in IgA antibodies.
- After first 3 days – true **milk production** begins.
    - Milk production depends on mechanical stimulation of nipples – sucking infant.
        - Stimulates hypothalamus → secretes **PRH** → burst of **prolactin** from Ant.Pituitary →stimulates milk production for the next feeding.
        - Also stimulates hypothalamic→post.pituitary release of **oxytocin** – causes the **let down reflex**, the actual **ejection of milk** from alveoli in both mammary glands.
            - Oxytocin also causes uterus to contract → to return to normal size.

Puerperium: (4-6 weeks post-partum)

- Mother's body readjusts to the loss of the foeto-placental unit

Breast Structure:

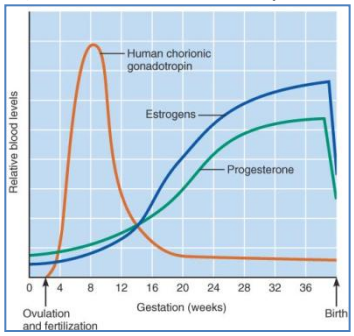
- Mammary glands exist in both sexes – only functional in females
- Contained within the breast – within the hypodermis (superficial fascia), anterior to pectoral muscles of the thorax.
- Areola – ring of pigmented skin surrounding nipple – contains large sebaceous glands (stop chapping)
- Nipple – protrudes from centre of areola
- Each gland consists of 15-20 lobes that open at the nipple.
    - Padded and separated from each other by conn.tissue (suspensory ligaments) and fat
    - Within the lobes are smaller lobules – containing glandular alveoli – produce milk during lactation.
- Compound alveolar glands pass milk into the lactiferous ducts →accumulates in a lactiferous sinus.



SPECIFIC OBSTETRIC NOTES:
PHYSIOLOGY OF PREGNANCY

PREGNANCY:

- **Aetiology:**
  - Unprotected Sex
- **Physiology:**
  - **Ovulation:**
    - Ovulation @ Day 14
    - Unfertilised Oocyte is viable up to 24 hrs after ovulation.
  - **Intercourse:**
    - Sperm are viable inside female for up to 2 days.
    - :. Fertilisation has a 3day window = Sex between Day 12-15.
  - **Fertilisation:**
    - Sperm fuses with Oocyte → Forms a "Zygote"
    - Mitosis Begins → Forms Blastocyst
  - **Implantation of Blastocyst:**
    - 6-7days after ovulation (Ie. Day 20-21)
    - Trophoblast Cells Bind, Proliferate, Invades & Burrows into to Endometrium.
    - Blastocyst becomes completely buried in the Endometrium, surrounded by Lacunae of Blood. (Day 25)
  - **Amenorrhoea:**
    - Trophoblasts secrete B-HCG (Human Chorionic Gonadotropin) → Promotes Corpus Luteum to continue secreting Oestrogen & Progesterone (Ie. Prevent Menstruation)
    - Trophoblasts → "Chorion" (continues secreting B-HCG)
      - **(NB: B-HCG *Below* expected for dates could = Ectopic/Abortion/Wrong Dates)**
    - Chorion → Placenta (Takes over from Corpus Luteum → Secretes ↑↑↑Oest & Prog)



  - **Embryogenesis:**
    - **Ectoderm** → Nervous System, Skin & Amnionic Sac
    - **(Mesoderm:** Formed later during Fastrulation → Becomes Heart, Vessels, & All Else.**)**
    - **Endoderm** → GI Mucosa, Resp.Tract Mucosa, and Urogenital Mucosa.

- **Maternal Changes in Pregnancy:**
  - **Reproductive System:**
    - ↑Vascularity & Size of Reproductive Organs
      - Vagina develops purplish hue (Chadwick's sign)
      - Breasts Enlarge + Darken.
    - Placenta Secretes **Relaxin** → Relaxes Pelvic Ligaments + Pubic Symphysis
    - Placenta Secretes **Human Chorionic Somatomammotropin (hCS)** →
      - → Breast Maturation.
      - → ↑Maternal Insulin Resistance → Glucose-Sparing Effect → More Nutrients for Foetus. (NB: Can cause Gestational Diabetes Melitis)
  - **Urinary System:**
    - Bladder Compression → Urinary Frequency
    - ↓Sphincter Tone (Progesterone → SM Relaxation) → Incontinence & ↑UTIs
    - Increased plasma volume → 50% ↑GFR → Polyuria
    - Glycosuria (Filtration outpaces Reabsorption)

- **Respiratory System:**
  - 20% ↑Oxygen Consumption
  - ↓Diaphragm Mobility → ↓Total Lung Capacity
    - Tachypnoea
    - ↑Rib Flaring
    - ↑Accessory Muscle Usage
  - (NB: No Changes in ABGs)
- **Cardiovascular System:**
  - ↑Cardiac Output Required → Hyperdynamic Circulation. Achieved by:
    - ↑HR
    - ↑Plasma Volume (25-40%) → Hypertension
  - Uterus can compress on Iliac Veins → ↓Venous Return from Legs
    - → Varicose Veins.
    - → Haemorrhoids
    - → Leg Oedema
- **Haematological System:**
  - ↑Plasma Volume (25-40%) → Spurious Anaemia (due to Haemodilution)
  - Hypercoaguable State (↑Almost ALL Coagulation Factors; + ↓Antithrombin-III)
    - → ↑DVT/PE Risk
  - ↑Leukocyte Count (But Impaired Function) → Relative Immunodeficiency
  - Gestational Thrombocytopaenia (Consumptive)
- **GI System:**
  - Morning Sickness – due to ↑Oestrogen & ↑Progesterone.
  - GIT Displaced Superiorly – (Eg. Appendicitis will present higher than usual)
  - ↑Intra-Abdominal Pressure → Heartburn/Reflux
  - ↓GIT Tone (Progesterone):
    - ↓Lower-Oesophageal Sphincter Tone → Heartburn/Reflux
    - ↓GI-Motility (More time available for Digestion & Absorption) → Constipation.
    - ↓Gallbladder Tone → Gallstones.
  - ↑ALP (Alk-Phos) – Excess Produced by the Placenta (Not Liver).
- **Endocrine:**
  - **Pancreas:**
    - ↑Insulin Secretion → To compensate for HCS-Mediated Insulin Resistance.
    - (NB: Maternal Insulin does NOT cross the Placenta – Bub makes its own Insulin)
  - **Thyroid:**
    - HCG → Stimulates Thyroid (In addition to Normal TSH)
      - ↑Size & Vascularity
      - ↑Thyroid Hormone Production
      - (But normal T3/T4 levels due to ↑Thyroid-Binding Globulin)
  - **Parathyroid:**
    - ↑PTH → ↑Bone Ca-Resorption → ↑Ca Available to Foetus's Bones.
  - **Adrenals:**
    - ↑ACTH Secretion →
      - ↑Aldosterone → Water Retention → Oedema
      - ↑Cortisol → Relative Immunodeficiency (Protects Foetus from Mum)
  - **Pituitary:**
    - ↑Prolactin Secretion → Lactation

- **Clinical Features:**
  - **Symptoms:**
    - Amenorrhoea
    - Nausea/Vomiting
    - Heartburn/Reflux
    - Breast Tenderness
    - Urinary Frequency
    - Constipation
    - Fatigue
  - **Signs:**
    - Softening of the cervix (4-6wks)
    - "Chadwick's Sign" - Bluish Hue of Vagina (Engorgement of pelvic vasculature)
    - Uterine Enlargement
    - Breast Enlargement & Darkening
  - **Diagnosis:**
    - **B-HCG:**
      - +ve in *Serum* @ 9days post-conception
      - +ve in *Urine* @ 28days post-conception
    - **Transvaginal USS:**
      - 5 wks: Gestational Sac Visible
      - 6 wks: Foetal Pole Visible
      - 7-8wks: Foetal Heartbeat Detectable
    - **Trans-Abdominal USS:**
      - >6 wks: Pregnancy Detectable

Fertilisation – Embryology - Pregnancy

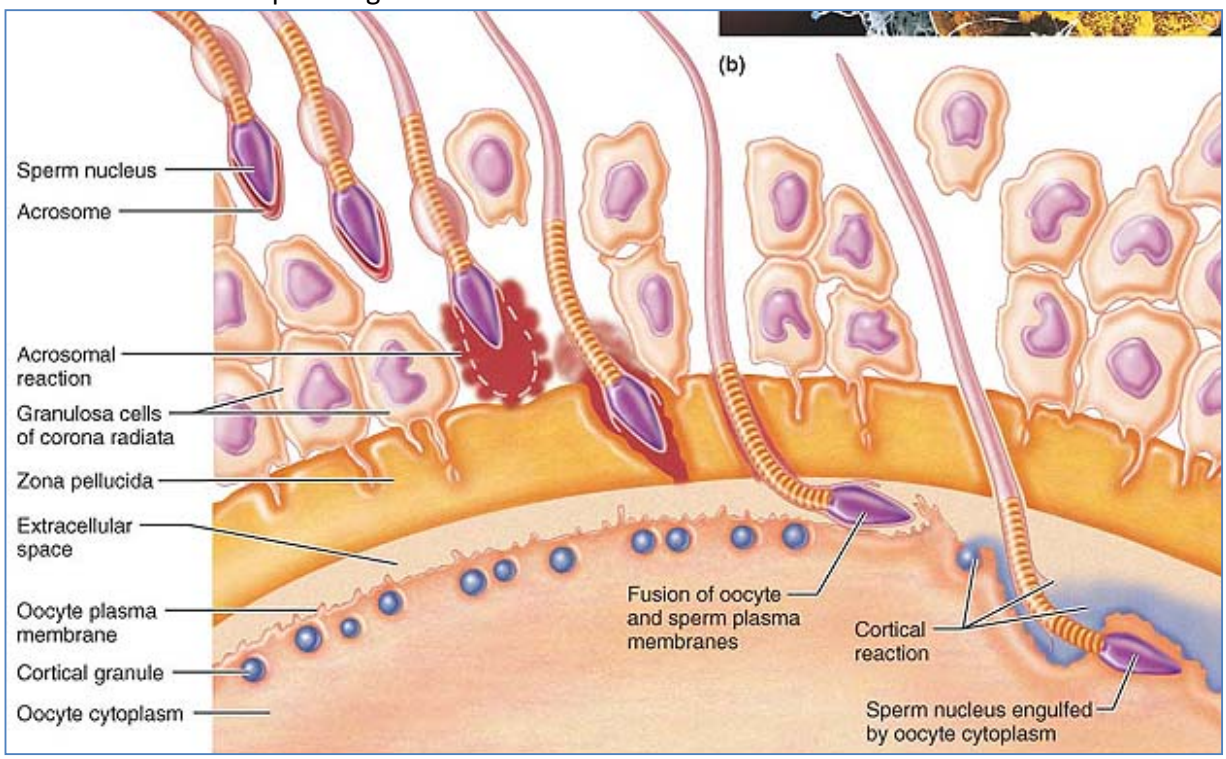
From Egg → Zygote:

Viability:

- Oocyte is viable up to 24 hrs after ovulation.
- Sperm are viable inside female for up to 2 days.
- 3 day viable window = Sex between 2 days before ovulation and 1 day after.

Fertilisation:

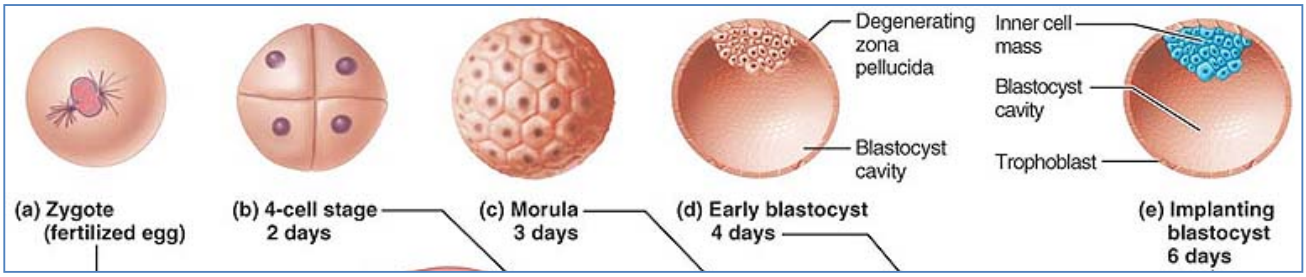
- Only a few thousand sperm reach the uterine tubes (site of fertilisation)
- Sperm must be capacitated (increased mobility + fragile acrosomal membranes) before they can penetrate the oocyte. (caused by secretions of female tract)
- Oocyte is encapsulated in a "corona radiata" and the deeper "zona pellucida", which both need to be breached before the oocyte itself can be penetrated by a sperm.
  1. Sperm penetrates the corona radiata, assisted by hyaluronidase, and corona radiata cells fall away from the oocyte.
  2. Sperm head binds to zona pellucida, which triggers the release of acrosomal enzymes → digest holes through the zona pellucida. (hundreds of sperm are needed to expose the oocyte membrane)
  3. Single sperm fuses with oocyte membrane, its nucleus is pulled into the oocyte cytoplasm.
  4. Ca^{2+} is released by oocyte ER, causing cortical reaction→cortical granules in oocyte membrane release enzymes (zonal inhibiting proteins) that destroy remaining sperm receptors + detach all sperm still bound.
  5. Secondary oocyte completes meiosis II, forming ovum pronucleus + 2nd polar body.
  6. The lucky sperm loses its tail + midpiece, & its nucleus swells to 5x. → forms male pronucleus.
  7. These 2 pronuclei fuse and form the zygote. (moment of fertilisation)
  8. Mitosis of conceptus begins.



Zygote → Blastocyst Implantation

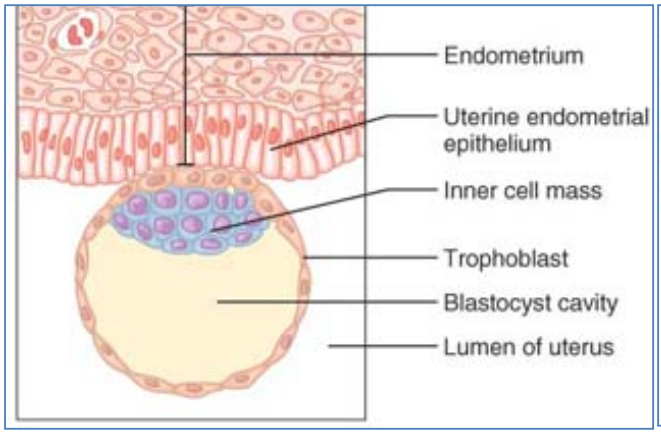
Cleavage & Blastocyst Formation:

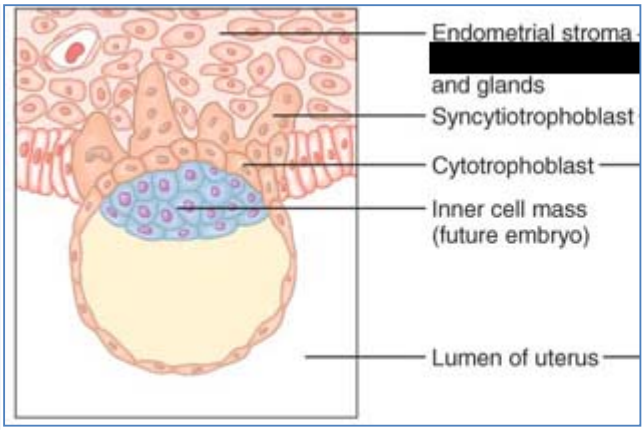
- Rapid series of mitotic divisions without intervening growth. → produces many smaller cells collectively called the **morula**.
- Cells of the morula compact (forming the inner cell mass), the zygote accumulates fluid and trophoblast cells develop around the inside of the zona pellucida. (Early blastocyst)
- Zona pellucida starts to break down and the blastocyst "hatches" from it.

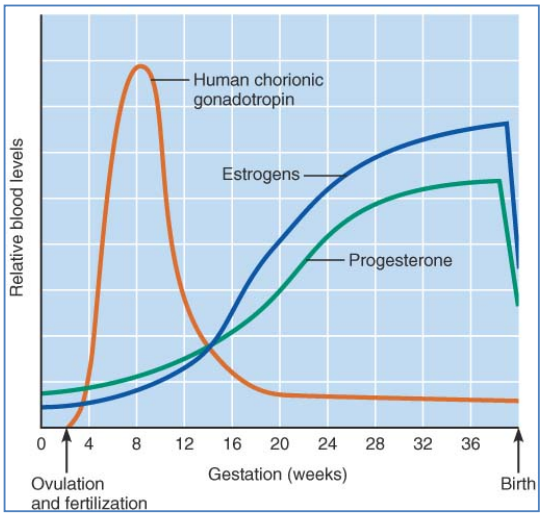


Implantation (6-7 days after ovulation)

- Endometrium must be receptive (at the height of the secretory stage) (caused by surging estrogens and progesterone)
- Trophoblast cells bind to the extracellular matrix of endometrial cells on the inner uterine wall. (implantation may be attempted several times where the blastocyst detaches from the immature Endometrium and floats south to try again)
- Endometrium thickens, blood vessels become more permeable/leaky and inflammatory cells invade the area.
- Trophoblast cells proliferate, forming 2 distinct layers: **cytotrophoblast** (adjacent to inner cell mass) and the **syncytiotrophoblast** (multinucleated mass – invades the Endometrium by digesting uterine cells.)
- At this stage implanted embryo obtains nutrition by digesting endometrial cells.
- Blastocyst burrows through the Endometrium and is surrounded by a pool of blood (**lacuna**). Blastocyst is covered over and sealed off from uterine cavity by endometrial cells.
- Successful implantation takes 5 days (12 days after ovulation – usually time of menstruation)
- To stop mensus, trophoblast cells secrete hCG (Human Corionic Gonadotropin) which promotes Corpus Luteum to continue secreting oestrogen and progesterone.
    - Trophoblast cells differentiate into the **chorion** which continues secreting hCG.
- Chorion later forms the **placenta**, which takes over from corpus luteum & produces shit loads of progesterone and oestrogen.



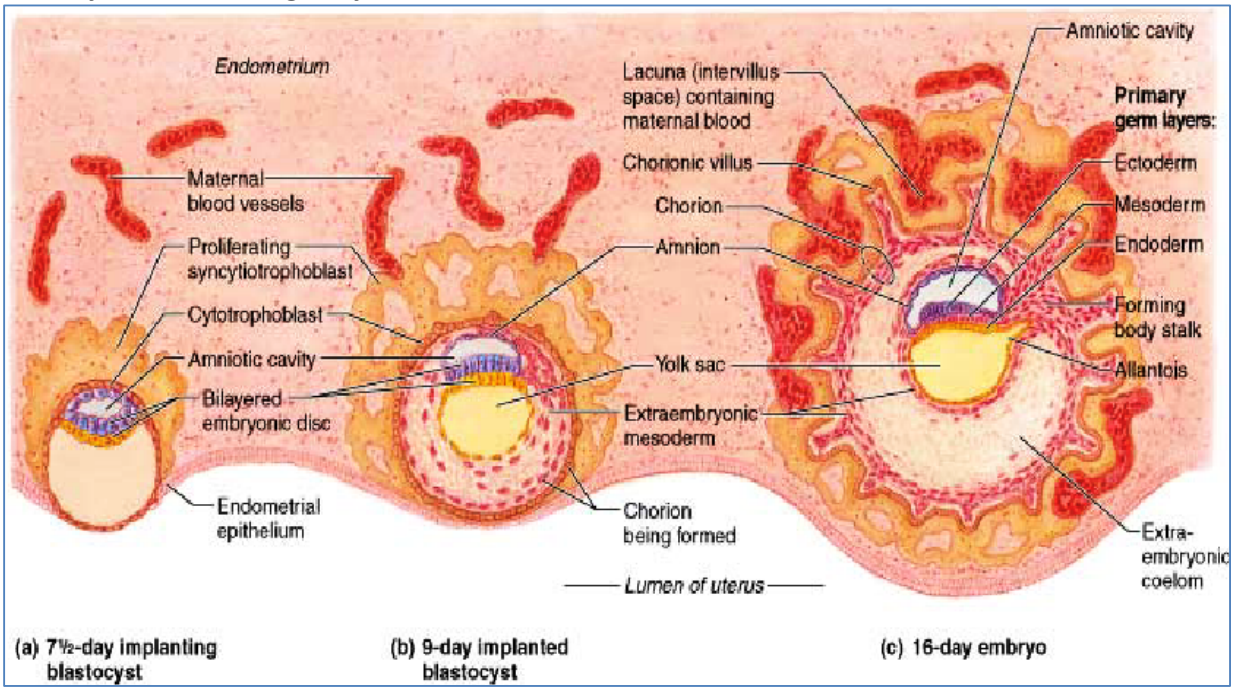




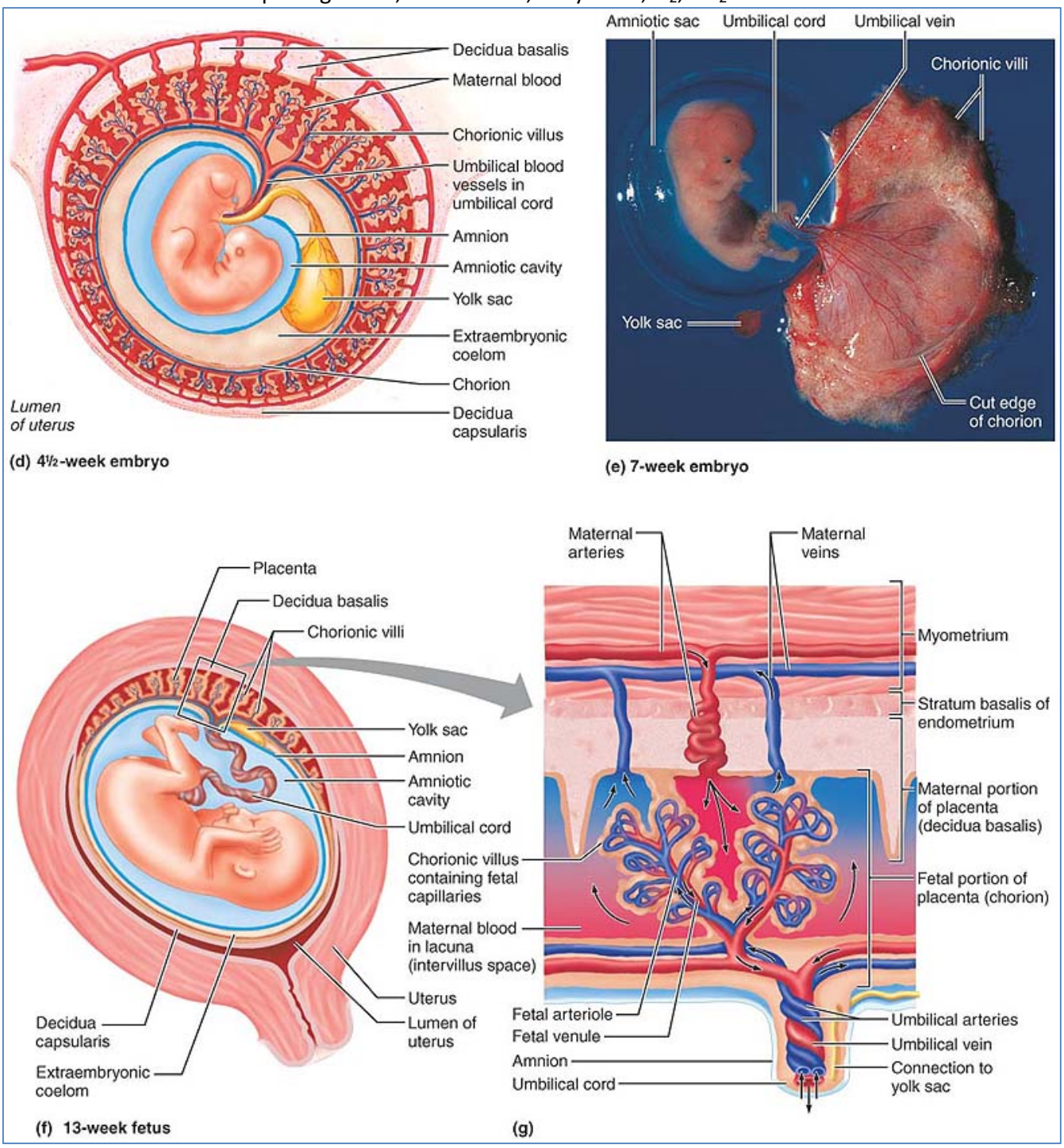
Placentation:

- Cells from original inner cell mass give rise to a layer of **extraembrionic mesoderm** which lines the inner surface of the cytotrophoblast. Together the **cytotrophoblast** and **extraembrionic mesoderm** form the **Chorion.**
- **Germ layers form:** inner cell mass facing blastocyst cavity form a flat disc with 2 layers:
    - **Ectoderm**
    - Forms nervous system and skin during gastrulation
    - – and the amnion formed from the ectoderm. (later fills with fluid → amnionic sack)
    - **(Mesoderm:** formed later during gastrulation – forms heart, blood vessels, conn. Tissue & everything else.**)**
    - **Endoderm**
    - Forms the mucosa (epithelial lining) of the GI tract, respiratory tract, and urogenital tract.
    - - and the yolk sack formed from the endoderm. (later forms part of the gut/digestive tube and produces the earliest blood cells & blood vessels.
    - – and the allantois – structural base of the umbilical chord.

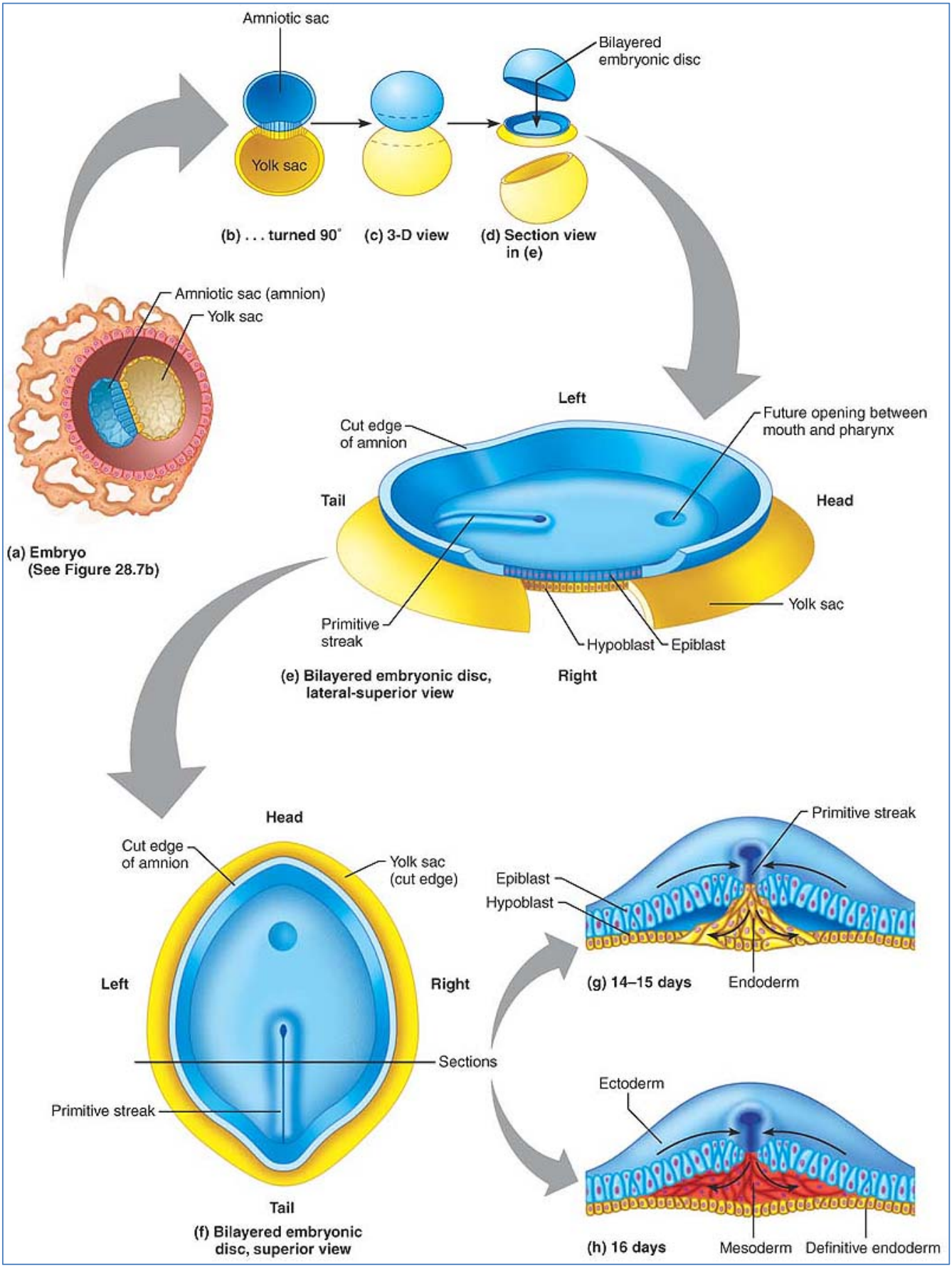
By 8 weeks all organ systems are formed.

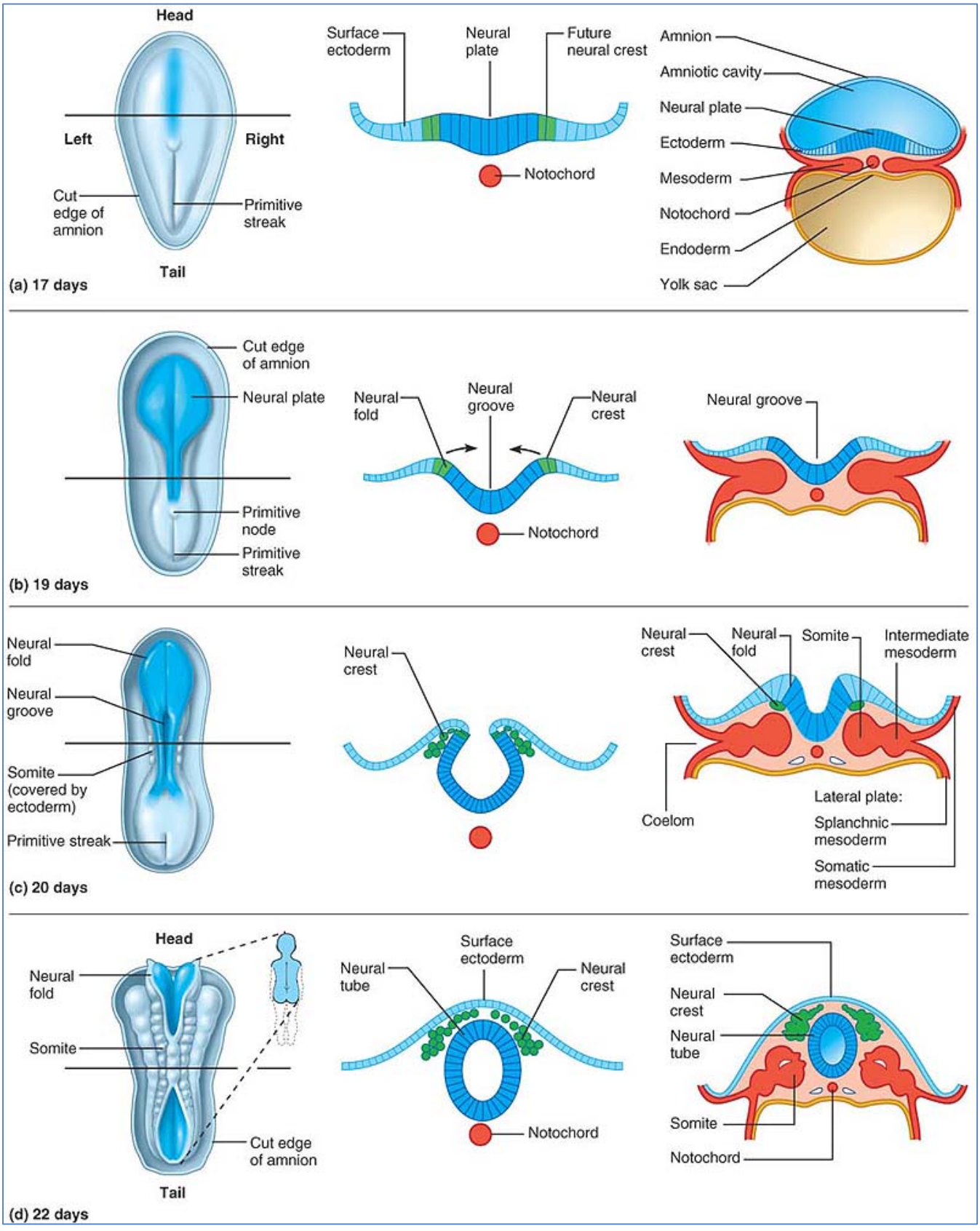


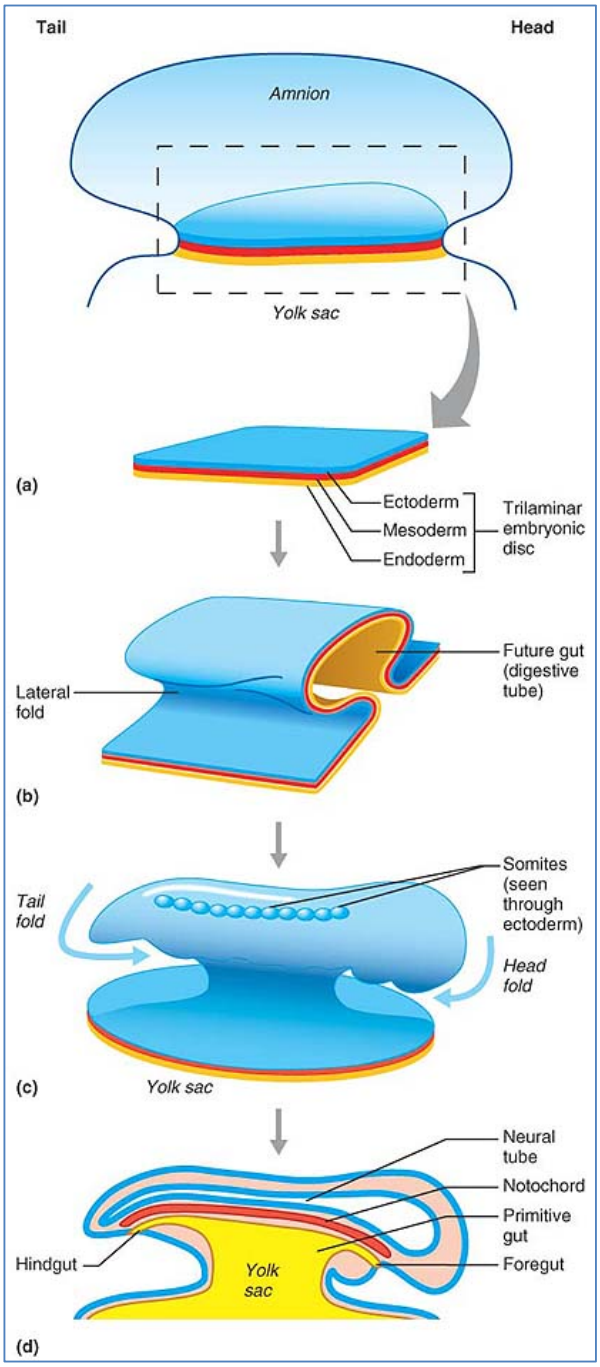
- **Chorion** develops fingerlike **chorionic villi** which are in contact with maternal blood and are highly vascularised.
- Large blood-filled **lacunae** form in the Endometrium and the villi become totally immersed in blood.
- Around the chorionic villi, the Endometrium splits into 2 parts: **the decidua basalis** (basal side/ stratum basalis [side of umbilical chord]) and **the decidua capsularis** (luminal side of embryo)
    - Decidua capsularis expands to accommodate foetus & villi are compressed & degenerate.
    - Decidua basalis forms the eventual placenta and villi increase in number and branch even more profusely.
    - Placenta functions as a nutritive, respiratory, excretory and endocrine organ.
        - Transports glucose, amino acids, fatty acids, O_2, CO_2 and other wastes.

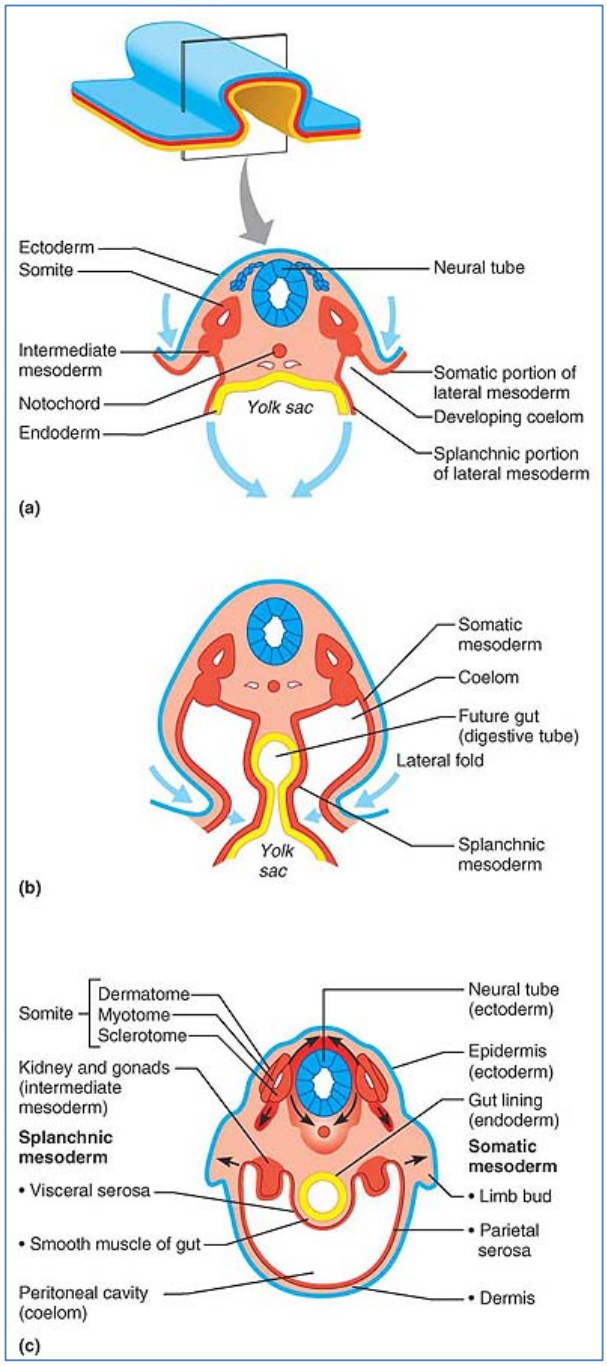


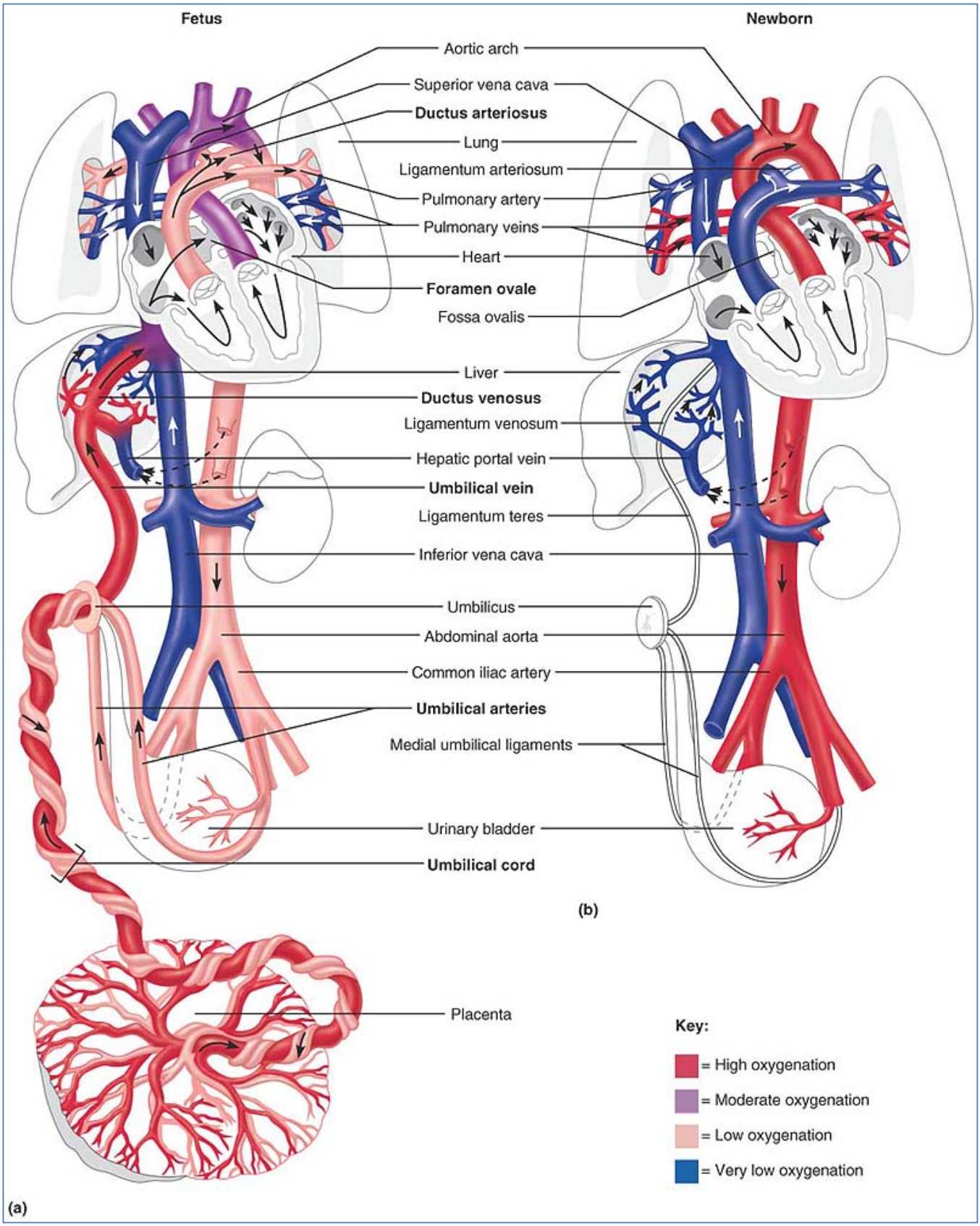
Embryonic Development











SPECIFIC GYNAECOLOGY NOTES:
EMERGENCY CONTRACEPTION & ABORTION

Emergency Contraception:

- **Goal:**
  - Last-chance contraception if current contraceptive failed (Eg. Broken Condom)
- **Timing:**
  - ASAP after unprotected sex. (<120hrs / 5days)
  - **NB: Completely ineffective Post-Implantation**
- **Methods:**
  - **#1 Prog-Only:** (AKA: ***"Plan-B"***) Single Dose – 1.5mg – (Effective <72hrs & Fewer Side Effects)
    - ***NB: NOT Effective After Implantation!!***
  - **Copper IUD:** Best efficacy; But inconvenient & invasive.
    - ***Still Effective After Implantation***
  - (NB: Combined [AKA: "Yupze Regimen"] <72hrs is Less Effective than "Plan-B" & has ↑SE's)

Abortion Pre-Requisites:

1. **Counselling on Alternatives (Eg. Adoption)**
2. **Informed Consent**
3. **Comprehensive History**
4. **Discuss Contraception *After* Abortion**
5. **STI-Screen & Education**
6. **Antibiotic Prophylaxis Prior to Abortion**

Abortion:

- **Early Medical Abortion (<6wks):**
  - **STAT DOSE - *Mifepristone/RU486*:**
    - *Progesterone Receptor Agonist* → Prevents Endometrium from supporting Fertilised Egg.
    - Effective <63days (7wks) since last period.
    - No need to come into Hosiptal
  - **Or Methotrexate – (Used more in Ectopic Pregnancies)**
- **Early Surgical Abortion (<14wks):**
  - **Dilation & Suction Curettage:**
    - Available up to 14wks Gestation
- **Late Medical Abortion (14-20wks):**
  - **STAT DOSE - *Mifepristone/RU486*:**
    - *Progesterone Receptor Agonist* → Prevents Endometrium from supporting Fertilised Egg.
    - Effective <63days (7wks) since last period.
  - **48HRS LATER – *Vaginal Misoprostol*:**
    - *Synthetic Prostaglandin* → Ripens Cervix & Induces Labour
    - + Analgesia, +/- Anti-Emetics, +/- Anti-D-Ig in Rh-Negative Mothers
    - Very Safe – Small risk of bleeding; <1:100000 risk of death.

SPECIFIC OBSTETRIC NOTES:
ANTEPARTUM HAEMORRHAGE

Case:

- 23yo Mrs. Smith
- G2P1
- c/o PV bleeding
- LMP (Last menstrual period) 6/52 ago

Further Hx Questions:

- How often?
- How much?
- Colour (Bright red = Frank; Brown = Old)
- Consistency (Clots = Large volume bleeding)
- Pain? (NILDOCARF).
    - NB: shoulder-tip pain = Haemoperitoneum → Diaphragmatic Irritation
- Any chance of pregnancy?
- Recent trauma/intercourse
- Last pap smear?
- Any IUDs?
- Menstrual History
    - Pain?
    - Regular?
    - Age of menarche?
    - Volume?
    - OCP?
- Chance/Hx of STIs
- (PMH)
- (PSH)
- (Meds/Allergies)
- (SocHx)
- (FamHx)

DDX:

- **Top 3:**
    - Ectopic Pregnancy / Heterotopic Pregnancy
    - Spontaneous Abortion / Miscarriage
    - Molar Pregnancy (Requires surgery)
- **Others:**
    - Gestation Trophoblastic Disease
    - Cervical Cancer
    - Vaginal Trauma
    - Vulval varicosities
    - Haemorrhoids
    - (NB: Not placenta praevia – only develops syx in 3rd trimester)

Examination:

- **Vitals**
- **General Appearance**
- **Abdo:**
    - Distension
    - Scars
    - Tenderness
    - Guarding
    - Rebound
    - Bowel Sounds
- **PV Exam:**

- Inspection
  - Skin
  - Infections
  - Bleeding
  - Prolapse
- Speculum:
  - Cervix
    - Normal?
    - Os open?
    - Trauma?
  - Vaginal Walls
- Bimanual:
  - Cx – Long, Post/Ant, Tender
  - Uterus – Anteverted/Retroverted, Size, Tender?
  - Adenexium – Mass/Tenderness

Ix:

- FBC (Hb/WBC)
- BHCG (Pregnant?) (Positive in this case)
- MSU
- Blood group (Rhesus Status & ?G&H)
- USS

USS Results:

- Thick Endometrium
- R-Adenexal Mass (3 cm)
- Some free fluid in pelvis

Dx = Ectopic Pregnancy in R-Fallopian Tube

Mx:

- Surgical Options
  - Salpingostomy (Better for preserving fertility; requires B-HCG followup; can have persistent ectopic)
  - Salpingectomy (Better for ↓risk of another ectopic; more definitive treatment; no followup required
- Medical Option – Methotrexate:
  - Once-off injection
  - Still 50% chance of rupture
  - 75% success rate (abortion rate)
  - BUT – Cytotoxic Side effects (Nausea, Vomiting, Hair loss, Fatigue, etc)
- Expectant Management (Watch and wait)
  - If the B-HCG is <120.
  - 48hrly B-HCG until resolved (Some spontaneously resolve)

Baby Checks: The Process:

DRABCS

Background History:

- **Neonatal History**
- **Mum's/Bub's Blood Group** (*Beware the Rh-Negative Mother with a Rh-Positive Baby)
- **Obstetric History** (Gestational Diabetes, Pre-Eclampsia)
- **Method Delivery** (Natural/Forceps/Suction, Caesarean)
- **Risk Assessment for Sepsis**

Vital Signs:

- **P:** **Bradycardia (Often responds to ventilation) → Resuscitate
- **R:** **Signs of Respiratory Distress (Treat cause) → Resuscitate
- **T:** **Fever could = Infection (Beware *Group B Strep*, and *Chlamydia*) → Investigate & ABs

General Inspection:

- **Colour** (Pink = Good; Yellow/Jaundice = Often Normal; Blue/Cyanotic = Bad)
- **Distress** (Respiratory/Other)
- **Red Reflex of Retina** (Ensure the baby has a retina)
- **10 Fingers, 10 Toes**
- **Patent Anus** (NB: Presence of Meconium ≠ Patent Anus; Beware any Ano-Vaginal Fistulas)
- **Normal Genitalia**

Palpation:

- **Fontanelles** (Ant, Post & 2x Laterals; Beware Bulging = Hydrocephalus, & Sunken = Dehydration)
- **Cleft Palate**
- **High-Arched Palate** (Marfans)
- **Rooting Reflex** (Stroke Cheek → Baby Turns Head to That Side)
- **Palmar Grasp Reflex**
- **Femoral Pulses** (120-180bpm)
- **Hip Examination** (Push down onto hips through the femur with 90^{o} Hip Flexion, then abduct both hips. Feel for clicking/dislocation)
    - "Barlows Hip" = Hips dislocate with the above manoeuvre
    - "Ortalana Hip" = Hip/s are *already* dislocated
- **Muscle Tone** (Beware a Floppy Baby)
- **Spine – Neural Tube Defects** (Spina Bifida)

Auscultation:

- **Heart Sounds:**
    - *Rate
    - Murmurs are Normal
    - ?Dextrocardia
- **Lung Sounds:**
    - Crackles
    - Wheezes
    - Stridor
- **Bowel Sounds:**
    - Present
    - Absent

Measurements:

- **Head Circumference** → Record on Growth Chart (*Bad if >90^{th}/<10^{th} Centile)
- **Length** → Record on Growth Chart (*Bad if >90^{th}/<10^{th} Centile)
- **Weight** → Record on Growth Chart (*Bad if >90^{th}/<10^{th} Centile)
- **BSL**

SPECIFIC OBSTETRIC NOTES:
CHORIOAMNIONITIS

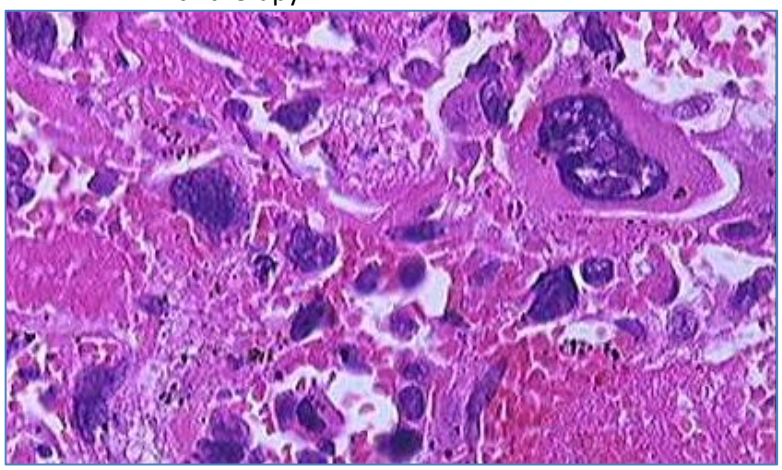
CHORIOAMNIONITIS:

- **Aetiology:**
  - Placental Infection
  - **Risk Factors:**
    - Premature Birth
    - PPROM
    - PROM
    - Prolonged Labour
- **Pathogenesis:**
  - Infection & inflammation of the Chorionic Membrane & Villi due to:
    - Ascending Infection from Vagina (Vaginal Flora, Candida, etc)
    - Blood-Spread from Systemic Infection (HSV, Syphilis, Toxoplasmosis, Rubella, CMV)
- **Morphology:**
  - **Macro:**
    - May have Abscess Formation
  - **Micro:**
    - Inflammation of Chorionic Plate (WBCs)
    - Vasculitis of Umbilical Vessels
    - Infarctions
- **Clinical Features:**
  - **Maternal Symptoms:**
    - Fever
    - Uterine Tenderness
  - ****Neonatal Complications – (TORCHS Syndrome: Toxoplasmosis, Rubella, CMV, Herpes, Syphilis):**
    - Neonatal Sepsis
    - Neonatal Asphyxia
    - Microcephaly
    - Brain Damage/Hearing Impairment
    - Neonatal Organomegaly
    - Miscarriage/Death
- **Treatment:**
  - **Antibiotics**
  - **+ Induction of Labour**
- **Prognosis:**
  - Low maternal mortality if treated.
  - Significant Risk of Neonatal Complications.

SPECIFIC OBSTETRIC NOTES:
CHORIOCARCINOMA (PLACENTAL CANCER)

CHORIOCARCINOMA (MALIGNANT):

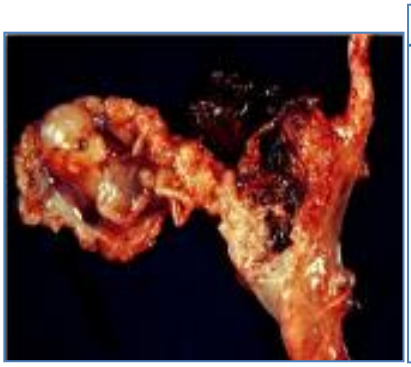
- **Aetiology:**
  - Risk Factors – Extremes of age <20, >40, previous abortion, abnormal gestation
  - May be De-Novo (Primary) or may progress from a Complete Mole (Secondary).
- **Pathogenesis:**
  - **May be De-Novo (Primary) or may progress from a Complete Mole (Secondary).**
    - High Grade Priamry Malignancy of the Trophoblasts
    - May evolve Secondary to an Invasive/Complete Hydatidiform Mole
- **Morphology:**
  - **Macro:**
    - Invasive
    - Haemorrhagic
    - Necrosis
- **Clinical Features:**
  - Irregular Vaginal Bleeding
  - Uneven Swelling of Uterus (Mass)
  - Abdominal/Pelvic Pain
  - **Diagnosis:**
    - Rising hCG
    - Abdo US → Abdo CT
  - **Metastasis to Lungs is common → Haemoptysis**
- **Treatment:**
  - **Surgical Excision**
  - **+ Chemotherapy (Methotrexate)** – Good Prognosis
- **Prognosis:**
  - Types:
    - Gonadal (in the ovary – Not related to gestational) – Poor prognosis
    - Gestational (in the uterus – associated with pregnancy) – Good Prognosis – 100% cure rate with therapy.

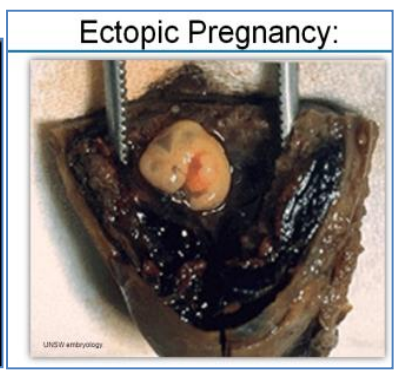


SPECIFIC OBSTETRIC NOTES:
ECTOPIC PREGNANCY

ECTOPIC PREGNANCY:

- **Aetiology:**
  - 50% Idiopathic
  - **Risk Factors:**
    - Obstruction
    - PID
    - Fallopian Stricture
    - IUD
    - Endometriosis
- **Pathogenesis:**
  - Implantation outside the uterus (Often within the fallopian tube wall)
- **Morphology:**
  - **Macro:**
    - 90% occur in Fallopian Tubes
    - May occur in the Abdomen
  - **Micro:**
    - Normal placental infiltration – Just in the wrong place.
- **Clinical Features:**
  - 1% of pregnancies
  - **Symptoms:**
    - May mimic a normal early pregnancy – (Missed Periods, Breast Tenderness, Nausea)
    - *Sharp, Stabbing Pain (Pelvic/Abdominal)
    - *Vaginal Bleeding/Spotting
    - **Peritonitis/Shoulder Pain if Rupture = MEDICAL EMERGENCY
  - **Diagnosis:**
    - B-hCG (Pregnancy Test)
    - Abdominal Ultrasound – (If scan is –Ve, re-test hCG & re-scan every 2-3 days until foetus can be located)
  - **Complications:**
    - **Rupture → Massive Intraperitoneal Haemorrhage → Shock → **Death
    - Spontaneous Abortion
    - Chorioamnionitis
- **Treatment:**
  - If early – Medical Abortion (Methotrexate + Misoprostol)
  - If later – Surgical Abortion (Laparoscopic Salpingotomy)
- **Prognosis:**
  - Good if treated
  - May → Some infertility.





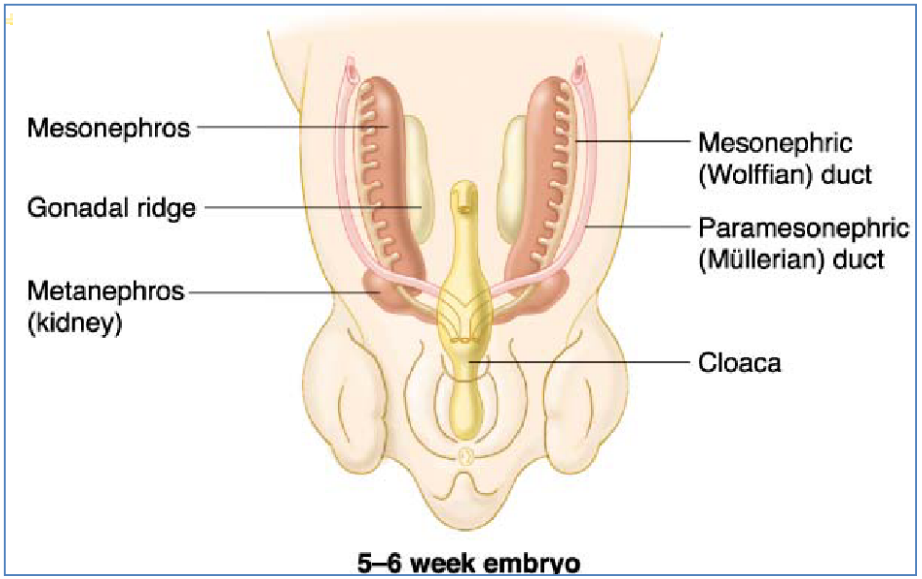
Endocrinology and Reproductive Processes
Reproductive Processes

Gametogenisis:

- The formation of gametes (spermatogenisis / oogenisis)
- Largely regulated by hormonal/endocrine system.
- A process of **meiosis**
- **Male:** Mitotic division of spermatogonia and entry to meiosis is continuous from onset at puberty until death.
- **Female:** Meiotic division is discontinuous – begins in embryo, lies dormant once born & is completed upon fertilisation by a sperm.
    - Events are cyclic between puberty and menopause – **menstrual cycle.**
- Gametes are *haploid* cells (½ Chromosome number – 23 –)
    - 22 pairs of somatic chromosomes
    - 1 pair of sex chromosomes (X, Y)
        - Sperm contains X & Y.
        - Egg only contains X & X.
        - **Combinations:**
            - XY = produces a male
            - XX = produces a female.
            - Y is the **sex determining** chromosome.

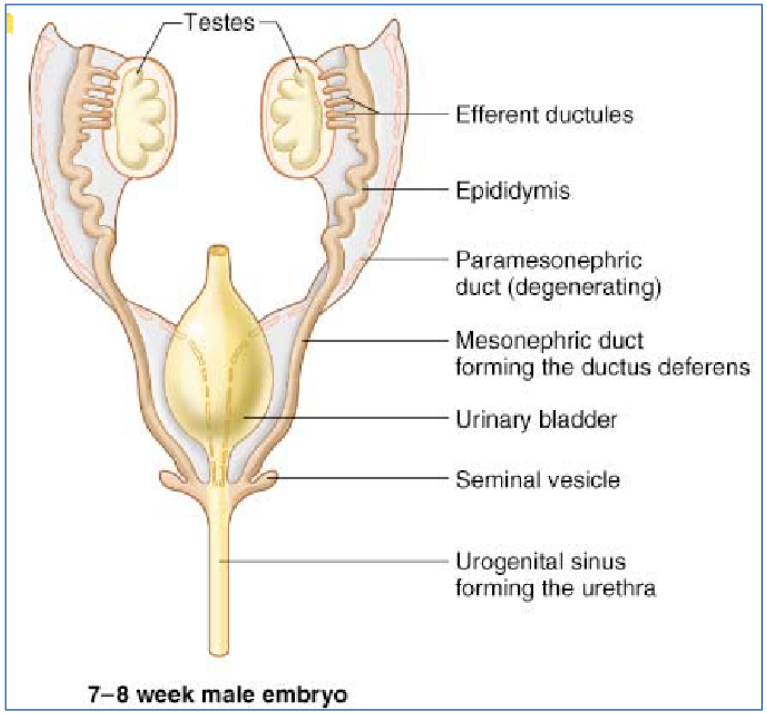
Embryo Sexual Differentiation:

- During Wk 5 of embryonic growth gonadal tissue develops into a **gonadal ridge.**
    - Primordial germ cells migrate into gonadal ridge which then develop into ovaries or testes depending on DNA.
    - Mesonephric (Wolffian) ducts = future male ducts
    - Paramesonephric (Mullerian) ducts = future female ducts.
        - Both empty into a common chamber = the **cloaca**
    - At this stage the embryo is said to be sexually indifferent as the gonadal ridge can develop either way.

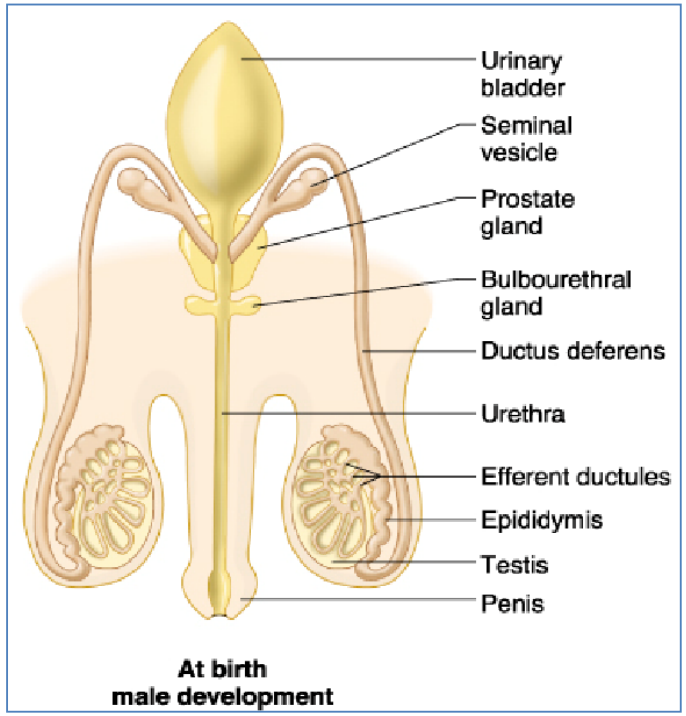


5–6 week embryo

- **Male:**
  - Y = The **sex determining** chromosome - causes the gonadal ridge to develop into testes. (ie. Females are the default sex)
  - Testes then produce testosterone
    - Testosterone causes the Mesonephric (Wolffian) duct to develop into the male tract.
  - Also produces AMH (anti-mullerian hormone)/MIH (mullerian inhibiting hormone) which causes the Paramesonephric (Mullerian) duct to degenerate.
  - External genitalia:
    - Penis develops from the genital tubercle.
    - Scrotum develops from the labiascrotal swellings.

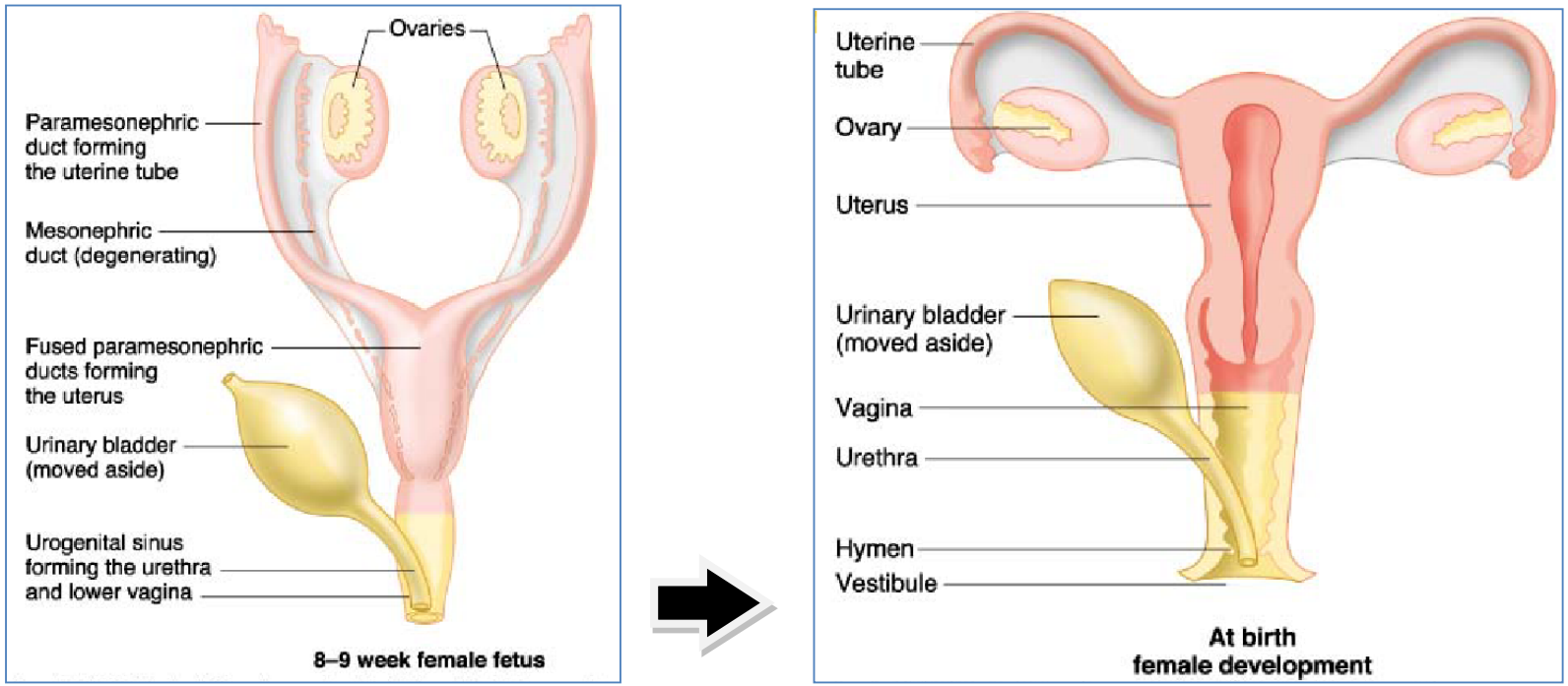


7–8 week male embryo

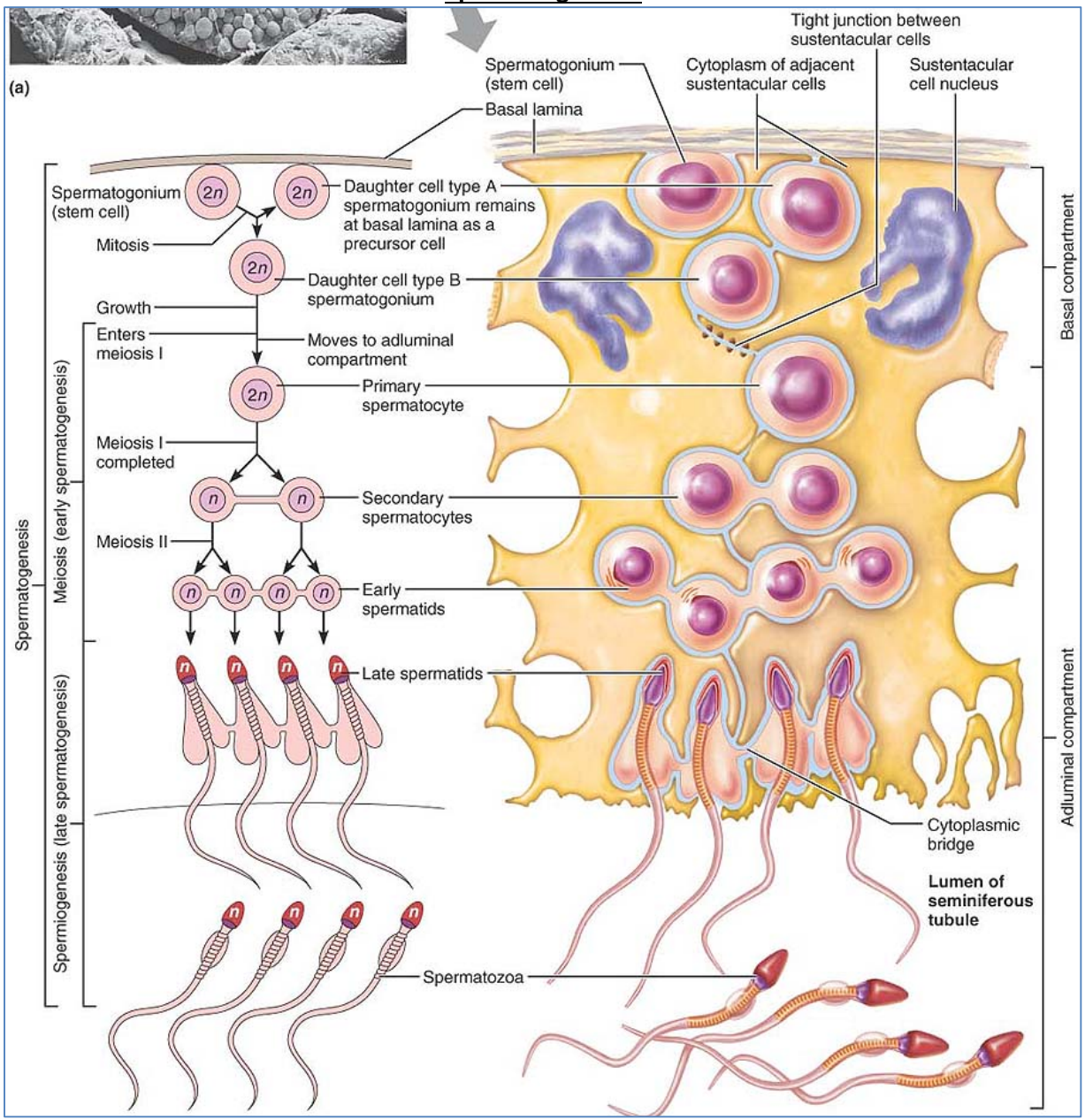


At birth male development

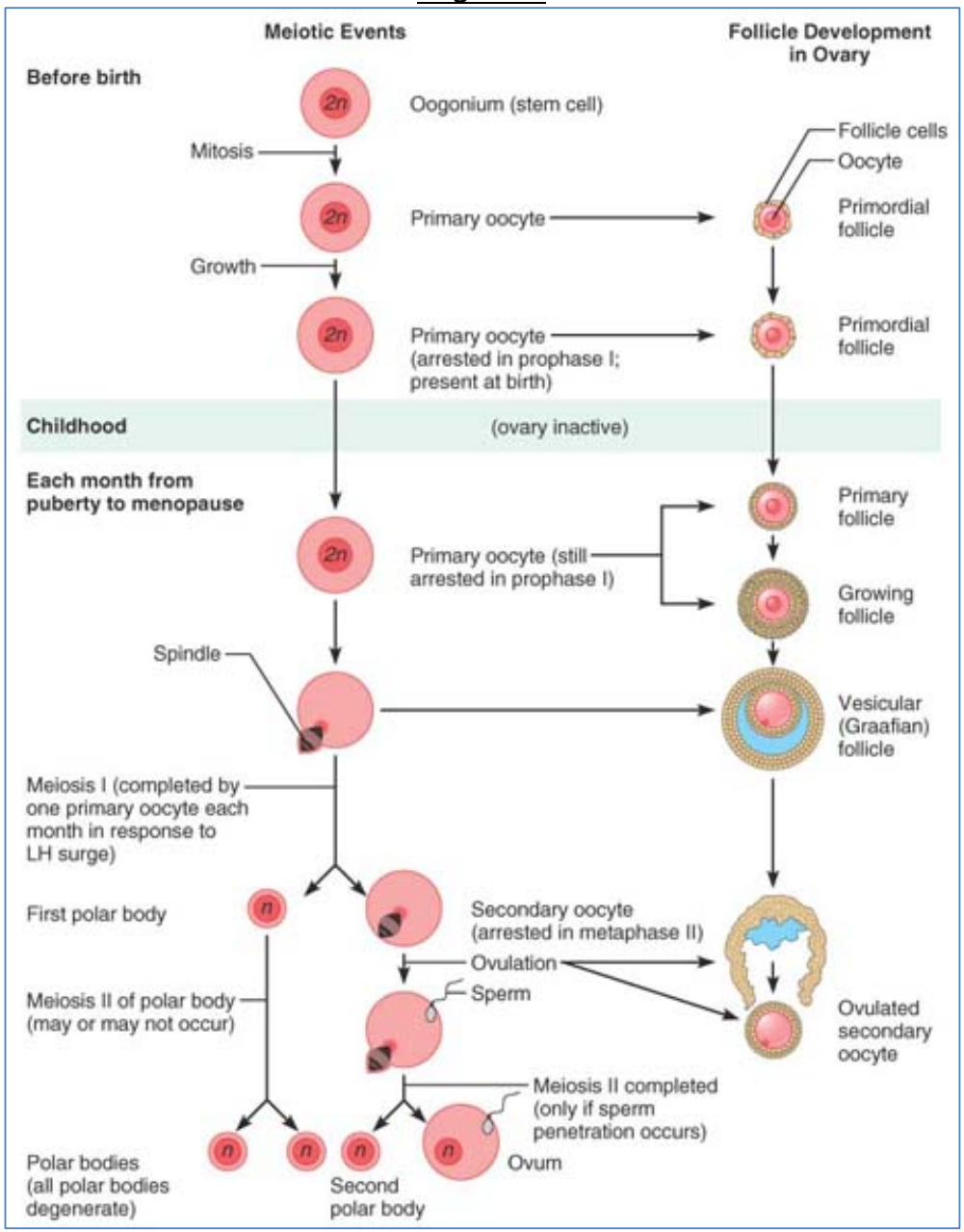
- **Female:**
  - By default, if there is no 'Y' chromosome → no testes, the female tract will develop.
    - The gonadal ridge develops into immature ovaries.
    - Without testosterone, the Mesonephric (Wolffian) ducts will degenerate.
    - The cortical (outer) part of the immature ovaries forms follicles.
    - Without AMH/MIH, the paramesonephric ducts differentiate into structures of the female duct system.
  - External genitalia:
    - glans clitoris develops from the genital tubercle.
    - Labia minora forms from the urethral groove.



Spermatogenisis:



Oogenisis:



SPECIFIC OBSTETRIC NOTES:
HYPERTENSIVE DISORDERS OF PREGNANCY

General Info:

- **Definition:**
  - **= "BP ≥ 140/90 on >2 Separate Occasions"**
- **Types – *In Order of Severity:***
  - **Chronic Hypertension:**
    - **= "HTN *Before* Pregnancy OR *Within 1st 20wks*".**
    - (No associated problems; But ↑Risk of → Pre-Eclampsia)
  - **Gestational Hypertension:**
    - **= "HTN occurring *After 20wks*"**
    - (No associated problems; But ↑Risk of → Pre-Eclampsia)
  - ***Pre-Eclampsia (Incl. Chronic HTN with Superimposed Pre-Eclampsia):**
    - **= "HTN in Pregnancy + ANY Sign of Organ Dysfunction"**
      - Eg. Kidney – (Proteinuria, ↑Creatinine)
      - Eg. Liver – (↑AST/ALT, RUQ Pain, Hepatitis)
      - Eg. Neuro – (Headaches, Visual Disturbance, Hyperreflexia, Clonus)
      - Eg. Haem – (DIC, Consumptive Thrombocytopaenia, Haemolysis)
      - Eg. Placental – (Foetal Growth Restriction)
  - ****Eclampsia:**
    - **= "Pre-Eclampsia *Complicated* by a Generalised Tonic-Clonic Seizure"**
    - **(ACUTE & LIFE-THREATENING:** Can → Maternal & Foetal Complications/Death**)**
- **General Management:**
  - ***ANY HTN IN PREGNANCY NEEDS INVESTIGATION & CLOSE MONITORING!!!***

PRE-ECCLAMPSIA / ECCLAMPSIA:

- **Aetiology:**
  - **Defective Placentation**
  - (**Risk Factors** – Primigravid, Older Mums, FamHx, Chronic HTN, Diabetes, Twins, Molar Pregnancy)
- **Pathogenesis:**
  - **Insufficient Placental Invasion into Spiral Arterioles → ↓Placental Blood Flow:**
    - **Pre-Eclampsia:** Placental Ischaemia → Vasoconstrictors → HTN
    - **Eclampsia:** Placental Infarction → Severe HTN → Seizures & Organ Failure.
- **Clinical Features:**
  - **Common:** 5-10% pregnancies.
  - **Symptoms:**
    - ****Headaches, Visual Disturbances** (Neuro Complications)
    - ***** **Abdo Pain** (Hepatits)
    - ***Pitting Oedema** (Renal Failure)
    - **!!Purpura & DIC** (HELLP Syndrome)
    - **!!Seizures** (IF Eclampsia)
- **Diagnosis:**
  - **Symptom Inquiry:**
    - Headaches/Visual Disturbances?
    - Epigastric Pain?
    - Oedema? (Seen as rapid weight gain)
    - Rashes?
  - **Take Blood Pressure (>140/90) →**
  - **Do Urine Dipstick/Urinalysis (Proteinuria) →**
- **Management:**
  - **Admit to BS for 4hrly Monitoring:**
    - Urinalysis (Protein++)
    - Serial BP's (Seated) every 4hrs
    - Daily UECs, FBC, LFTs
    - Daily USS for Foetal Growth & Amniotic Fluid Volume
  - **Drugs:**
    - **Antenatal Corticosteroids – (*Betamethasone*)**
    - **CaChBlockers – (*24hr Magnesium Sulfate Infusion* or *Nifedipine*)**
    - **B-Blockers – (*Labetalol*)**
  - ****Definitive = Delivery (Early *Induction of Labour*)**
  - ****If → ECCLAMPSIA (Ie. Seizures):**
    - **1. Stabilize with *Magnesium Sulfate* (NB: Do NOT use Anticonvulsants!)**
    - **2. Immediate *Delivery***
    - **3. Recovery in *HDU/ICU* for >4days *AFTER BP HAS NORMALISED*.**
  - *(CONTRAINDICATED – ACEi's/ARBs & Diuretics)
- **Complications:**
  - **Foetal Growth Restriction**
  - **Liver Failure**
  - **Acute Renal Failure**
  - **HELLP Syndrome – (*Haemolysis*, *Elevated Liver Eenzymes*, *Low Platelets*)**
    - → Jaundice, Epigastric Pain, Vomiting.
  - **DIC**
  - **Eclampsia → Seizures →**
    - **Placental Abruption**
    - **Cerebral Haemorrhage**
    - **Aspiration Pneumonia**
    - **Death**
- **Prognosis:**
  - **Eclampsia is rare with proper treatment; BUT has 20% Mortality!!**

Infant Respiratory Distress

NB: Background Info:

- **<37 wks = Pre-Term**
- **37-42 wks = Term**
- **>42 wks = Post-Term**

- **(>23 wks & <500g = NON-Viable. Ie. A Miscarriage. [Not obligated to resuscitate])**
- **(>24wks & >500g = Viable. Ie. A Birth. [Obligated to resuscitate])**

Causes for Respiratory Distress:

General Causes	**Primary Causes:** Surfactant Deficiency (Hyaline Membrane Disease) Infection (Pneumonia) with Group B Strep, or Chlamydia Meconium Aspiration (Mec. is produced later once the gut is developed) **Other Causes:** Transient Tachypnoea of the Newborn (Not pathological) Aspiration (Meconium, Milk, Blood) Pneumothorax (Overventilation) → Requires pleural tap @ 2nd ICS Pleural Effusion (Heart failure, Anaemia, Congenital Heart Failure) **Rarer Causes:** Cystic Fibrosis Congenital Cyanotic Heart Diseases Muscle Weaknesses (Myaesthenia Gravis, Muscular Dystrophies, etc)
(#1 Pre-Term Cause)	Surfactant Deficiency (Hyaline Membrane Disease)
(#1 Term Cause)	Infection (Pneumonia) with Group B Strep, or Chlamydia
(#1 Post-Term Cause)	Meconium Aspiration (Mec. is produced later once the gut is developed)

Signs of Respiratory Distress:

- **Tachypnoea** (Rate Compensation. A baby's diaphragm is already flat, so breathing deeper isn't an option)
- **Tachycardia** Initially → **Bradycardia** (Stroke Volume Compensation – Via Frank Starling Mechanism)
- **Accessory Muscle Usage**
  - "Head Bobbing" (Sternocleidomastoid Muscle Usage)
  - Intercostal Recession
  - Tracheal Tugging
  - Supraclavicular Recession
  - "Harrison's Sulcus" (Subcostal Recession)
- **Grunting** (An autonomous *Valsalva Manoeuvre* to ↑Positive Airway Pressure)
- **Nasal Flairing**

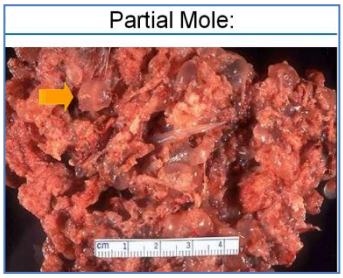
Signs of Decompensation:

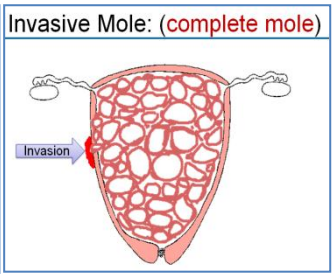
- Bradycardia
- Cyanosis
- Desaturation

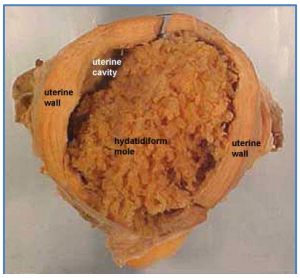
SPECIFIC OBSTETRIC NOTES:
MOLAR PREGNANCIES

HYDATIDIFORM MOLES – (PARTIAL & COMPLETE):

- **Aetiology:**
  - Error in Fusion of Gametes → Abnormal Karyotype → NOT compatible with life.
- **Pathogenesis:**
  - **Overproduction of Trophoblastic (Placental) Tissue** due to:
    - **1. Fusion of 2x Sperms with 1x Ovum → Triploidy → *Partial Mole***
      - → Abnormal Placenta & Some Foetal Development.
    - **2. Fusion of 1x Sperm with an Ovum that has LOST its DNA; OR Fusion of 2x Sperm inside an EMPTY Ovum → Sperm Duplicates → DIPLOIDY → *Complete Mole*.**
      - → Abnormal Placenta, but NO Foetus.
  - **→ Hyperplasia of Trophoblastic Tissue + Vesicular Distension of Chorionic Villi.**
  - **(NB: ↑↑↑β-hCG can mimic TSH → Secondary Hyperthyroidism)**
- **Morphology:**
  - **Partial Mole:**
    - **Macro:**
      - Partially cystic & few Blood vessels
      - **ONLY SOME Chorionic Villi are Cystic (Hence "*Partial*")**
    - **Micro:**
      - **Focal** Hyperplasia of Trophoblasts
  - **Complete Mole:**
    - **Macro:**
      - All Villi are Cystic, NO Blood Vessels within villi
      - **ALL Chorionic Villi are Cystic (Hence "*Complete*")**
      - Grape-Like Appearance of Villi
      - Entire Uterine cavity is filled with swollen villi
    - **Micro:**
      - **Diffuse** Hyperplasia of Trophoblasts
- **Clinical Features:**
  - Abnormal Growth of Uterus
  - Severe Morning Sickness (N/V)
  - Painless Vaginal Bleeding in 1st Trimester
  - Symptoms of Hyperthyroidism (Heat Intolerance, Diarrhoea, Tachycardia, Tremor)
  - Symptoms of Pre-Eclampsia (Hypertension, Oedema)
- **Diagnosis:**
  - B-hCG Level (↑in Partial; ↑↑↑ in Complete/Invasive; ↑↑↑ in Choriocarcinoma)
  - **↑ βHCG → High grade → Poor prognosis.**
  - Pregnancy Ultrasound → Abnormal Placenta ("Snowstorm"/"Grape Cluster" Uterus)
- **Treatment:**
  - Surgical Termination (D&C) + Followup B-HCG levels
  - (NB: Invasive/Metastatic Moles may require Chemotherapy – Methotrexate)
- **Prognosis:**
  - 80% are Benign (Partial) Moles
  - 20% may become Invasive (Complete) Moles → Choriocarcinoma
  - Good Prognosis (~100%) with Treatment.







Neonatal Adaptations To Extrauterine Life

After Birth:

- cast out of its watery, warm environment
- placental life supports are severed
- Now must independently respire, obtain nutrients, excrete, and maintain its body temperature

Respiratory Changes:

- Cortisol stimulates surfactant production in foetal lungs - last months of pregnancy
- After birth, CO_2 accumulates in blood → acidosis → excites respiratory centres →triggers 1st breath.
  - Adrenal Medulla→Adrenaline (from stress of birthing) also supports surfactant secretion.
- Surfactant reduces surface tension of alveolar fluid – makes initial breathing easier.
- Premature babies (no surfactant) are treated with Cortisol OR ACTH (AdrenoCorticoTropic Hormone)
  - Cortisol → Surfactant
  - ACTH→Ant.Pituitary→Adrenal Cortex→Glucocorticoids (eg. cortisol)→ surfactant.

Circulatory Changes:

- Foetal circulation is different from neonatal circulation.
  - Has to integrate circulation of placenta.
  - Blood flow to non-functional lungs & liver are partially bypassed.
- "Bypasses" / "Shunts" of foetal circulatory system:
  - **Ductus Venosus**
    - Directs the oxygenated blood from the placental vein into inferior vena cava→heart
    - Partially bypasses the liver sinusoids
  - **Foramen Ovale**
    - An opening in the **interatrial septum** loosely closed by a flap of tissue.
    - Directs some of blood entering the right atrium into the left atrium → Aorta.
    - Partially bypasses the lungs.
  - **Ductus Arteriosus**
    - Directs most blood from right atrium of the heart directly into aorta
    - Partially bypasses the lungs
  - **All of these "shunts" are occluded at birth due to pressure changes.

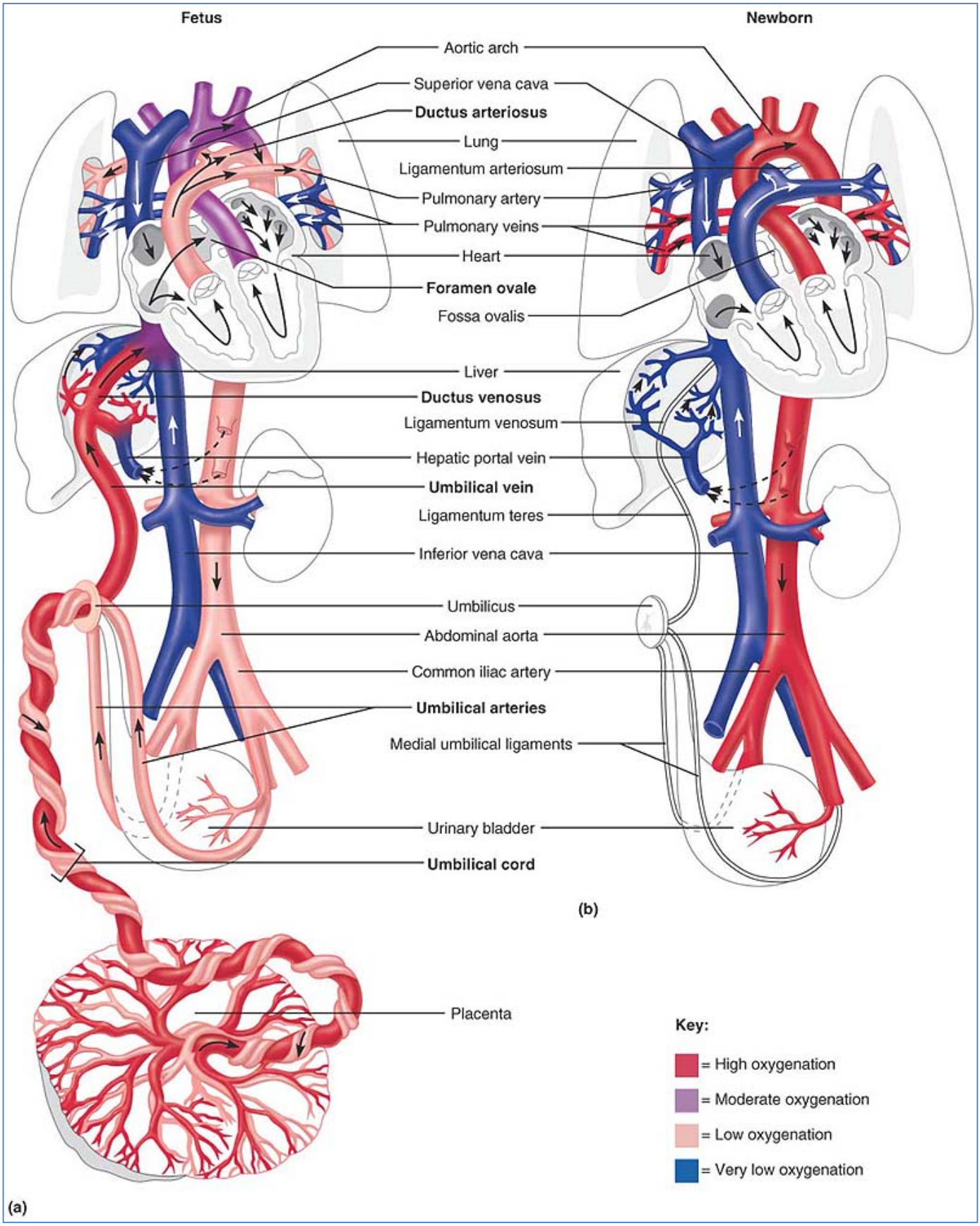
Thermoregulatory Changes:

- Newborns have high "Surface Area – Body Mass" ratio → potentiates heat loss
  - Especially head
- Newborns cannot increase heat production by shivering.
  - Instead, heat is produced by **uncouplers** in the mitochondria of **brown adipose tissue.**

Apgar Score:

- Measures a baby's health against several criteria (expressed as #/10)

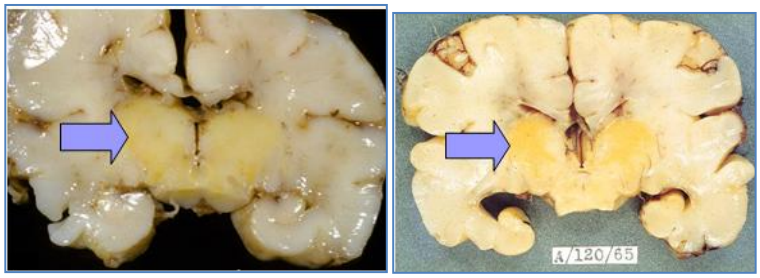
Sign	Score = 0	Score = 1	Score = 2
Heart Rate	Absent	Below 100 beats per minute	Above 100 beats per minute
Respiratory Effort	Absent	Weak, irregular or gasping	Good, crying
Muscle Tone	Flaccid (limp)	Some flexion of extremities	Well flexed, or active movements of extremities
Reflex Irritability	No response	Grimace or weak cry	Good cry
Color	Blue all over, or pale	Body pink, extremities blue	Pink all over



SPECIFIC OBSTETRIC NOTES:
NEONATAL JAUNDICE & KERNICTERUS

KERNICTERUS:

- **Aetiology:**
  - Neonatal Jaundice due to:
    - ↑RBC Breakdown
    - & ↓Ability of Liver to Conjugate Bilirubin
- **Pathogenesis:**
  - Neonates have shorter-living RBCs (Ie. ↑RBC Breakdown) & Immature Livers with limited Conjugation Capacity → → $Hyperbilirubinaemia_{(Unconjugated)}$.
  - NB: If Bilirubin levels are Extreme, it can collect in Brain Tissue
    - →→ Brain Damage & Deafness
- **Morphology:**
  - **Macro:**
    - "Kern"-"Icterus" = "Yellow Nuclei" = Yellowing of the Basal Ganglia & Thalamus.
- **Clinical Features:**
  - Jaundice Within 1st week of life.
  - Poor Feeding
  - Hypersomnolence
  - Absent Startle Reflex
  - Bulging Fontanel
- **Complications:**
  - High-Frequency Deafness
  - Mental Retardation, Speech Difficulties.
  - Seizures
- **Treatment:**
  - Phototherapy
  - Exchange Transfusions
  - Vitamin K



SPECIFIC OBSTETRIC NOTES:
OBSTETRIC DEFINITIONS

Births, Miscarriages & Abortions

Abortion	Termination of pregnancy (Spontaneous/Intentional) @ <20wks gestation OR <500g
Birth	Complete expulsion of the foetus >20wks OR >500g (Irrespective of placenta)
Miscarriages	**Missed Miscarriage:** Asymptomatic Intrauterine Foetal Death (IUFD) **Threatened Miscarriage:** Uterine bleeding +/- Contractions (NB: WITHOUT Labour) **Inevitable Miscarriage:** Uterine bleeding + Contractions + Dilation (Ie. WITH labour) **Incomplete Miscarriage:** An ACTIVE miscarriage with retained products **Complete Miscarriage:** An INACTIVE miscarriage with complete expulsion
Neonatal Death	**Early Neonatal Death:** Death of baby within 1wk of birth **Late Neonatal Death:** Death of baby within 4wks of birth
Still Birth	A "Birth" (Ie. >20wks &/or >500g) of a baby **showing no signs of life**. (Cf. Live Birth – Has spontaneous breathing/heartbeat/movement
TOP – Termination of Pregnancy	**Medical TOP** – Stat Dose Mifepristone/RU486 → 48hrs later – Vaginal Misoprostol **Surgical TOP** – Dilation & Curettage / Manual evacuation of foetus.
Viability	Gestation of >24wks Or birthweight of >500g

Timing:

Trimesters	**T1:** 0-12wks **T2:** 12-28wks **T3:** 28-40wks
Pre-Term **Term** **Post-Term**	**Pre-Term:** <37wks **Term:** 37-42wks **Post Term:** >42wks
Perinatal Period	Time within 28days of a "*Birth*". (Ie. Does NOT include Abortions)

Counting Babies:

Gravidity	Number of pregnancies (including current pregnancy) (Nulligravida – Never been pregnant) (Primigravida – First pregnancy)
Parity	Number of births @ >20wks gestation (Incl. Stillborns) (Nullipara – Never carried a pregnancy to >20wks) (Primipara – 1 previous "birth") (Multipara - >1 previous "births") (Grand-Multipara - >5 previous "births") (Great-Grand-Multipara - >10 previous "births")
Twins – Chorionicity & Amnionicity	**Chorionicity** = # of Placentas (Monochorionic = Single; Dichorionic = Double; etc) **Amnionicity** = # of Amnionic Sacs (Monoamnionic = Single; Diamnionic = Double)

Antenatal Screening Anomalies:

AFI – Amniotic Fluid Index	Sum of the amniotic fluid depth (cm) of the largest vertical pockets in each of the 4 uterine quadrants. **(N=8-24cm)** **(<8= Oligohydramnios** = Not Enough Amniotic Fluid**)** **(>24= Polyhydramnios** = Too Much Amniotic Fluid**)**
GBS	Group B Streptococcus
GDM	Gestational Diabetes Mellitis

Growth Anomalies:

CPD – Cephalo-Pelvic Disproportion	Foetal head is too big for the maternal pelvis
IUGR – Intrauterine Growth Retardation	Failure to reach genetic growth potential. (Incl. A plateau/decline in growth velocity) NB: Not necessarily *"small for gestational age" (SGA)*
LBW – Low Birth Weight	**LBW:** Low Birth Weight <2500g **VLBW:** Very low birth weight <1500g **ELBW:** Extremely low birth weight <1000g
SGA/SFD	**Small for Gestational Age/Small for Dates** = Foetus <10th percentile for Gest.Age. (NB: NOT necessarily IUGR)

Bleeding:

APH – Antepartum Haemorrhage	PV bleeding after 24wks gestation (Incl. During Labour).
PPH: Post-Partum Haemorrhage	**Primary PPH:** PV Bleeding >500mL WITHIN 24hrs of Delivery **Secondary PPH:** PV Bleeding WITHIN 6wks of Delivery
Placenta Praevia	A persistent low-lying placenta after 24wks gestation. (<24wks, it is a "Low-lying placenta")

Lie, Presentation, Attitude & Position:

Foetal Lie	Longitudinal or Transverse (Occasionally Oblique, but will → either Long/Trans during labour)
Foetal Presentation	The presenting part of the foetal body @ the birth canal. - Cephalic or Breech (If Longitudinal Lie) - Shoulder (If Transverse Lie)
Foetal Attitude/Posture	Relation of the Foetal parts to each other
Foetal Position	Right or Left side of Uterus
Breech Presentation	1. **Frank Breech –** Bum first, legs extended ("Pike") 2. **Complete Breech –** Bum first, legs flexed ("Tuck") 3. **Footling Breech –** Foot first (one or two) – (Ie. At least one hip extended)

Membrane Rupture:

ARM	Artificial rupture of membranes (using Amniohook)
PROM/PPROM	**PROM:** Premature Rupture of Membranes = ROM @>37wks, >24hrs *prior* to Onset of Labour **PPROM:** Preterm Premature Rupture of Membranes = PROM @ <37wks without onset of labour.
SROM	**Spontaneous Rupture of Membranes** (with or without labour) (NB: Can be a PROM or PPROM)

Labour/Delivery:

Engagement	Descent of the presenting part of the foetus into the mother's pelvis. Eg. If Cephalic, the head may be 4/5ths above the brim.
Labour	Regular Painful Contractions + Cervical Dilatation/Effacement + Descent of the Foetus - **Stage 1:** Onset of labour → Fully Dilated Cervix - **Stage 2:** Fully Dilated Cervix → Birth of Foetus - **Stage 3:** Birth → Delivery of Placenta & Membranes
Pre-Term **Term** **Post-Term**	**Pre-Term:** <37wks **Term:** 37-42wks **Post Term:** >42wks
LUCS	Lower-Uterine Caesarean Section (Lower horizontal incision) (Cf. Classical Caesarean – Midline Incision)
SVD/SVB	Spontaneous Vaginal Delivery/Birth
Trial of Scar/VBAC (Vaginal Birth After Caesar)	**Trial of Scar** = Attempt at vaginal delivery after Caesarean Section **VBAC** = *Successful* Vaginal Delivery after CS

Perinatal:

IRDS – Infant Respiratory Distress Syndrome	(AKA: "Hyaline Membrane Disease") Signs of increased respiratory effort in a baby due to insufficient surfactant &/or structural immaturity of the lungs.
Perinatal Period	Time within 28days of a "*Birth*". (Ie. Does NOT include Abortions)

Post Partum Haemorrhage:

Definitions:

- **PPH = >500mls Blood Loss...**
- **Primary Vs. Secondary:**
  - Primary PPH = <24hrs during/after labour
  - Secondary PPH = <6-12wks after labour
- **Minor Vs. Major:**
  - Minor PPH: 500-1000mL Blood Loss (5-15% Prevalence)
  - Major PPH: >1000mL Blood Loss (1-3% Prevalence)

Importance:

- A major cause of Mortality:
  - 1:100000 women in Australia
  - 1:1000 women in Developing Countries

Pathophysiology of PPH:

- **(Normally Haemostasis is Achieved by 2 things):**
  - 1. Myometrial Contraction → Constricts the Placenta Bed
  - 2. Normal ↑ in Thrombin during Pregnancy → Hypercoaguable State
- **PPH is Due to the "4x 'T's":**
  - **Tone** (Ie. Atonic Uterus/Insufficient Contraction)
  - **Trauma** (Ie. Uterine Tear/Perineal Tear/Vaginal Tear/Instrumental)
  - **Tissue** (Ie. RPOC – Retained Products preventing uterine contraction)
  - **Thrombin** (Ie. Bleeding Disorders)

Complications:

- Hypovolaemia
- Organ Failure (Renal/Hepatic/etc)
- Postpartum Pituitary Failure (Aka: "*Sheehan's Syndrome*")
- Shock
- Death
- ARDS (in this case due to acute haemorrhagic anaemia)
- DIC

NBs:

- Uterotonic agents (Eg. Oxytotics – Syntocinon/Syntometrin/Ergometrin) in the 3rd stage of labour enhances haemostasis via uterine contraction → ↓Risk by 2xfold
- "An intact, empty and contracted uterus WILL NOT BLEED"
- 1x Resuscitation should be enough; if you need to do it twice, TAKE HER TO THEATRE!!

Primary PPH:

(From beginning of labour to within 24hrs after)

Risk Factors:

- **Intrapartum:**
  - Prolonged Labour (→ Tired Uterus)
  - Trauma (Instrumental/Tears)
  - Caesarean Section
  - Tocolytics during labour (→ ↓Contractility of Uterus)
  - Dystocia (→ Prolonged Labour / ↑Risk of Tearing)
- **Postpartum:**
  - Previous Hx of PPH
  - Bleeding Disorders (Incl. Anticoagulation)
  - Multiparity (→ Stretched Uterus)
    - Incl. Multiple Pregnancy (Eg. Twins/Triplets/etc)
    - Incl. Polyhydramnios
    - Incl. Macrosomia
  - Chorioamnionitis
  - Placental Abnormalities
    - Accreta (Abnormally Deep Placenta – Into Myometrium; 1/500 pregnancies)
    - Praevia (Placenta Close/Covering Cervical Canal)
    - Placental Abruption
  - RPOC - Retained Products of Conception

Prevention & Avoiding Complications:

- Detect Risk Factors
- IV Access
- G&H
- X-Match
- Syntocinon @ the ready
- Active Mx of 3rd Stage Labour (Ie. Oxytotics - Syntocinon/Syntometrin/Ergometrin – Usually mixed with Hartmann's Solution)

Mx:

- **Minor PPH (500-1000mLs) (5-15% Incidence)**
  - Assess
  - 1x Large Bore IVC
  - Resuscitate with Fluids (Crystalloids)
  - Call for help (Reg/Consultant)
  - Bloods:
    - G&H
    - X-Match
    - Rhesus status
    - FBC (Baseline Hb)
  - Give Syntocinon
  - Massage Fundus (of Uterus) → Expels Clots/RPOCs
  - Manual Removal of RPOCs
  - Bimanual Compression if bad.
  - +....Treat Cause

- **Major PPH (>1000mLs) (1-3% Incidence)**
  - All of Above....PLUS:
  - Call Operating Theatre
  - Call Anaesthetist
  - Additional Large Bore IVC
  - IDC – Monitor Urine Output
  - Additional Oxytotics (Ie. Syntocinon/Syntometrin/Ergometrin)
  - Blood Transfusion/s:
    - Packed Red Cells
    - FFP
    - Cryoprecipitate
    - Etc.
  - +.... Treat Cause

Rx:

- **1. Stop Bleeding:**
  - **Tone**
    - **Oxytotics:** (Syntocinon/Syntometrin/Ergometrin)
    - **Prostaglandins:** (Misoprostol, PGF2a Injection)
    - **Bimanual Compression**
    - **Theatre:**
      - Balloon Tamponade
      - "B-Lynch Suture"
      - Uterine Artery Ligation
      - Hysterectomy
  - **Tissue** (Manual/Surgical Removal of Incomplete RPOCs)
  - **Trauma** (Repair tears/Uterine Ruptures/Cervical Rupture/etc)
  - **Thrombin** (Give FFP infusion)
- **2. Transfuse (Usually by Anaesthetist):**
  - Packed RBCs (4-6 Units)
  - FFP (4 Units [per 6units of RBCs])
  - Platelets
  - Cryoprecipitate/Recombinant Factor-VII
- **3. Close Monitoring in ICU for Organ Failure:**
  - BP
  - Urine Output

Secondary PPH

(>24hrs after labour → 6-12mths later)

Causes:

- Infection
- RPOC
- Gestational Trophoblastic Diseases (Rare)
    - Eg. Molar Pregnancy
    - Eg. Invasive Trophoblastic Disease
    - Eg. Choriocarcinoma
    - Eg. Placental Site Tumour

Presentation:

- Typically slower bleed (A Trickle)

Mx:

- Curette for RPOC
- Antibiotics
- B-HCG (check for molar pregnancy)
- Pelvic USS

The 3 Postnatal Mood Disorders:

1. **"The Baby Blues" (MILD)**
    a. Affects <80% of new mothers
    b. Typical onset between Day 3-10 after birth.
    c. Symptoms:
        i. Tearfulness
        ii. Anxiety
        iii. mood swings
        iv. irritability.
    d. Treatment:
        i. "The Baby Blues" are transient and pass with supportive therapy.

2. **"Postnatal Depression" (MODERATE)**
    a. Affects 15% of women and 10% of men
    b. Onset anywhere from 24 hours-several months after delivery
    c. Symptoms:
        i. Insomnia
        ii. Anorexia
        iii. Crying
        iv. Acopia with daily tasks
        v. Exhaustion
        vi. Irritability
        vii. Anxiety
        viii. Fear of social contact
        ix. Fear of being alone
        x. Guilt
        xi. Low confidence
        xii. Suicidal thoughts.
        xiii. **"There is no joy in anything any more"**
    d. **Treatment:**
        i. **Emotional support from family and friends.**
        ii. Antidepressants are also effective.

3. **"Postnatal Psychosis" (SEVERE)**.
    a. Affects 1 in 500 mothers
    b. Onset within the 1st month of delivery.
    c. Symptoms:
        i. The mother may be unaware she is ill (Due to psychosis).
        ii. Severe mood disturbance (Manic and/or Depressive)
        iii. Thought disturbance (either processing or bizarre thoughts)
        iv. Insomnia
        v. Inappropriate responses to the baby.
        vi. Can be LIFE THREATENING for both mum & bub if undiagnosed.
    d. Treatment:
        i. **Requires hospitalisation.**
        ii. Anti-psychotics and/or Antidepressants

Family Studies Tutorial
Postnatal Depression

Presentation:

- Anxiety/Depression/Acopia
- Referral from external source (Midwives, comm. health)
- Inability to cope
- Husband/family member presents with concerns
- **Symptoms:**
    - Somatic Symptoms
    - Issues with children
    - Irribability & Tearfulness
    - Avoiding personal discussion
    - Anxiety
    - Denial
    - Delayed attachment
    - Negative feelings to infant
- **Risk Factors:**
    - Indigenous background
    - Lower Socio-economic status
    - Younger age
    - Absence of partner
    - Medical complications
    - Marital problems
    - History of abuse
    - Not breast-feeding
    - No job to return to
    - Problematic births
    - Reluctance to seek help
- **Protective Factors:**
    - Optimism & Self esteem
    - Higher education
    - Good SES
    - Strong relationship with partner
- **Management:**
    -
- **Effect on Infant:**
    - Insecure infant – lack of trust, poor interaction with caregiver
    - Attachment issues – discipline, behaviour & aggression problems
    - Infant withdrawn, passive
    - Slow to reach milestones
    - High risk of mental health issues in child.
- **Dads:**
    - Fathers can get depressed too
    -

12-15% of mothers

Depression:

- **= Depressed mood, or loss of interest or pleasure.**
    - **+ 4 of the following:**
        - Sadness or fear
        - Inability to feel emotion (Anhedonia)
        - Decreased pleasure derived from previously pleasurable activities
        - Changing appetite and weight gain/loss
        - Insomnia
        - Restlessness

        - Fatigue
        - **Guilt**, helplessness, anxiety, fear
        - Decreased self-esteem
        - REminating on death or suicide
- **Post natal Depression –** Non-psychotic depression occurring in the first 3mths

Screening:

- K10
- EPDS - Edinburgh postnatal depression scale (10qs, 5mins, responses graded).

<u>Other Postpartum Distresses:</u>

- **Post-partum Anxiety**
- **Postpartum OCD**
- **Postpartum Psychosis (Hallucinations & Delusions)**
- **Exacerbation of Pre-existing mental illness**
- **Baby Blues:**
    - 70-80% of women
    - Feelings of depression, anger, anxiety & guilt lasting for several days
    - Rx: Supportive management & Explanation
    - Disappears within a few days

Eg.

- **22yo F pw. Inability to sleep. 2wks post delivery.**
- **Questions:**
    - How is bub sleeping?
    - How are you managing at home?
    - Who is supporting you?
    - Hows your diet?
    - Do you feel down?
    -

Postnatal Menstruation & Contraception:

- **Amenorrhoea while Breastfeeding:**
  - Breastfeeding can cause amenorrhoea for between 2-12mths (Highly variable)
  - (If not breastfeeding, periods usually return within a few weeks)
- **Postnatal Menstruation:**
  - The 1st period after a baby may be heavier and more uncomfortable than usual.
  - Remember conception is still possible before this period, so contraception is advised.
- **Contraception**
  - **Lactational Amenorrhoea Method:**
    - Oxytocin inhibits ovulation for the first few months
    - This is only effective (But not guaranteed) if:
      - Exclusively breastfeeding **day and night**
      - Haven't had the 1st period yet
      - Baby is less than six months old.
  - **The Minipill (Prog. Only OCP):**
    - Safe to use while breastfeeding
  - Condoms:
    - Safe to use while breastfeeding.
  - **NB:** The C**ombined oral contraceptive pill should NOT be used when breastfeeding** because it decreases the milk supply.

Pregnancy Advice for New Mothers

http://raisingchildren.net.au/articles/grownups_pregnancy_nutshell.html/context/401

Staying healthy

- **Regular**, **moderate exercise** (Eg. Walking)
  - Helps you keep strong for the birth
  - Lifts your mood
  - Helps maintain a healthy weight.
- **Healthy eating**
  - keeps you feeling good
  - gives your baby the essential nutrients he needs in utero.
- **Avoid certain foods**
  - Soft cheeses
  - Raw fish.
  - **Limit caffeine**.
- **Drugs & Alcohol:**
  - **For prescribed drugs**, check with your doctor that they are safe
  - **Don't smoke**
  - **No recreational drugs**
  - **No alcohol**.

Folic Acid: preventing spinal abnormality (Eg. Spina Bifida)

- Folate Supplement Guidelines – To reduce risk of Neural Tube Defects by <70%:
  - Folate tablets (0.5mg OD)
  - **>1mth BEFORE conception**
  - **& DURING the first 3 months of pregnancy**
- Every woman who could become pregnant should take folic acid tablets.
- NB: Folate = A natural vitamin found in most plants (esp. green vegetables, wholegrain breads and cereals, peas and dried beans)

Pregnancy hormones

- Between the first 6-12 weeks of pregnancy, oestrogen rises significantly.
- This **can cause nausea and vomiting (Aka. morning sickness)**
  - Some women get it, some don't.
  - Morning sickness *usually* abates after 1st 3-4mths, but can last <9mths.
- **Can also cause mood swings**.

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    d. Treatment:
        i. **Requires hospitalisation.**
        ii. Anti-psychotics and/or Antidepressants

PROM & PPROM

Definitions:

- **Premature rupture of membranes** (**PROM**) = Rupture of the amniotic sac **>1hour *before* onset of labor**.
  - ***Prolonged PROM*** = >18 hours before labor.
- ***Preterm* PROM (PPROM)** = PROM before 37 weeks gestation.

(NB: PROM is a variation of normal; whereas PPROM is often pathological and can be dangerous)
(NB: Typically, labour begins <48hrs of PROM)
(NB: Sometimes the rupture may heal spontaneously)

Risk Factors:

- Bacterial Infection
  - Eg. Choioamnionitis
  - Eg. Maternal Sepsis
- Smoking
- Anatomic Defect of:
  - the amniotic sac
  - uterus
  - cervix
- Previous PROM/PPROM

Assessment

- proper medical history
- Spec gynecological exam
- nitrazine
- ultrasound
- Amniotic fluid smear cytology/microscopy (dried for 10 minutes on a slide shows a characteristic fernlike pattern)
- Actin-PROM.

Management

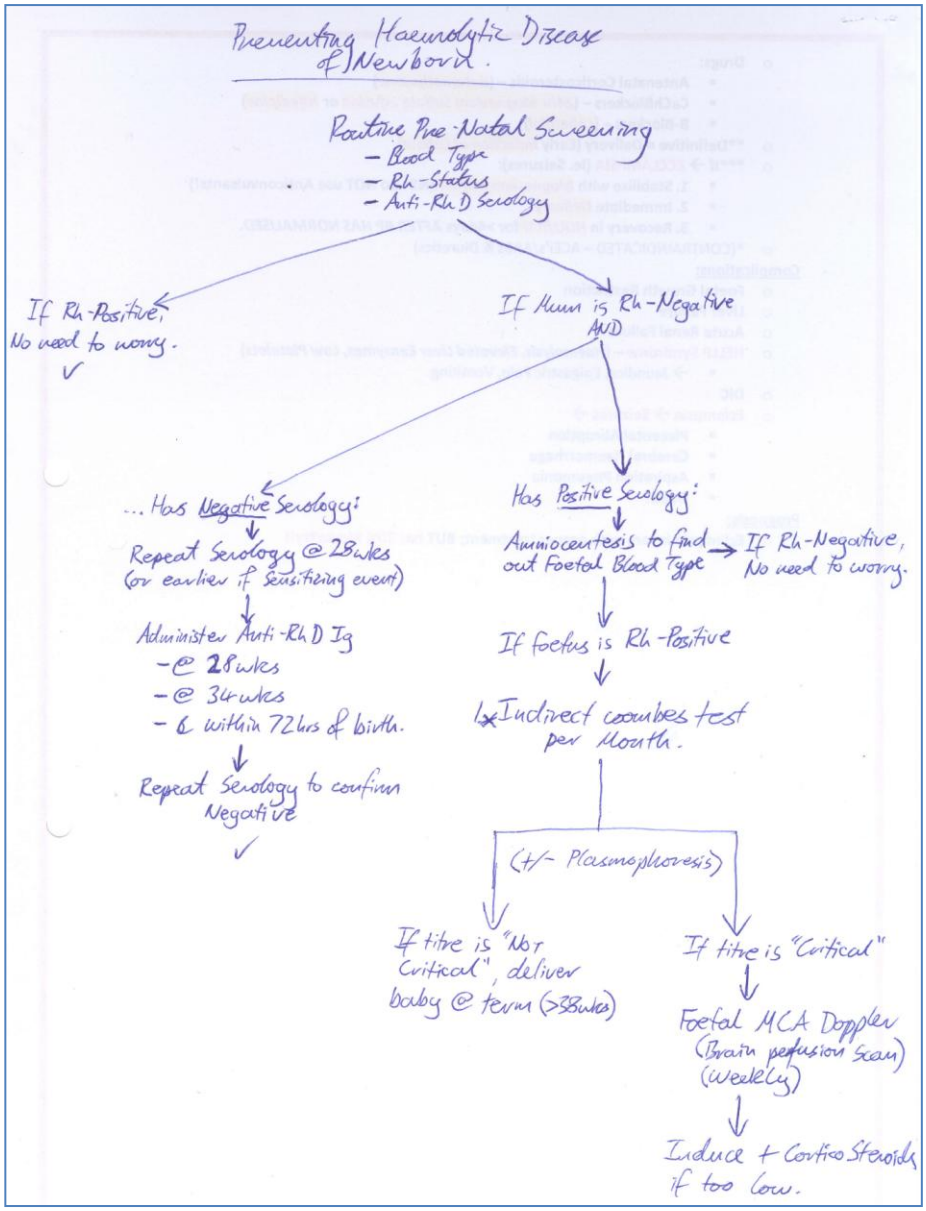
- PROM (>37wks):
  - Permit spontaneous labor (up to 12hrs)
  - Induction of labor (@ 12 hours) if it has not already begun
  - Consider Group B Streptococcal prophylaxis (@ 18 hours).
- PPROM (<37wks):
  - Admit mother
  - Steroids (2days)
  - Watch for Preterm Labour
  - Watch for Chorioamnionitis
    - Antibiotic prophylaxis (Ampicillin/Erythromycin) to delay delivery → ↑Foetal Maturation, & prevent sepsis
  - Avoid labour *prior to 34wks* (Tocolysis may be used).
  - Induction of labor @ 34 weeks.
- Infection
  - Chorioamnionitis:
    - Antibiotic therapy to avoid sepsis
    - Induction/Delivery is indicated.
  - Fetal:
    - If the GBS status of the mother is unknown, Antibiotic Prophylaxis (Penicillin/other) protects against vertical transmission of Group B streptococcal infection.

SPECIFIC OBSTETRIC NOTES:

RHESUS DISEASE

HAEMOLYTIC DISEASE OF THE FOETUS/NEWBORN:

- **What is it?**
  - Condition of the **Foetus**, due to **Maternal IgG-Anti-RhD Abs** cross the Placenta→Foetal Circulation.
    - →Foetal Immune-Mediated Haemolysis → Anaemia → **Stillborn/Miscarriage**
- **Pathogenesis:**
  - **Mother is usually Rh-Negative, :.** Does NOT Have Anti-D Antibodies.
  - **During Pregnancy:**
    - 1. Foetal Blood can mix with Maternal Blood in "Sensitizing Events"
      - Eg. Placental Abruption
      - Eg. Placental Injury
      - Eg. Amniocentesis/Chorionic Villus Sampling
      - Eg. Miscarriage/Abortion/D&C
      - Eg. Ectopic Pregnancy
      - Eg. Antepartum Haemorrhage
      - Eg. During Labour
      - Eg. External Cephalic Version
    - 2. If Foetus is RH-*Positive*, The Mother forms Anti-RhD Antibodies.
    - 4. Maternal Anti-RhD Antibodies can cross the Placenta → Foetus.
    - 5. Antibodies Attack Foetal RBCs → Haemolysis/Jaundice → Anaemia → Death
  - **NB: Once Sensitized to RhD, ALL SUBSEQUENT PREGNANCIES ARE AT RISK!**
- **Prevention:**



Continue Reading For Bonus Supplementary Study Materials…

OB Obstetrics

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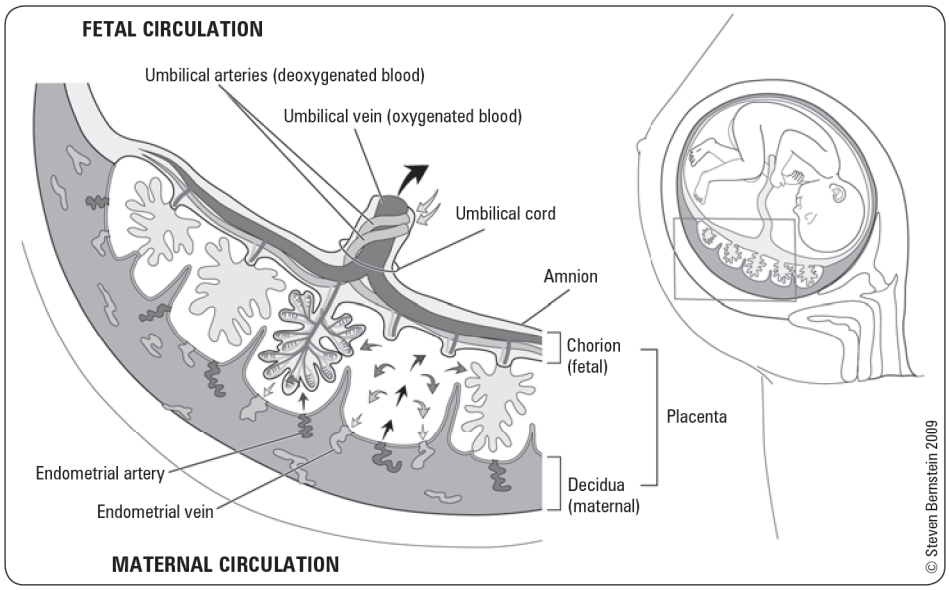

Figure 1. Placental blood flow

Placenta

- site of fetal nutritive, respiratory, and excretory function
- discoid mass composed of fetal (chorion frondosum) and maternal (decidua basalis) tissues divided by fissures into cotyledons (lobules) on the uterine side
- produces hormones such as progesterone, placental lactogen, estrogen, relaxin, β-hCG, and IGFs
- poor implantation can lead to spontaneous abortion
- abnormal location, implantation, or detachment can lead to antepartum hemorrhage (see *Obstetrical Hemorrhage*, OB14)

Pregnancy

Diagnosis of Pregnancy

History

- obstetrical and gynecological history
- obtain the year, location, mode of delivery, duration of labour, sex, gestational age, birth weight, and complications of every pregnancy; organize into GTPAL format
  - **Gravidity (G)**
    - **G:** total number of pregnancies of any gestation (multiple gestation=one pregnancy)
  - includes current pregnancy, abortions, ectopic pregnancies, and hydatidiform moles
  - **Parity (TPAL)**
    - **T:** number of term infants delivered (>37 wk)
    - **P:** number of premature infants delivered (20-36+6 wk)
    - **A:** number of abortions (loss of intrauterine pregnancy prior to viability of fetus <20 wk and/or <500 g fetal weight)
      - induced (therapeutic) and spontaneous (miscarriage)
    - **L:** number of living children
- symptoms: amenorrhea, nausea and/or vomiting, breast tenderness, urinary frequency, and fatigue

Physical Signs

- Goodell's sign: softening of the cervix (4-6 wk)
- Chadwick's sign: bluish discolouration of the cervix and vagina due to pelvic vasculature engorgement (6 wk)
- Hegar's sign: softening of the cervical isthmus (6-8 wk)
- uterine enlargement
- breast engorgement, areolae darkening, and prominent vascular patterns

Acronyms

AC	abdominal circumference
ACOG	American Congress of Obstetricians and Gynecologists
AFI	amniotic fluid index
AFLP	acute fatty liver of pregnancy
AFV	amniotic fluid volume
AP	anteroposterior
APS	antiphospholipid antibody syndrome
BPP	biophysical profile
C/S	Cesarean section
CPD	cephalopelvic disproportion
CTG	cardiotocography
CVS	chorionic villus sampling
D&C	dilatation and curettage
DIC	disseminated intravascular coagulation
DVT	deep vein thrombosis
ECV	external cephalic version
EDC	estimated date of confinement
EFM	electronic fetal monitoring
EFW	estimated fetal weight
FDP	fibrin degradation products
FHR	fetal heart rate
FISH	fluorescence *in situ* hybridization
FL	femur length
FM	fetal movement
FPG	fasting plasma glucose
FTS	first trimester screen
GA	gestational age
GBS	Group B *Streptococcus*
GDM	gestational diabetes mellitus
GTN	gestational trophoblastic neoplasia
HC	head circumference
HELLP	hemolysis, elevated liver enzymes, low platelets
IGF	infant growth factors
IMM	intramyometrial
IOL	induction of labour
IPS	integrated prenatal screen
IUFD	intrauterine fetal death
IUGR	intrauterine growth restriction
IVH	intraventricular hemorrhage
L/S	lecithin-sphingomyelin ratio
LLDP	left lateral decubitus position
LMP	last menstrual period
MSAFP	maternal serum α-fetoprotein
MSS	maternal serum screen
MTX	methotrexate
NIPT	non-invasive prenatal testing
NST	non-stress test
NTDs	neural tube defects
NTUS	nuchal translucency ultrasound
OA	occiput anterior
OGTT	oral glucose tolerance test
oNTD	open neural tube defect
OP	occiput posterior
OT	occiput transverse
PAPP-A	pregnancy-associated plasma protein a
PG	plasma glucose
PPD	postpartum depression
PPH	postpartum hemorrhage
PPROM	preterm premature rupture of membranes
PROM	premature rupture of membranes
PTL	preterm labour
RDS	respiratory distress syndrome
ROM	rupture of membranes
SFH	symphysis fundal height
SOGC	Society of Obstetricians and Gynaecologists of Canada
SVD	spontaneous vaginal delivery
TENS	transcutaneous electrical nerve stimulation
TPN	total parenteral nutrition
UTI	urinary tract infection
VBAC	vaginal birth after Cesarean

Umbilical Vessels
Always check the umbilical cord for 2 arteries and 1 vein: approximately 1/3 of babies with a single uterine artery will have another anomaly, IUGR or aneuploidy

Investigations

- β-hCG: peptide hormone composed of α and β subunits produced by placental trophoblastic cells – maintains the corpus luteum during pregnancy
  - positive in serum 9 d post-conception, positive in urine 28 d after first day of LMP
  - plasma levels double every 1-2 d, peak at 8-10 wk, then fall to a plateau until delivery
    - levels less than expected suggest: ectopic pregnancy, abortion, or inaccurate dates
    - levels greater than expected suggest: multiple gestation, molar pregnancy, Trisomy 21, or inaccurate dates
- U/S
  - transvaginal
    - 5 wk amenorrhea: gestational sac visible
    - 6 wk: fetal pole visible
    - 7-8 wk: fetal heart activity visible
  - transabdominal
    - 6-8 wk: intrauterine pregnancy visible (β-hCG ≥6,500 mIU/mL)

β-hCG Rule of 10s
10 IU at time of missed menses
100,000 IU at 10 wk (peak)
10,000 IU at term

Trimesters
- T1 (first trimester): 0-14 wk
- T2 (second trimester): 14-28 wk
- T3 (third trimester): 28-42 wk
- Normal pregnancy term: 37-42 wk

Maternal Physiologic Adaptations to Pregnancy

Table 1. Physiologic Changes During Pregnancy

Skin	Increased pigmentation of perineum and areola, chloasma (pigmentation changes under eyes and on bridge of nose), linea nigra (midline abdominal pigmentation) Other: spider angiomas, palmar erythema due to increased estrogen, striae gravidarum due to connective tissue changes
Cardiovascular	Hyper-dynamic circulation Increased CO, HR, and blood volume Decreased blood pressure due to decreased PVR and decreased venous return from enlarging uterus compressing IVC and pelvic veins Increased venous pressure leads to risk of varicose veins, hemorrhoids, leg edema
Hematologic	Hemodilution causes physiologic anemia and apparent decrease in hemoglobin and hematocrit Increased leukocyte count but impaired function leads to improvement in autoimmune diseases Gestational thrombocytopenia: mild (platelets >70,000/μL) and asymptomatic, normalizes within 2-12 wk following delivery Hypercoagulable state: increased risk of DVT and PE but also decreased bleeding at delivery
Respiratory	Increased incidence of nasal congestion and epistaxis Increased O_2 consumption to meet increased metabolic requirements Elevated diaphragm (i.e. patient appears more "barrel-chested") Increased minute ventilation leads to decreased CO_2 resulting in mild respiratory alkalosis that helps CO_2 diffuse across the placenta from fetal to maternal circulation No change in VC and FEV_1 Decreased TLC, FRC, and RV
Gastrointestinal	GERD due to increased intra-abdominal pressure and progesterone (causing decreased sphincter tone and delayed gastric emptying) Increased gallstones due to progesterone causing increased gallbladder stasis Constipation and hemorrhoids due to progesterone causing decreased GI motility
Genitourinary	Increased urinary frequency due to increased total urinary output Increased incidence of UTI and pyelonephritis due to urinary stasis (see *Urinary Tract Infection*, OB29) Glycosuria that can be physiologic especially in the 3rd trimester; must test for GDM Ureters and renal pelvis dilation (R>L) due to progesterone-induced smooth muscle relaxation and uterine enlargement Increased CO and thus increased GFR leads to decreased creatinine (normal in pregnancy 35-44 mmol/L), uric acid, and BUN
Neurologic	Increased incidence of carpal tunnel syndrome and Bell's palsy
Endocrine	Thyroid: moderate enlargement and increased basal metabolic rate Increased total thyroxine and thyroxine binding globulin (TBG) Free thyroxine index and TSH levels are normal Adrenal: maternal cortisol rises throughout pregnancy (total and free) Calcium: decreased total maternal Ca^{2+} due to decreased albumin Free ionized Ca^{2+} (i.e. active) proportion remains the same due to parathyroid hormone (PTH), results in increased bone resorption and gut absorption, increased bone turnover (but no loss of bone density due to estrogen inhibition)

Antepartum Care

- provided by obstetrician, family doctor, midwife, or multidisciplinary team (based on patient preference and risk factors)
- Antenatal Records (province specific)

Family doctors and midwives to consider OB consultation if:
- Insulin-dependent GDM
- VBAC
- HTN
- Multiple gestation
- Malpresentation
- Active antepartum hemorrhage
- PTL/PPROM
- Failure to progress/descend
- Induction/augmentation if high risk
- Tears: 3rd or 4th degree
- Retained placenta

Note: Guidelines vary by institution

Preconception Counselling

- 3-8 wk GA is a critical period of organogenesis, so early preparation is vital

- **past medical history:** optimize illnesses and medications prior to pregnancy (see *Medical Complications of Pregnancy*, OB26, and *Medications in Pregnancy*, OB11)

- **supplementation**
  - folic acid: encourage diet rich in folic acid and supplement 8-12 wk preconception until end of T1 to prevent NTDs
    - 0.4-1 mg daily in all women; 5 mg if previous NTD, antiepileptic medications, DM, or BMI >35 kg/m^2
- iron supplementation, prenatal vitamins

Advise all women capable of becoming pregnant to supplement their diet with 0.4 mg/d of folic acid (CTFPHC Grade II-2-A Evidence)

- **risk modification**
  - lifestyle: balanced nutrition and physical fitness
  - medications: discuss teratogenicity of medications so they may be adjusted or stopped if necessary
  - infection screening: rubella, HBsAg, VDRL, Pap smear, gonorrhea/chlamydia, HIV
  - genetic testing as appropriate for high risk groups (see *Prenatal Screening*, Table 2); consider genetics referral in known carriers, recurrent pregnancy loss/stillbirth, family members with developmental delay or birth anomalies
  - social: alcohol, smoking, street drugs, domestic violence (see Family Medicine, FM11, FM13, FM27)

Prenatal and genetic screening are voluntary and require proper counselling and informed consent before proceeding

HIV is done automatically in some provinces as opt-out testing, need to inform patient

Initial Prenatal Visit

- usually within 8- 12 wk of the first day of LMP or earlier if <20 or >35 yr old, bleeding, very nauseous, or other risk factors present
- Antenatal Records are filled out on the first prenatal visit

History

- gestational age by dates from the first day of the LMP
- if LMP unreliable, get a dating ultrasound which could coincide with nuchal translucency at ~12 wks
- dates should change if T1 U/S is greater than 5 days in difference from LMP due date
  - Naegle's rule: 1st day of LMP + 7 d – 3 mo
  - e.g. LMP = 1 Apr 2014, EDC = 8 Jan 2015 (modify if cycle >28 d by adding number of d >28)
- history of present pregnancy (e.g. bleeding, N/V) and all previous pregnancies
- past medical, surgical, and gynecological history
- prescription and non-prescription medications
- family history: genetic diseases, birth defects, multiple gestation, consanguinity
- social history: smoking, alcohol, drug use, domestic violence (see Family Medicine, FM11, FM13, FM27)

In history of previous pregnancies, **ALWAYS** ask:
GTPAL
Year
Sex
Weight
Gestational age (GA)
Mode of delivery
Length of labour
Complications

Ask every woman about abuse – not just those whose situations raise suspicion of abuse AND ask as early as possible in pregnancy

Physical Exam

- complete physical exam to obtain baseline patient information
- BP and weight important for interpreting subsequent changes
- pelvic exam

Investigations

- blood work
  - CBC, blood group and Rh status, antibody screen, infection screening as per preconception counselling
- urine R&M, midstream urine C&S
  - screen for bacteriuria and proteinuria
- pelvic exam
  - Pap smear (only if required according to patient history and provincial screening guidelines), cervical or urine PCR for *N. gonorrhoeae* (GC) and *C. trachomatis*

Nausea and Vomiting

Epidemiology
- affects 50-90% of pregnant women
- often limited to T1 but may persist

Management
- rule out other causes of N/V
- weigh frequently, assess level of hydration, test urine for ketones
- **non-pharmacological**
  - avoid mixing fluids and solids, frequent small meals
  - stop prenatal vitamins (folic acid must continue until >12 wk)
  - increase sleep/rest
  - ginger (maximum 1,000 mg/d)
  - acupuncture, acupressure
- **pharmacological**
  - first line: Diclectin® (10 mg doxylamine succinate with vitamin B6) 4 tablets PO daily to maximum of 8 tablets
  - if no improvement, try dimenhydrinate (50-100 mg q4-6h PO), followed by hydroxyzine, pyridoxine, phenothiazine, or metoclopramide
  - vitamin B6 lollipops
  - if patient dehydrated, assess fluid replacement needs and resuscitate accordingly
- **severe/refractory**
  - consider homecare with IV fluids and parenteral anti-emetics, hospitalization

Hyperemesis Gravidarum

Definition
- intractable N/V, usually presents in T1 then diminishes; occasionally persists throughout pregnancy
- affects ~1% of pregnancies

Etiology
- multifactorial with hormonal, immunologic, and psychologic components
- rapidly rising β-hCG ± estrogen levels may be implicated

Investigations
- rule out systemic causes: GI inflammation, pyelonephritis, thyrotoxicosis
- rule out obstetrical causes: multiple gestation, GTN, HELLP syndrome
- CBC, electrolytes, BUN, creatinine, LFTs, urinalysis
- ultrasound

Management
- thiamine supplementation may be indicated
- non-pharmacological (see *Nausea and Vomiting*, OB5)
- pharmacological options
  - Diclectin® (for dosage, see *Nausea and Vomiting*, OB5)
  - Dimenhydrinate can be safely used as an adjunct to Diclectin® (1 suppository bid or 25 mg PO qid)
  - other adjuncts: hydroxyzine, pyridoxine, phenothiazine, metoclopramide
  - also consider: ondansetron or methylprednisolone
  - if severe: admit to hospital, NPO initially then small frequent meals, correct hypovolemia, electrolyte disturbance, and ketosis, TPN (if very severe) to reverse catabolic state

Complications
- maternal
  - dehydration, electrolyte and acid-base disturbances
  - Mallory-Weiss tear
  - Wernicke's encephalopathy, if protracted course
  - death
- fetal: usually none, IUGR is 15x more common in women losing >5% of pre-pregnancy weight

Subsequent Prenatal Visits

Timing
- for uncomplicated pregnancies, SOGC recommends q4-6wk until 30 wk, q2-3wk from 30 wk, and q1-2 from 36 wk until delivery

Assess at Every Visit
- estimated GA
- history: fetal movements, uterine bleeding, leaking, cramping, questions, concerns
- physical exam: BP, weight gain, SFH, Leopold's maneuvers (T3) for lie, position, and presentation of fetus
- investigations: urinalysis for glucosuria, proteinuria; fetal heart rate starting at 10-12 wk using Doppler U/S

Leopold's Maneuvers
- performed after 30-32 wk gestation
- first maneuver: to determine which fetal part is lying furthest away from the pelvic inlet
- second maneuver: to determine the location of the fetal back
- third maneuver: to determine which fetal part is lying above the pelvic inlet
- fourth maneuver: to locate the fetal brow

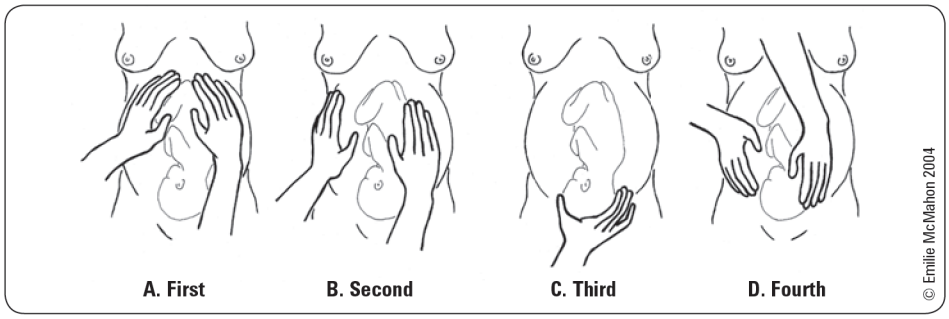

Figure 2. Leopold's maneuvers (T3)
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Small for Dates
- Date miscalculation
- IUGR
- Fetal demise
- Oligohydramnios

Large for Dates
- Date miscalculation
- Multiple gestation
- Polyhydramnios
- LGA (familial, DM)
- Fibroids

Prenatal Screening and Diagnostic Tests

Screening Tests
- testing should only occur following counselling and with the informed consent from the patient

Table 2. High-Risk Population Screening Tests

Disease (Inheritance)	Population(s) at Risk	Screening Test(s)
Thalassemia (AR)	**Mediterranean, South East Asian, Western Pacific**, African, Middle Eastern, Caribbean, South American	CBC (MCV and MCH), Hb electrophoresis, or HPLC
Sickle Cell (AR)	**African, Caribbean**, Mediterranean, Middle Eastern, Indian, South American	CBC (MCV and MCH), Hb electrophoresis, or HPLC
Cystic Fibrosis (CF) (AR)	Family history of CF in patient or partner or medical condition linked to CF like male infertility	CFTR gene DNA analysis
Tay Sachs Disease (AR)	Ashkenazi Jewish*, French Canadians, Cajun	Enzyme assay HEXA, or DNA analysis HEXA gene
Fragile X Syndrome (X-linked)	Family history – confirmed or suspected	DNA analysis: FMR-1 gene

AR = autosomal recessive; HEXA = hexosaminidase A; HPLC = high performance liquid chromatography
*If both partners are Ashkenazi Jewish, test for Canavan disease and Familial Dysautonomia (FD); if family history of a specific condition, look for carrier status: e.g. Gaucher, CF, Bloom syndrome, Niemann-Pick disease, etc. In all cases, if both partners positive, refer for genetic counselling

Table 3. Gestation-Dependent Screening Investigations

Gestational Age (wk)	Investigations	Details
8-12	Dating U/S, possible Pap smear, chlamydia/gonorrhea cultures, urine C&S, HIV, VDRL, HepBSAg, Rubella IgG, Parvovirus IgM or IgG if high risk (small child at home or daycare worker/primary teacher), Varicella IgG if no history of disease/immunization, CBC, blood group and screen	
10-12	CVS	
11-14	FTS IPS Part 1	Measures 1. Nuchal translucency on U/S 2. β-hCG 3. PAPP-A
11-14	Nuchal translucency U/S	
15-16 to term	Amniocentesis	
15-20	IPS Part 2 (or MSAFP only for patients who did FTS earlier)	Measures 1. MSAFP 2. β-hCG 3. Unconjugated estrogen (estriol or μE3) 4. Inhibin A
15-20	MSS (or MSAFP only for patients who did FTS earlier)	Measures 1. MSAFP 2. β-hCG 3. Unconjugated estrogen (estriol or μE3) 4. Inhibin A
18-20 to term	Fetal movements (quickening)	
18-20	U/S for dates, fetal growth, and anatomy assessment	
24-28	Gestational Diabetes Screen 50 g OGCT	See *Diabetes Mellitus*, OB27
28	Repeat CBC RhIG for all Rh negative women	
35-37	GBS screen	See *Group B Streptococcus*, OB29
6 wk postpartum	Discuss contraception, menses, breastfeeding, depression, mental health, support Physical exam: breast exam, pelvic exam including Pap smear (only if due as per provincial screening)	

Maternal serum screen is also referred to as Triple Screen; if Inhibin A is also tested, it is referred to as Quadruple Screen
Ideally testing for MSS and IPS Part 2 occur between 15-18 wk to give women more time to make decisions and move ahead with diagnostic testing should the resulting screen be positive

Routine T2 U/S at 18-22 wk Helps Determine
- Number of fetuses
- GA (if no prior U/S)
- Location of placenta
- Fetal anomalies

DDx of Increased MSAFP
- Incorrect GA
- >1 fetus (e.g. twins)
- Fetal demise
- oNTD
- Abdominal wall defects (e.g. omphalocele)

DDx of Decreased MSAFP
- Incorrect GA
- Gestational trophoblastic neoplasia
- Missed abortion
- Chromosomal anomalies
- Maternal DM

Ultrasound Screening
- 8-12 wk GA: Dating Ultrasound (most accurate form of pregnancy dating)
  - measurement of crown-rump length (margin of error ± 5 d)
  - change EDC to U/S date if >5 d discrepancy from EDC based on LMP
- 11-14 wk GA: NTUS
  - measures the amount of fluid behind the neck of the fetus
  - early screen for Trisomy 21 (may also detect cardiac and other aneuploidies like Turner's syndrome)
  - NT measurement is necessary for the FTS and IPS Part 1
- 18-20 wk GA: Growth and Anatomy U/S (margin of error ± 10 d)
- earlier or subsequent ultrasounds performed when medically indicated

Symphysis Fundal Height (SFH)

12 wk	Uterine fundus at pubic symphysis
20 wk	Fundus at umbilicus, SFH should be within 2 cm of GA between 20-36 wk
37 wk	Fundus at sternum

Non-Invasive Prenatal Testing (NIPT)
- non-invasive screening for Down syndrome and other chromosomal abnormalities in spontaneous singleton pregnancies
- analyses maternal blood for circulating cell free fetal DNA (ccffDNA) at 10 wk GA

Advantage
- high sensitivity (98-99%), FP<2%

Disadvantages
- less sensitive for Trisomy 18 and 13
- low specificity (all positive results must be confirmed with amniocentesis)
- does not screen for oNTD

Table 4. Comparison of FTS, MSS, and IPS

FTS	MSS	IPS
11-14 wk	15-20 wk	11-13 wk U/S-Nuchal Translucency 11-14 wk: FTS blood 15-20 wk : MSS blood including inhibin
Risk estimate for 1. Down syndrome (Trisomy 21): increased NT, increased β-hCG, decreased PAPP-A 2. Trisomy 18 : increased NT, decreased PAPP-A, decreased β-hCG Note: does not measure risk of open neural tube defect (oNTD) and should be combined with MSAFP at 15-20 wk Useful where patient wants results within the first trimester More accurate estimate of Down syndrome risk than MSS, sensitivity ~85% (when combined with age) 5% false positive rate Patients with positive screen should be offered CVS, amniocentesis, or NIPT (covered in some provinces, self-pay in others)	Risk estimate for 1. oNTD: increased MSAFP (sensitivity 80-90%) 2. Trisomy 21: decreased MSAFP, increased β-hCG, decreased μE3 (sensitivity 65%) 3. Trisomy 18: decreased MSAFP, decreased β-hCG, decreased μE3, decreased inhibin (sensitivity 80%) Only offered alone if patient missed the time window for IPS or FTS 8% baseline false positive rate for Trisomy 21, lower for oNTD and Trisomy 18 Patients with positive screen should be offered U/S, amniocentesis, or NIPT (covered in some provinces, self-pay in others)	Risk estimate for oNTD, Trisomy 21, Trisomy 18 Sensitivity ~85-90% 2% false positive rate Patients with positive screen should be offered U/S and/or amniocentesis or NIPT (covered in some provinces, self-pay in others)

Note: In twins, FTS, MSS, and IPS are not applicable; screen with NT for chromosomal abnormalities and MSAFP for oNTDs

Diagnostic Tests

Indications
- age >35 yr (increased risk of chromosomal anomalies)
- risk factors in current pregnancy
  - abnormal U/S
  - abnormal prenatal screen (IPS, FTS, or MSS)
- past history/family history of
  - previous pregnancy or family history of chromosomal anomaly or genetic disease
  - either parent a known carrier of a genetic disorder or balanced translocation
  - consanguinity
  - >3 spontaneous abortions

AMNIOCENTESIS
- U/S-guided transabdominal extraction of amniotic fluid

Indications
- identification of genetic anomalies (15-16 wk gestation) as per indications above
- confirmation of positive NIPT testing
- positive FTS/IPS
- assessment of fetal lung maturity (T3) via the L/S ratio (lecithin:sphingomyelin)
  - if >2:1, RDS is less likely to occur

Advantages
- also screens for oNTD (acetylcholinesterase and amniotic AFP) – 96% accurate
- in women >35 yr, the risk of chromosomal anomaly (1/180) is greater than the risk of miscarriage from the procedure
- more accurate genetic testing than CVS

Disadvantages
- 1/400-1/500 risk of spontaneous abortion
- results take 14-28 d; FISH can be done on chromosomes X, Y, 21, 13, 18 to give preliminary results in 48 h

Compared to CVS, amniocentesis has a higher accuracy of prenatal cytogenetic diagnosis (99.8% vs. 97.5%) and lower risk of spontaneous abortion (0.5% vs. 1-2%)

Risk Factors for Neural Tube Defects

GRIMM
Genetics: family history of NTD (risk of having second child with NTD is increased to 2-5%), consanguinity, chromosomal (characteristic of Trisomy 13, 18, and 21)
Race: European Caucasians > African Americans, 3-fold higher in Hispanics
Insufficient vitamins: zinc and folate
Maternal chronic disease (e.g. DM)
Maternal use of antiepileptic drugs

General population risk for NTD is 0.1%

CHORIONIC VILLUS SAMPLING
- biopsy of fetal-derived chorion using a transabdominal needle or transcervical catheter at 10-12 wk

Advantages
- enables pregnancy to be terminated earlier than with amniocentesis
- rapid karyotyping and biochemical assay within 48 h, including FISH analysis
- high sensitivity and specificity

Disadvantages
- 1-2% risk of spontaneous abortion
- does not screen for oNTD
- 1-2% incidence of genetic mosaicism "false negative" results

ISOIMMUNIZATION SCREENING

Definition
- isoimmunization: antibodies (Ab) produced against a specific RBC antigen (Ag) as a result of antigenic stimulation with RBC of another individual

Etiology
- maternal-fetal circulation normally separated by placental barrier, but sensitization can occur and can affect the current pregnancy, or more commonly, future pregnancies
- Anti-Rh Ab produced by a sensitized Rh-negative mother can lead to fetal hemolytic anemia
- Risk of isoimmunization of an Rh-negative mother with an Rh-positive ABO-compatible infant is 16%
- sensitization routes
- incompatible blood transfusions
- previous fetal-maternal transplacental hemorrhage (e.g. ectopic pregnancy, abruption)
- invasive procedures in pregnancy (e.g. prenatal diagnosis, cerclage, D&C)
- any type of abortion
- labour and delivery

Rh Antibody Titre
A positive titre (≥1:16) indicates an increased risk of fetal hemolytic anemia

Investigations
- Screening with indirect Coombs test at first visit for blood group, Rh status, and antibodies
- Kleihauer-Betke test used to determine extent of fetomaternal hemorrhage by estimating volume of fetal blood volume that entered maternal circulation
- detailed U/S for hydrops fetalis

Prophylaxis
- exogenous Rh IgG (Rhogam® or WinRho®) binds to Rh antigens of fetal cells and prevents them from contacting maternal immune system
- Rhogam® (300 μg) given to all Rh negative and antibody screen negative women in the following scenarios
  - routinely at 28 wk GA (provides protection for ~12 wk)
  - within 72 h of the birth of an Rh positive fetus
  - with a positive Kleihauer-Betke test
  - with any invasive procedure in pregnancy (CVS, amniocentesis)
  - in ectopic pregnancy
  - with miscarriage or therapeutic abortion
  - with an antepartum hemorrhage
- if Rh negative and Ab screen positive, follow mother with serial monthly Ab titres throughout pregnancy ± serial amniocentesis as needed (Rhogam® has no benefit)

Standard dose of 300 μg of Rhogam® sufficient for 30 mL of fetal blood. Give additional 10 μg of Rhogam® for every mL of fetal blood over 30 mL

Investigations
- MCA dopplers are done to assess degree of fetal anemia or if not available bilirubin is measured by serial amniocentesis to assess the severity of hemolysis
- cordocentesis for fetal Hb should be used cautiously (not first line)

Treatment
- falling biliary pigment warrants no intervention (usually indicative of either unaffected or mildly affected fetus)
- intrauterine transfusion of O-negative pRBCs may be required for severely affected fetus or early delivery of the fetus for exchange transfusion

Complications
- anti-Rh IgG can cross the placenta and cause fetal RBC hemolysis resulting in fetal anemia, CHF, edema, ascites
- severe cases can lead to fetal hydrops (edema in at least two fetal compartments due to fetal heart failure secondary to anemia) or erythroblastosis fetalis (moderate to severe immune-mediated hemolytic anemia)

Counselling of the Pregnant Woman

Nutrition

- Canada's Food Guide to Healthy Eating suggests
  - 3-4 servings of milk products daily (greater if multiple gestation)
  - a daily caloric increase of ~100 cal/d in the 1st trimester, ~300 cal/d in the second and third trimesters and ~450 cal/d during lactation
  - daily multivitamin should be continued in the 2nd trimester for women who do not consume an adequate diet; otherwise routine vitamin supplementation is not necessary (avoid excess vitamin A)
- nutrients important during pregnancy
  - folate: 0.4 mg/d for first 12 wk (5 mg/d if high risk)
    - supports increase in blood volume, growth of maternal and fetal tissue, decreases incidence of NTD
    - foods rich in folic acid include: spinach, lentils, chick peas, asparagus, broccoli, peas, brussels sprouts, corn, and oranges
  - calcium: 1200-1500 mg/d
    - maintains integrity of maternal bones, skeletal development of fetus, breast milk production
  - vitamin D: 1,000 IU
    - promotes calcium absorption
  - iron: 0.8 mg/d in T1, 4-5 mg/d in T2, and >6 mg/d in T3
    - supports maternal increase in blood cell mass, supports fetal and placental tissue
    - required amounts exceed normal body stores and typical intake, and therefore need supplemental iron
    - iron is the only known nutrient for which requirements during pregnancy cannot be met by diet alone (see *Iron Deficiency Anemia*, OB26)
  - essential fatty acids – supports fetal neural and visual development
    - contained in vegetable oils, margarines, peanuts, fatty fish

Caffeine

- diuretic and stimulant that readily crosses placenta
- less than 300 mg/d is not thought to contribute to miscarriage or preterm birth (ACOG)
  - relationship between caffeine and IUGR is unknown (ACOG)
  - SOGC states 1-2 cups/d are safe during pregnancy

Sources of Caffeine
- 5 oz cup coffee: 40-180 mg
- 5 oz brewed tea: 20-90 mg
- 12 oz cola: 46 mg
- Red Bull®: 67 mg
- Dark chocolate bar: 10 mg
- 8 oz hot chocolate: 5 mg

Herbal Teas and Preparations

- not enough scientific information about safety of various herbs and herbal products to recommend their use during pregnancy
- some herbal teas can have toxic or pharmacological effects on the mother or fetus
- chamomiles have been reported to exhibit adverse effects on the uterus

Herbal Teas Considered Safe in Moderation (2-3 cups/d)
- Citrus peel
- Ginger
- Lemon balm
- Linden flower – not with prior cardiac condition
- Orange peel
- Rose hip

Foodborne Illnesses

- microbiological contamination of food may occur through cross-contamination and/or improper food handling
  - listeriosis (*Listeria monocytogenes*) and toxoplasmosis (*Toxoplasma gondii*) are of concern during pregnancy
  - avoid consumption of raw meats, fish, poultry, raw eggs, and unpasteurized dairy products
  - avoid unpasteurized soft cheeses, deli meats, smoked salmon, and pates as they may be sources of *Listeria*
- chemical contamination of food
  - current guideline for mercury of 0.5 ppm in fish is not considered harmful for the general population, including pregnant women
  - Health Canada advises pregnant women to limit consumption of top predator fish such as shark, swordfish, king mackeral, tilefish

Lifestyle

- exercise under physician guidance
- absolute contraindications
  - ruptured membranes, preterm labour, hypertensive disorders of pregnancy, incompetent cervix, IUGR, multiple gestations (>3), placenta previa after 28th wk, persistent 2nd or 3rd trimester bleeding, uncontrolled type I DM, uncontrolled thyroid disease, or other serious cardiovascular, respiratory, or systemic disorder

- relative contraindications
  - previous preterm birth, mild/moderate cardiovascular or respiratory disorder, anemia (Hb ≤10 g/dL), malnutrition or eating disorder, twin pregnancy after 28th wk, other significant medical conditions
- weight gain: optimal gain depends on pre-pregnancy BMI (varies from 6.8-18.2 kg)
- work: strenuous work, extended hours and shift work during pregnancy may be associated with greater risk of low birth weight, prematurity, and spontaneous abortion
- air travel is acceptable in second trimester; airline cut off for travel is 36-38 wk gestation depending on the airline to avoid giving birth on the plane
- sexual intercourse: may continue, except in patients at risk for: abortion, preterm labour, or placenta previa; breast stimulation may induce uterine activity and is discouraged in high-risk patients near term
- smoking: assist/encourage to reduce or quit smoking
  - increased risk of decreased birth weight, placenta previa/abruption, spontaneous abortion, preterm labour, stillbirth
- alcohol: no amount of alcohol is safe in pregnancy; encourage abstinence from alcohol during pregnancy; alcohol increases incidence of abortion, stillbirth, and congenital anomalies
  - fetal alcohol syndrome (see Pediatrics, P25)
- cocaine: microcephaly, growth retardation, prematurity, abruptio placentae

Expected Weight Gain

BMI (kg/m^2)	Weight (kg)
<19	12.7-18.2
19-25	11.3-15.9
>25	6.8-11.3

General Rule: 1-3.5 kg/wk during T1, then 0.45 kg/wk until delivery

Medications

- most drugs cross the placenta to some extent
- very few drugs are teratogenic, but very few drugs have proven safety in pregnancy
- use any drug with caution and only if necessary
- analgesics: acetaminophen preferable to ASA or ibuprofen

Drug Resources During Pregnancy and Breastfeeding
- Motherisk at the Hospital for Sick Children in Toronto: www.motherisk.org
- Hale T. Medications and mothers' milk, 11th ed. Pharmasoft Publishing, 2004

Table 5. Documented Adverse Effects, Contraindicated

Contraindicated Medication	Adverse Effect
ACEI	Fetal renal defects, IUGR, oligohydramnios
Tetracycline	Stains infant's teeth, may affect long bone development
Retinoids (e.g. Accutane®)	CNS, craniofacial, cardiac, and thymic anomalies
Misoprostol	Mobius syndrome (congenital facial paralysis with or without limb defects, spontaneous abortion, preterm labour)

Table 6. Documented Adverse Effects, Weigh Benefits vs. Risks, and Consider Medication Change

Medication	Adverse Effect
Phenytoin	Fetal hydantoin syndrome in 5-10% (IUGR, mental retardation, facial dysmorphogenesis, congenital anomalies)
Valproate	oNTD in 1%
Carbamazepine	oNTD in 1-2%
Lithium	Ebstein's cardiac anomaly, goitre, hyponatremia
Warfarin	Increased incidence of spontaneous abortion, stillbirth, prematurity, IUGR, fetal warfarin syndrome (nasal hypoplasia, epiphyseal stippling, optic atrophy, mental retardation, intracranial hemorrhage)
Erythromycin	Maternal liver damage (acute fatty liver)
Sulpha drugs	Anti-folate properties, therefore theoretical risk in T1; risk of kernicterus in T3
Chloramphenicol	Grey baby syndrome (fetal circulatory collapse 2° to toxic accumulation)

Immunizations

Intrapartum
- administration is dependent on the risk of infection vs. risk of immunization complications
- safe: tetanus toxoid, diphtheria, influenza, hepatitis B, pertussis
- avoid live vaccines (risk of placental and fetal infection): polio, measles/mumps/rubella, varicella
- contraindicated: oral typhoid

Postpartum
- rubella vaccine for all non-immune mothers
- hepatitis B vaccine should be given to infant within 12 h of birth if maternal status unknown or positive – follow-up doses at 1 and 6 mo
- human papillomavirus (HPV) vaccine – if meets criteria
- safe: tetanus toxoid, diphtheria, influenza, hepatitis B, pertussis, varicella

Radiation

- ionizing radiation exposure is considered teratogenic at high doses
  - if indicated for maternal health, should be done
- imaging not involving direct abdominal/pelvic high dosage is not associated with adverse effects
  - higher dosage to fetus: plain x-ray of lumbar spine/abdomen/pelvis, barium enema, CT abdomen, pelvis, lumbar spine
- most investigations involve minimal radiation exposure
- radioactive isotopes of iodine are contraindicated
- no known adverse effects from U/S or MRI (long-term effects of gadolinium unknown, avoid if possible)

Table 7. Approximate Fetal Doses from Common Diagnostic Procedures

Examination	Estimated Fetal Dose (rad)	Number of Exams Safe in Pregnancy
Plain Film		
Abdomen	0-14	35
Pelvis	0-11	45
Lumbar spine	0-17	29
Thoracic spine	0.009	555
Chest (2 views)	<0.001	5000
CT		
Abdomen	0-8	6
Pelvis	2-5	2
Lumbar spine	0-24	20
Chest	0.006	833

Adapted from: Cohen-Kerem, et al. 2005 and Valentin 2000

Radiation in Pregnancy
- Necessary amount to cause miscarriage: >5 rads
- Necessary amount to cause malformations: >20-30 rads

Antenatal Fetal Surveillance

Fetal Movements

- patients will generally first notice fetal movement ("quickening") at 18-20 wk in primigravidas; can occur 1-2 wk earlier in multigravidas; can occur 1-2 wk later if placenta is implanted on the anterior wall of uterus
- if the patient is concerned about decreased fetal movement, she is counselled to choose a time when the fetus is normally active to count movements (usually recommended after 26 wk)
- all high risk women should be told to do FM counts
  - if there is a subjective decrease in fetal movement, try drinking juice, eating, changing position, or moving to a quiet room and count for 2 h; ≥6 movements in 2 h expected
  - if there are <6 movement counts in 2 h, patient should present to labour and delivery triage

DDx of Decreased Fetal Movements

DASH
Death of fetus
Amniotic fluid decreased
Sleep cycle of fetus
Hunger/Thirst

NON-STRESS TEST

Definition
- FHR tracing ≥20 min using an external Doppler to assess FHR and its relationship to fetal movement (see *Fetal Monitoring in Labour*, OB35)

Normal NST: 2 accels, >15 bpm from baseline, lasting >15 s in 20 min

Indication
- any suggestion of uteroplacental insufficiency or suspected compromise in fetal well-being

Table 8. Classification of Antepartum Non-Stress Test

Parameter	Normal NST (Previously "Reactive")	Atypical NST (Previously "Non-Reactive")	Abnormal NST (Previously "Non-Reactive")
Baseline	110-160 bpm	100-110 bpm or >160 bpm for <30 min Rising baseline	Bradycardia <100 bpm Tachycardia >160 for >30 min Erratic baseline
Variability	6-25 bpm (moderate) ≤5 (absent or minimal) for <40 min	5 (absent or minimal) for 40-80 min	≤5 for 80 min Sinusoidal 25 bpm for >10 min
Decelerations	None or occasional variable <30 s	Variable decelerations 30-60 s duration	Variable decelerations >60 s Late deceleration(s)
Accelerations in Term Fetus	2 accelerations with acme of ≥15 bpm, lasting 15 s over <40 min of testing	2 accelerations with acme of ≥15 bpm, lasting 15 s in 40-80 min	<2 accelerations with acme of ≥15 bpm, lasting 15 s in >80 min
Accelerations in Preterm Fetus (<32 wk)	>2 accelerations with acme of >10 bpm, lasting 10 s in <40 min	<2 accelerations with acme of >10 bpm, lasting 10 s in 40-80 min	<2 accelerations with acme of >10 bpm, lasting 10 s in >80 min
Action	FURTHER ASSESSMENT OPTIONAL, based on total clinical picture	FURTHER ASSESSMENT REQUIRED	URGENT ACTION REQUIRED An overall assessment of the situation and further investigation with U/S or BPP is required; some situations will require delivery

Adapted from: SOGC, Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline, September 2007

Operating Characteristics
- false positive rate depends on duration; false negative rate = 0.2-0.3%

Interpretation
- normal: at least 2 accelerations of FHR >15 bpm from the baseline lasting >15 s, in 20 min
- abnormal: <2 accelerations of FHR in 40 min
- if no observed accelerations or fetal movement in the first 20 min, stimulate fetus (fundal pressure, acoustic/vibratory stimulation) and continue monitoring for 30 min
- if NST abnormal, then perform BPP

BIOPHYSICAL PROFILE

Definition
- U/S assessment of the fetus ± NST

Indications
- abnormal or atypical NST
- post-term pregnancy
- decreased fetal movement
- IUGR
- any other suggestion of fetal distress or uteroplacental insufficiency

Operating Characteristics
- false positive rate ≤30%, false negative rate = 0.1%

Table 9. Scoring of the BPP

Parameter	Reassuring (2 points)
Limb Extension then Flexion	At least one episode of limb extension followed by flexion
AFV*	Fluid pocket of 2 cm in 2 axes
Movement	Three discrete movements
Breathing	At least one episode of breathing lasting at least 30 s

*AFV is a marker of chronic hypoxia, all other parameters indicate acute hypoxia

Reassuring BPP (8/8)

LAMB
Limb extension + flexion
AFV 2 cm x 2 cm
Movement (3 discrete)
Breathing (one episode x 30 s)

Interpretation
- 8: perinatal mortality rate 1:1,000; repeat BPP as clinically indicated
- 6: perinatal mortality 31:1,000; repeat BPP in 24 h
- 0-4: perinatal mortality rate 200:1,000; deliver fetus if benefits of delivery outweigh risks

Obstetrical Hemorrhage

Definition
- vaginal bleeding from 20 wk to term

Differential Diagnosis
- bloody show (shedding of cervical mucous plug) – most common etiology in T3
- placenta previa
- abruptio placentae – most common pathological etiology in T3
- vasa previa
- cervical lesion (cervicitis, polyp, ectropion, cervical cancer)
- uterine rupture
- other: bleeding from bowel or bladder, placenta accreta, abnormal coagulation

Key Questions to Ask in Antepartum Hemorrhage
- How much bleeding?
- Are there contractions/cramping/pain?
- Description? Colour, clotting, etc.

Table 10. Comparison of Placenta Previa vs. Abruptio Placentae

	Placenta Previa	Abruptio Placentae
Definition	Abnormal location of the placenta near, partially, or completely over the internal cervical os	Premature separation of a normally implanted placenta after 20 wk GA
Etiology	Idiopathic	Idiopathic
Epidemiology	0.5-0.8% of all pregnancies	1-2% of all pregnancies
Risk Factors	• History of placenta previa (4-8% recurrence risk) • Multiparity • Increased maternal age • Multiple gestation • Uterine tumour (e.g. fibroids) or other uterine anomalies • Uterine scar due to previous abortion, C/S, D&C, myomectomy	• Previous abruption (recurrence rate 5-16%) • Maternal HTN (chronic or gestational HTN in 50% of abruptions) or vascular disease • Cigarette smoking (>1 pack/d), excessive alcohol consumption, cocaine • Multiparity and/or maternal age >35 yr • PPROM • Rapid decompression of a distended uterus (polyhydramnios, multiple gestation) • Uterine anomaly, fibroids • Trauma (e.g. motor vehicle collision, maternal battery)
Bleeding	PAINLESS	Usually PAINFUL

Levels of Abnormal Placental Invasion
Placenta Accreta: AT myometrium (most common)
Placenta Increta: INTO myometrium
Placenta Percreta: PASSES through myometrium

Placenta Previa

Definition
- placenta implanted in the lower segment of the uterus, presenting ahead of the leading pole of the fetus
- the distance of the placental edge from the internal os is described in "millimetres away" from the internal os or "millimetres of overlap" over the internal os
- greater than 20 millimetres of overlap over the internal os in the third trimester of pregnancy is highly predictive of the need for a C/S
- any degree of overlap after 35 wk is an indication for a C/S

Clinical Features
- PAINLESS bright red vaginal bleeding (recurrent), may be minimized and cease spontaneously, but can become catastrophic
- mean onset of bleeding is 30 wk GA, but onset depends on degree of previa
- **physical exam**
  - uterus soft and non-tender
  - presenting fetal part high or displaced
  - FHR usually normal
  - shock/anemia correspond to degree of apparent blood loss
- **complications**
  - fetal
    - perinatal mortality low but still higher than with a normal pregnancy
    - prematurity (bleeding often dictates early C/S)
    - intrauterine hypoxia (acute or IUGR)
    - fetal malpresentation
    - PPROM
    - risk of fetal blood loss from placenta, especially if incised during C/S
  - maternal
    - <1% maternal mortality
    - hemorrhage and hypovolemic shock, anemia, acute renal failure, pituitary necrosis (Sheehan syndrome)
    - placenta accreta – especially if previous uterine surgery, anterior placenta previa
    - hysterectomy

Investigations
- transvaginal U/S is more accurate than transabdominal U/S at diagnosing placenta previa at any gestational age
- if the placenta lies between 20 mm of overlap and 20 mm away from the internal os after 20 wk transvaginal ultrasounds should be repeated in the third trimester as continued change in the placental location is likely

Do NOT perform a vaginal exam until placenta previa has been ruled out by U/S

Management
- goal: keep pregnancy intrauterine until the risk of delivery < risk of continuing pregnancy
- stabilize and monitor
  - maternal stabilization: large bore IV with hydration, O2 for hypotensive patients
  - maternal monitoring: vitals, urine output, blood loss, blood work (hematocrit, CBC, INR/PTT, platelets, fibrinogen, FDP, type and cross match)
  - electronic fetal monitoring
  - U/S assessment: when fetal and maternal condition permit, determine fetal viability, gestational age, and placental status/position
- Rhogam® if mother is Rh negative
  - Kleihauer-Betke test to determine extent of fetomaternal transfusion so that appropriate dose of Rhogam® can be given
- GA <37 wk and minimal bleeding: expectant management
  - admit to hospital
  - limited physical activity, no douches, enemas, or sexual intercourse
  - consider corticosteroids for fetal lung maturity
  - delivery when fetus is mature or hemorrhage dictates
- GA ≥37 wk, profuse bleeding, or L/S ratio is >2:1 – deliver by C/S

Abruptio Placentae

Clinical Features
- **classification**
  - total (fetal death inevitable) vs. partial
  - external/revealed/apparent: blood dissects downward toward cervix
  - internal/concealed (20%): blood dissects upward toward fetus
  - most are mixed
- **presentation**
  - **usually PAINFUL** (80%) vaginal bleeding (bleeding not always present if abruption is concealed), uterine tenderness, uterine contractions
  - pain: sudden onset, constant, localized to lower back and uterus
  - shock/anemia out of proportion to apparent blood loss
  - ± fetal distress, fetal demise (15% present with demise), bloody amniotic fluid (fetal presentation typically normal)
  - ± coagulopathy

Abruptio placentae is the most common cause of DIC in pregnancy

Complications
- fetal complications: perinatal mortality 25-60%, prematurity, intrauterine hypoxia
- maternal complications: <1% maternal mortality, DIC (in 20% of abruptions), acute renal failure, anemia, hemorrhagic shock, pituitary necrosis (Sheehan syndrome), amniotic fluid embolus

Investigations
- clinical diagnosis, U/S not sensitive for diagnosing abruption (sensitivity = 15%)

Management
- maternal stabilization: large bore IV with hydration, O2 for hypotensive patients
- maternal monitoring: vitals, urine output, blood loss, blood work (hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match)
- EFM
- blood products on hand (red cells, platelets, cryoprecipitate) because of DIC risk
- Rhogam® if Rh negative
  - Kleihauer-Betke test may confirm abruption
- mild abruption:
  - GA <37 wk: use serial Hct to assess concealed bleeding, deliver when fetus is mature or when hemorrhage dictates
  - GA ≥37 wk: stabilize and deliver
- moderate to severe abruption:
  - hydrate and restore blood loss and correct coagulation defect if present
  - vaginal delivery if no contraindication and no evidence of fetal or maternal distress OR fetal demise
  - C/S if live fetus and fetal or maternal distress develops with fluid/blood replacement, labour fails to progress or if vaginal delivery otherwise contraindicated

Kleihauer-Betke Test
Quantifies fetal cells in the maternal circulation

Vasa Previa

Definition
- unprotected fetal vessels pass over the cervical os; associated with velamentous insertion of cord into membranes of placenta or succenturiate (accessory) lobe

Epidemiology
- 1 in 5,000 deliveries – higher in twin pregnancies

Clinical Features
- PAINLESS vaginal bleeding and fetal distress (tachy- to bradyarrhythmia)
- 50% perinatal mortality, increasing to 75% if membranes rupture (most infants die of exsanguination)

Investigations
- Apt test (NaOH mixed with the blood) can be done immediately to determine if the source of bleeding is fetal (supernatant turns pink) or maternal (supernatant turns yellow)
- Wright stain on blood smear and look for nucleated red blood cells (in cord, not maternal blood)

Management
- emergency C/S (since bleeding is from fetus, a small amount of blood loss can have catastrophic consequences)

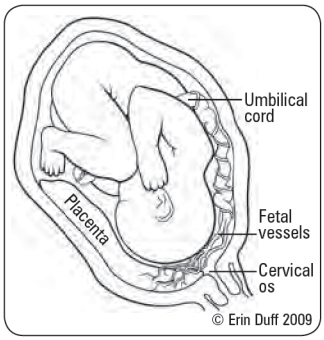


Figure 3. Vasa previa

Obstetrical Complications

Preterm Labour

Definition
- uterine contractions intense and frequent enough to cause cervical effacement and dilation between 20 and 37 wk gestation

Etiology
- idiopathic (most common)
- maternal: infection (recurrent pyelonephritis, untreated bacteriuria, chorioamnionitis), HTN, DM, chronic illness, mechanical factors, previous obstetric, gynecological, and abdominal surgeries, socio-environmental (poor nutrition, smoking, drugs, alcohol, stress)
- maternal-fetal: PPROM (common), polyhydramnios, placenta previa, placental abruption, or placental insufficiency
- fetal: multiple gestation, congenital abnormalities of fetus, fetal hydrops, stress
- uterine: incompetent cervix, excessive enlargement (hydramnios, multiple gestation), malformations (leiomyomas, septate uterus, mullerian duct abnormalities, fibroids)

Epidemiology
- preterm labour complicates about 12% of pregnancies, most common cause of neonatal mortality in US

Risk Factors and Prediction of PTL
- maternal risk scoring using above etiologies fails to identify up to 70% of preterm deliveries and is therefore of limited use
- prior history of spontaneous PTL: most important risk factor
- prior history cervical excisions (LEEPs/cone biopsy) or mechanical dilatation (D&C)
- cervical length: measured by transvaginal U/S (cervical length >30 mm has high negative predictive value for PTL before 34 wk)
- identification of bacterial vaginosis (Rx metronidazole if symptomatic or high-risk for PTL) and ureaplasma urealyticum (Rx erythromycin) infections: routine screening not supported by current data but it is reasonable to screen high risk women
- family history of preterm birth
- fetal fibronectin: a glycoprotein in amniotic fluid and placental tissue functioning to maintain integrity of chorionic-decidual interface in asymptomatic women
  - positive if >50 ng/mL
  - in symptomatic women (i.e. preterm contractions), fetal fibronectin is most effectively combined with U/S detecting cervical length
  - if cervical length is not short and fetal fibronectin is negative, preterm labour is highly unlikely

Positive fetal fibronectin in cervicovaginal fluid (>50 ng/mL) at 24 wk gestation predicted spontaneous PTL at <34 wk with sensitivity of 23%, specificity of 97%, PPV of 25%, NPV of 96%

Clinical Features
- regular contractions (2 in 10 min, >6/h)
- cervix >1 cm dilated, >80% effaced, or length <2.5 cm

Management
A. Initial
- transfer to appropriate facility if stable
- hydration (NS at 150 mL/h)
- bed rest in LLDP
- sedation (morphine)
- avoid repeated pelvic exams (increased infection risk)
- U/S examination of fetus (GA, BPP, position, placenta location, estimated fetal weight)
- prophylactic antibiotics; (for GBS) important to consider if PPROM (e,g, erythromycin controversial but may help to delay delivery),

B. Suppression of Labour – Tocolysis
- does not inhibit preterm labour completely, but may delay delivery (used for <48 h) to allow for betamethasone valerate (Celestone®) and/or transfer to appropriate centre for care of the premature infant
- requirements (all must be satisfied)
  - preterm labour
  - live, immature fetus, intact membranes, cervical dilatation of <4 cm
  - absence of maternal or fetal contraindications
- contraindications
  - maternal: bleeding (placenta previa or abruption), maternal disease (HTN, DM, heart disease), preeclampsia or eclampsia, chorioamnionitis
  - fetal: erythroblastosis fetalis, severe congenital anomalies, fetal distress/demise, IUGR, multiple gestation (relative)
- agents
  - calcium channel blockers: nifedipine
    - 20 mg PO loading dose followed by 20 mg PO 90 min later
    - 20 mg can be continued q3-8h for 72 h or to a max of 180 mg
    - 10 mg PO q20min x 4 doses
    - contraindications: nifedipine allergy, hypotension, hepatic dysfunction, concurrent beta-mimetics or MgSO4 use, transdermal nitrates, or other antihypertensive medications
  - prostaglandin synthesis inhibitors: indomethacin
    - 1st line for early preterm labour (<30 wk GA) or polyhydramnios
    - 50-100 mg PR loading dose followed by 50 mg q6h x 8 doses
  - magnesium sulphate was previously used for tocolysis; currently, its primary use in obstetrics is limited to neuroprotection or prevention of eclampsia
    - indicated if preterm delivery is inevitable between 24 and 31+6 wks GA for neuroprotection
    - 4 g IV loading dose followed by 1g q1h maintenance until birth

Tocolytics for Preterm Premature Rupture of Membranes
Cochrane DB Syst Rev 2014;2:CD007062
Purpose: To assess the potential benefits and harms of tocolysis in women with PPROM.
Selection Criteria: Pregnant women with singleton pregnancies and PPROM (23-36 wk and 6 d GA).
Results: 8 studies with 408 women total. Prophylactic tocolysis with PPROM was associated with increased overall latency, without additional benefits for maternal/neonatal outcomes. For women with PPROM before 34 wk, there was a significantly increased risk of chorioamnionitis in women who received tocolysis. Neonatal outcomes were not significantly different.
Conclusion: Although there are limitations to the studies, there is currently insufficient evidence to support tocolytic therapy for women with PPROM, as there was an increase in maternal chorioamnionitis without significant benefits to the infant.

C. Enhancement of Fetal Pulmonary Maturity
- betamethasone valerate (Celestone®) 12 mg IM q24h x 2 doses or dexamethasone 6 mg IM q12h x 4 doses
  - 28-34 wk GA: reduces incidence of RDS
  - 24-28 wk GA: reduces severity of RDS, overall mortality and rate of IVH
  - specific maternal contraindications: active TB

D. Cervical Cerclage
- definition: placement of cervical sutures at the level of the internal os, usually at the end of the first trimester or in the second trimester and removed in the third trimester
- indications: cervical incompetence (i.e. cervical dilation and effacement in the absence of increased uterine contractility)
  - emerging evidence indicates that progesterone suppositories are superior to cerclage in preventing preterm labour not due to cervical incompetence; (neither is effective in multiple gestations)
- diagnosis of cervical incompetence
  - obstetrical Hx: silent cervical dilation, 2nd trimester losses, procedures on cervix
  - ability of cervix to hold an inflated Foley catheter during a hysterosonogram
- proven benefit in the prevention of PTL in women with primary structural abnormality of the cervix (e.g. conization of the cervix, connective tissue disorders)

Cerclage for Short Cervix on Ultrasonography in Women With Singleton Gestations and Previous Preterm Birth
Obstet Gynecol 2011;117:663-671
Purpose: To determine if cerclage prevents preterm birth (<35 wk gestation) and perinatal mortality and morbidity among women with previous spontaneous preterm birth, asymptomatic singleton gestation, and short cervical length (<25 mm before 24 wk gestation) on transvaginal ultrasonography.
Methods: Meta-analysis of randomized trials identified using searches on MEDLINE, PUBMED, EMBASE, and the Cochrane Library.
Results: 5 trials included. Preterm birth was significantly lower among women receiving cerclage vs. those not (RR = 0.70, 95% CI 0.55-0.89). Cerclage also significantly reduced preterm birth before 24, 28, 32, and 37 wk gestation. Perinatal mortality and morbidity were significantly lower in the cerclage group (RR = 0.64, 95% CI 0.45-0.91).
Conclusions: Cerclage significantly prevents preterm birth and perinatal mortality and morbidity in this specific group of women.

Prognosis
- prematurity is the leading cause of perinatal morbidity and mortality
- 30 wk or 1,500 g (3.3 lb) = 90% survival
- 33 wk or 2,000 g (4.4 lb) = 99% survival
- morbidity due to asphyxia, hypoxia, sepsis, RDS, intraventricular cerebral hemorrhage, thermal instability, retinopathy of prematurity, bronchopulmonary dysplasia, necrotizing enterocolitis

Prevention of Preterm Labour
- currently there are no agents approved by Health Canada to arrest preterm labour
- preventative measures: good prenatal care, identify pregnancies at risk, treat silent vaginal infection or UTI, patient education
- transvaginal ultrasound of cervical length is recommended only for high-risk pregnancies and only before 30 weeks GA

Premature Rupture of Membranes

Definitions
- PROM: rupture of membranes prior to labour at any GA
- prolonged ROM: >24 h elapsed between rupture of membranes and onset of labour
- preterm ROM: ROM occurring before 37 wk gestation
- PPROM: rupture of membranes before 37 wk AND prior to onset of labour

Risk Factors
- maternal: multiparity, cervical incompetence, infection (cervicitis, vaginitis, STI, UTI), family history of PROM, low socioeconomic class/poor nutrition
- fetal: congenital anomaly, multiple gestation
- other risk factors associated with PTL

Clinical Features
- history of fluid gush or continued leakage

Investigations
- sterile speculum exam (avoid introduction of infection)
  - pooling of fluid in the posterior fornix
  - may observe fluid leaking out of cervix on cough/Valsalva ("cascade")
- nitrazine (amniotic fluid turns nitrazine paper blue)
  - low specificity as can be positive with blood, urine, or semen
- ferning (high salt in amniotic fluid evaporates, looks like ferns under microscope)
- U/S to rule out fetal anomalies, assess GA, and BPP

Membrane Status determined by
- Pooling of fluid on speculum exam
- Increased pH of vaginal fluid (nitrazine test)
- Ferning of fluid under light microscopy
- Decreased AFV on U/S

Management
- admit for expectant management and monitor vitals q4h, daily BPP and WBC count
- avoid introducing infection with examinations (do NOT do a bimanual exam)
- cultures (lower vagina for GBS)
- assess fetal lung maturity by L/S ratio of amniotic fluid
  - consider administration of betamethasone valerate (Celestone®) to accelerate maturity if <32 wk and no evidence of infection
  - consider tocolysis for 48 h to permit administration of steroids if PPROM induces labour
- if not in labour or labour not indicated, consider antibiotics (controversial)
  - studies show broad spectrum coverage increases the time to onset of labour from PROM by 5-7 d with no increase in maternal or neonatal morbidity or mortality
- deliver urgently if evidence of fetal distress and/or chorioamnionitis

L/S Ratio (Lecithin:Sphingomyelin Ratio)
Lecithin levels increase rapidly after 35 wk gestation, whereas sphingomyelin levels remain relatively constant. The L/S ratio is a measure of fetal lung maturity – less than 2:1 indicates pulmonary immaturity. Presence of blood or meconium in the amniotic fluid can affect the ratio

Table 11. PROM Management

Degree of Prematurity	Management
<24 wk	Consider termination (poor outcome due to pulmonary hypoplasia)
24-25 wk	Individual consideration with counselling of parents regarding risks to preterm infants
26-34 wk	Expectant management as prematurity complications are significant
34-36 wk	"Grey zone" where risk of death from RDS and neonatal sepsis is the same
≥37 wk	Induction of labour since the risk of death from sepsis is greater than RDS

Prognosis
- varies with gestational age
- 90% of women with PROM at 28-34 wk GA go into spontaneous labour within 1 wk
- 50% of women with PROM at <26 wk GA go into spontaneous labour within 1 wk
- complications: cord prolapse, intrauterine infection (chorioamnionitis), premature delivery, limb contracture

Prolonged Pregnancy

Definition
- pregnancy >42 wk GA

Epidemiology
- 41 wk GA: up to 27%
- >42 wk GA: 5.5%

Etiology
- most cases idiopathic
- anencephalic fetus with no pituitary gland
- placental sulfatase deficiency (X-linked recessive condition in 1/2,000-1/6,000 infants) – rare

Clinical Features
- postmaturity syndrome (10-20% of post-term pregnancies): fetal weight loss, reduced subcutaneous fat, scaling, dry skin from placental insufficiency, long thin body, open-eyed, alert and worried look, long nails, palms and soles wrinkled)
- with increasing GA, higher rates of: intrauterine infection, asphyxia, meconium aspiration syndrome, placental insufficiency, placental aging and infarction, macrosomia, dystocia, fetal distress, operative deliveries, pneumonia, seizures, and requirement of NICU admission

Management
- GA 40-41 wk: expectant management
  - no evidence to support IOL or C/S unless other risk factors for morbidity are present (see prognosis)
- GA >41 wk: offer IOL if vaginal delivery is not contraindicated
  - IOL shown to decrease C/S, fetal heart rate changes, meconium staining, macrosomia, and death when compared with expectant management
- GA >41 wk and expectant management elected: serial fetal surveillance
  - fetal movement count by the mother
  - BPP q3-4d
  - if AFI is decreased, labour should be induced

Prognosis
- if >42 wk, perinatal mortality 2-3x higher (due to progressive uteroplacental insufficiency)
- morbidity increased with HTN in pregnancy, DM, abruption, IUGR, and multiple gestation

Intrauterine Fetal Death

Definition
- fetal death *in utero* after 20 wk GA

Epidemiology
- 1% of pregnancies

Etiology
- 50% idiopathic
- 50% secondary to HTN, DM, erythroblastosis fetalis, congenital anomalies, umbilical cord or placental complications, intrauterine infection, APS

Clinical Features
- decreased perception of fetal movement by mother
- SFH and maternal weight not increasing
- absent fetal heart tones on Doppler(not diagnostic)
- high MSAFP
- on U/S: no fetal heart rate. Depending on timing of death may see skull collapse, brain tissue retraction, empty fetal bladder, non-filled aorta, poor visualization of midline flax

Management
- diagnosis: absent cardiac activity and fetal movement on U/S required for diagnosis
- determine secondary cause
  - maternal: HbA1c, fasting glucose, TSH, Kleihauer-Betke, VDRL, ANA, CBC, anticardiolipins, antibody screens, INR/PTT, serum/urine toxicology screens, cervical and vaginal cultures, TORCH screen
  - fetal: karyotype, cord blood, skin biopsy, genetics evaluation, autopsy, amniotic fluid culture for CMV, parvovirus B19, herpes
  - placenta: pathology, bacterial cultures

DIC: Generalized Coagulation and Fibrinolysis Leading to Depletion of Coagulation Factors

Obstetrical Causes
- Abruption
- Gestational HTN
- Fetal demise
- PPH

DIC-specific Blood Work
- Platelets
- aPTT and PT
- FDP
- Fibrinogen

Treatment
- Treat underlying cause
- Supportive
  - Fluids
  - Blood products
    - FFP, platelets, cryoprecipitate
- Consider anti-coagulation as VTE prophylaxis

Treatment
- <12 wk: dilation and curettage
- 13-20 wk: dilation and evacuation or sometimes IOL
- induction of labour
- monitor for maternal coagulopathy (10% risk of DIC)
- parental psychological care as per hospital protocol
- comprehensive discussion within 3 mo about final investigation and post-mortem results, help make plans for future pregnancies

Intrauterine Growth Restriction

Definition
- infant weight <10th percentile for GA or <2,500 g

Etiology/Risk Factors
- 50% unknown
- maternal causes
  - malnutrition, smoking, drug abuse, alcoholism, cyanotic heart disease, type 1 DM, SLE, pulmonary insufficiency, previous IUGR (25% risk, most important risk factor), chronic HTN
- maternal-fetal
  - any disease causing placental insufficiency
  - includes gestational HTN, chronic renal insufficiency, gross placental morphological abnormalities (infarction, hemangiomas, placenta previa, placenta accreta, abnormal cord insertion), prolonged gestation
- fetal causes
  - TORCH infections, multiple gestation, congenital anomalies / chromosomal abnormalities (10%)

TORCH
Toxoplasmosis
Others: e.g. syphilis
Rubella
CMV
HSV
- See Table 12, OB19

Clinical Features
- symmetric/type I (25-30%): occurs early in pregnancy
  - reduced growth of both head and abdomen
  - head:abdomen ratio may be normal (>1 up to 32 wk; =1 at 32-34 wk; <1 after 34 wk GA)
  - usually associated with congenital anomalies or TORCH infections
- asymmetric/type II (70%): occurs late in pregnancy
  - fetal abdomen is disproportionately smaller than fetal head
  - brain is spared, therefore head:abdomen ratio increased
  - usually associated with placental insufficiency
  - more favourable prognosis than type I
- complications
  - prone to meconium aspiration, asphyxia, polycythemia, hypoglycemia, hypocalcemia, hyperphosphatemia hyponatremia, and mental retardation
  - greater risk of perinatal morbidity and mortality

Differential Diagnosis of Incorrect Uterine Size for Dates
- Inaccurate dates
- Maternal: DM
- Maternal-fetal: polyhydramnios, oligohydramnios
- Fetal: abnormal karyotype, IUGR, macrosomia, fetal anomaly, abnormal lie, multiple gestation

Investigations
- SFH measurements at every antepartum visit
- if mother at high risk or SFH lags >2 cm behind GA
  - U/S for biparietal diameter, head and abdominal circumference ratio, femur length, fetal weight, and AFV (decrease associated with IUGR)
  - ± BPP
  - Doppler analysis of umbilical cord blood flow

Monitoring Fetal Growth with U/S
Done biweekly to show growth beyond the margin of error

Management
- prevention via risk modification prior to pregnancy is ideal
- modify controllable factors: smoking, alcohol, nutrition, and treat maternal illness
- bed rest in LLDP
- serial BPP (monitor fetal growth) and determine cause of IUGR, if possible
- delivery when extrauterine existence is less dangerous than continued intrauterine existence (abnormal function tests, absent growth, severe oligohydramnios) especially if GA >34 wk
- liberal use of C/S since IUGR fetus withstands labour poorly

Macrosomia

Definition
- infant weight >90th percentile for a particular GA or >4,000 g

Etiology/Risk Factors
- maternal obesity, GDM, past history of macrosomic infant, prolonged gestation, multiparity

Clinical Features
- increased risk of perinatal mortality
- CPD and birth injuries (shoulder dystocia, fetal bone fracture) more common
- complications of DM in labour (see Table 15, OB28)

Investigations
- serial SFH
- further investigations if mother at high risk or SFH >2 cm ahead of GA
- U/S predictors
  - polyhydramnios
  - third trimester AC >1.5 cm/wk
  - HC/AC ratio <10th percentile
  - FL/AC ratio <20th percentile

Management
- prophylactic C/S is a reasonable option where EFW >5,000 g in non-diabetic woman and EFW >4,500 g in diabetic woman
  - no evidence that prophylactic C/S improves outcomes
- early induction of labour is not recommended for non-diabetic mothers
- risks and benefits of early induction (risk of C/S vs. risk of dystocia) must be weighed in diabetic mothers, as current research is unclear

Polyhydramnios/Oligohydramnios

Table 12. Polyhydramnios and Oligohydramnios

	Polyhydramnios	Oligohydramnios
Definition	AFI >25 cm U/S: single deepest pocket >8 cm	AFI <5 cm U/S: single deepest pocket ≤2 cm
Etiology	Idiopathic most common Maternal • Type 1 DM: abnormalities of transchorionic flow Maternal-fetal • Chorioangiomas • Multiple gestation • Fetal hydrops (increased erythroblastosis) Fetal • Chromosomal anomaly (up to 2/3 of fetuses have severe polyhydramnios) • Respiratory: cystic adenomatoid malformed lung • CNS: anencephaly, hydrocephalus, meningocele • GI: tracheoesophageal fistula, duodenal atresia, facial clefts (interfere with swallowing)	Idiopathic most common Maternal • Uteroplacental insufficiency (preeclampsia, nephropathy) • Medications (ACEI) Fetal • Congenital urinary tract anomalies (renal agenesis, obstruction, posterior urethral valves) • Demise/chronic hypoxemia (blood shunt away from kidneys to perfuse brain) • IUGR • Ruptured membranes: prolonged amniotic fluid leak • Amniotic fluid normally decreases after 35 wk
Epidemiology	Occur in 0.2-1.6% of all pregnancies	Occur in ~4.5% of all pregnancies Severe form in <0.7% Common in pregnancies >41 wk (~12%)
Clinical Features and Complications	Uterus large for dates, difficulty palpating fetal parts and hearing FHR Maternal complications • Pressure symptoms from overdistended uterus (dyspnea, edema, hydronephrosis) Obstetrical complications • Cord prolapse, placental abruption, malpresentation, preterm labour, uterine dysfunction, and PPH	Uterus small for dates Fetal complications • 15-25% have fetal anomalies • Amniotic fluid bands (T1) can lead to Potter's facies, limb deformities, abdominal wall defects Obstetrical complications • Cord compression • Increased risk of adverse fetal outcomes • Pulmonary hypoplasia (late-onset) • Marker for infants who may not tolerate labour well
Management	Determine underlying cause: • Screen for maternal disease/infection • Complete fetal U/S evaluation Depends on severity • Mild to moderate cases require no treatment • If severe, hospitalize and consider therapeutic amniocentesis	Always warrants admission and investigation: • Rule out ROM • Fetal monitoring (NST, BPP) • U/S Doppler studies (umbilical cord and uterine artery) Maternal hydration with oral or IV fluids to help increase amniotic fluid Vesicoamniotic shunt: if etiology is related to fetal obstuctive uropathy; however, pulmonary function may not be restored with restoration of amniotic fluid Injection of fluid via amniocentesis will improve condition for ~1 wk – may be most helpful for visualizing any associated fetal anomalies Consider delivery if term Amnio-infusion may be considered during labour via intrauterine catheter
Prognosis	2-5 fold increase in risk of perinatal mortality	Poorer with early onset High mortality related to congenital malformations and pulmonary hypoplasia when diagnosed during T2

Multi-Fetal Gestation and Malpresentation

Epidemiology

- incidence of twins is 1/80 and triplets 1/6,400 in North America
- 2/3 of twins are dizygotic (fraternal)
  - risk factors for dizygotic twins: IVF, increased maternal age, newly discontinued OCP, ethnicity (e.g. certain African regions)
- monozygous twinning occurs at a constant rate worldwide (1/250)
- determine zygosity by number of placentas, thickness of membranes, sex, blood type

Clinical Features

Table 13. Complications Associated with Multiple Gestation

Maternal	Uteroplacental	Fetal
Hyperemesis gravidarum	Increased PROM/PTL	Prematurity*
GDM	Polyhydramnios	IUGR
Gestational HTN	Placenta previa	Malpresentation
Anemia	Placental abruption	Congenital anomalies
Increased physiological stress on all systems	PPH (uterine atony)	Twin-twin transfusion
	Umbilical cord prolapse	Increased perinatal morbidity and mortality
Increased compressive symptoms	Cord anomalies	Twin interlocking
C/S	(velamentous insertion, 2 vessel cord)	(twin A breech, twin B vertex)
		Single fetal demise

*Most common cause of perinatal mortality in multiple gestation

The Ps of Multiple Gestation Complications

Increased rates of
Puking
Pallor (anemia)
Preeclampsia/**P**IH
Pressure (compressive symptoms)
PTL/**P**ROM/**PPROM**
Polyhydramnios
Placenta previa/abruptio
PPH/APH
Prolonged labour
Cord **P**rolapse
Prematurity
Mal**p**resentation
Perinatal morbidity and mortality
Parental distress
Postpartum depression

Management

- U/S determination of chorionicity must be done within first trimester (ideally 8-12 wk GA)
- increased antenatal surveillance
  - serial U/S q 2-3wk from 24 wk GA to assess growth (uncomplicated diamniotic dichorionitic)
  - increased frequency of ultrasounds in monochorionic diamniotic and monochorionic monoamniotic twins
  - Doppler flow studies weekly if discordant fetal growth (>30%)
  - BPP as needed
- may attempt vaginal delivery if twin A presents as vertex, otherwise C/S (40-50% of all twin deliveries, 15% of cases have twin A delivered vaginally and twin B delivered by C/S)
- mode of delivery depends on fetal weight, GA, presentation

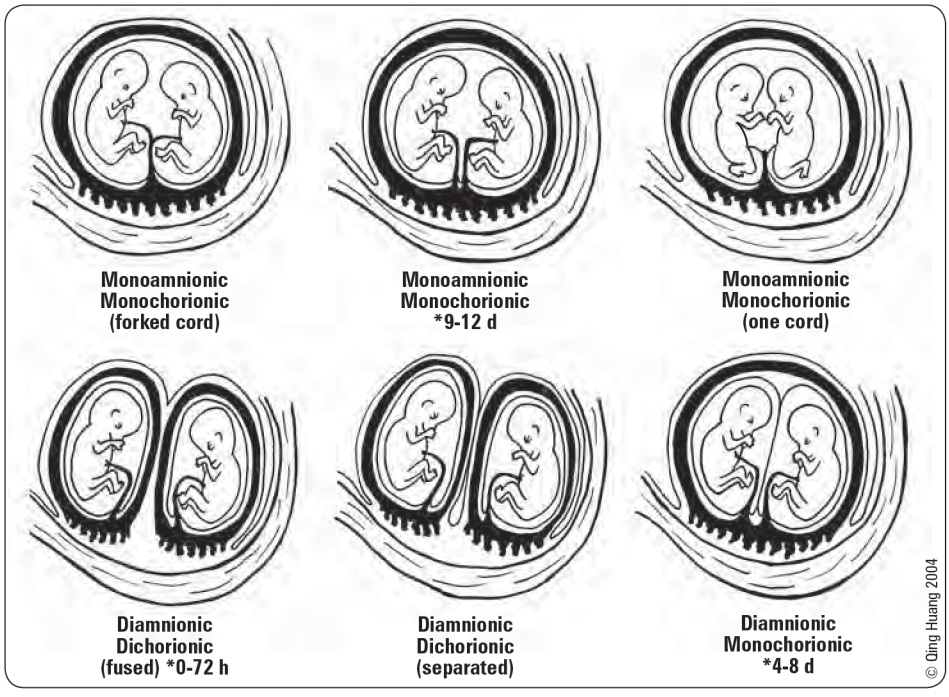


Figure 4. Classification of twin pregnancies *Indicates time of cleavage

Twin-Twin Transfusion Syndrome

Epidemiology
- 10% of monochorionic twins
- concern if >30% discordance in estimated fetal weight

Etiology
- arterial blood from donor twin passes through placenta into vein of recipient twin

Clinical Features
- donor twin: IUGR, hypovolemia, hypotension, anemia, oligohydramnios
- recipient twin: hypervolemia, HTN, CHF, polycythemia, edema, polyhydramnios, kernicterus in neonatal period

Investigations
- detected by U/S screening, Doppler flow analysis

Management
- therapeutic serial amniocentesis to decompress polyhydramnios of recipient twin and decrease pressure in cavity and on placenta
- intrauterine blood transfusion to donor twin if necessary
- laparoscopic occlusion of placental vessels

Breech Presentation

Definition
- fetal buttocks or lower extremity is the presenting part as determined on U/S
- complete (10%): hips and knees both flexed
- frank (60%): hips flexed, knees extended, buttocks present at cervix
  - most common type of breech presentation
  - most common breech presentation to be delivered vaginally
- incomplete (30%): both or one hip flexed and both or one knee present below the buttocks, feet or knees present first (footling breech, kneeling breech)

Epidemiology
- occurs in 3-4% of pregnancies at term (25% <28 wk)

Risk Factors
- maternal: pelvis (contracted), uterus (shape abnormalities, intrauterine tumours, fibroids, previous breech), pelvic tumours causing compression, grand multiparity
- maternal-fetal: placenta (previa), amniotic fluid (poly-/oligohydramnios)
- fetal: prematurity, multiple gestation, congenital malformations (found in 6% of breeches; 2-3% if in vertex presentations), abnormalities in fetal tone and movement, aneuploidy, hydrocephalus, anencephalus

Management
- ECV: repositioning of singleton fetus within uterus under U/S guidance
  - overall success rate of 65%
  - criteria: >36 wk GA, singleton, unengaged presenting part, reactive NST, not in labour
  - contraindications: previous T3 bleed, prior classical C/S, previous myomectomy, oligohydramnios, PROM, placenta previa, abnormal U/S, suspected IUGR, HTN, uteroplacental insufficiency, nuchal cord
  - risks: abruption, cord compression, cord accident, ROM, labour, fetal bradycardia requiring C/S (<1% risk), alloummunization, fetal death 1:5,000
  - method: tocometry, followed by U/S guided transabdominal manipulation of fetus with constant fetal heart monitoring
  - if patient Rh negative, give Rhogam® prior to procedure
  - good prognostic factors (for a successful version)
    - multiparous, good fluid volume, small baby, skilled obstetrician, posterior placenta
- pre- or early labour ultrasound to assess type of breech presentation, fetal growth, estimated weight, placenta position, attitude of fetal head (flexed is preferable); if ultrasound unavailable, recommend C/S
- ECV and elective C/S should be presented as options with the risks and benefits outlined; obtain informed consent
- method for vaginal breech delivery
  - encourage effective maternal pushing efforts
  - at delivery of after-coming head, assistant must apply suprapubic pressure to flex and engage fetal head
  - delivery can be spontaneous or assisted; avoid fetal traction
  - apply fetal manipulation only after spontaneous delivery to level of umbilicus

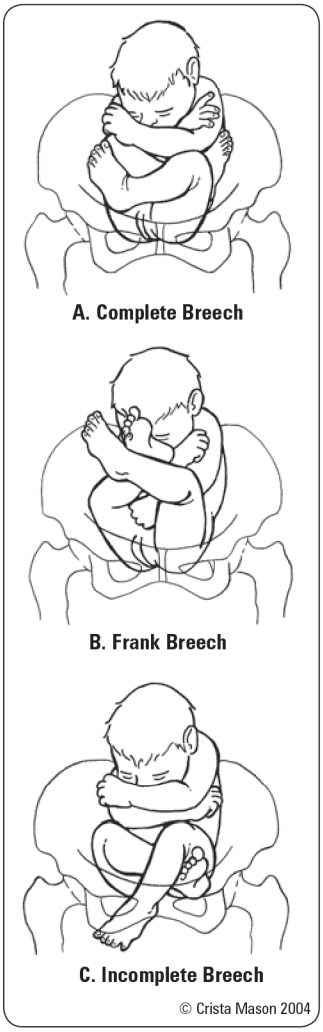


Figure 5. Types of breech presentation

Criteria for Vaginal Breech Delivery
- Frank or complete breech, GA >36 wk
- EFW 2,500-3,800 g based on clinical and U/S assessment (5.5–8.5 lb)
- Fetal head flexed
- Continuous fetal monitoring
- 2 experienced obstetricians, assistant, and anesthetist present
- Ability to perform emergency C/S within 30 min if required

- C/S recommended if: the breech has not descended to the perineum in the second stage of labour after 2 h, in the absence of active pushing, or if vaginal delivery is not imminent after 1 h of active pushing
- contraindications to vaginal breech delivery:
  - cord presentation
  - clinically inadequate maternal pelvis
  - fetal factors incompatible with vaginal delivery (e.g. hydrocephalus), macrosomia, fetal growth restriction

Prognosis
- regardless of route of delivery, breech infants have lower birth weights and higher rates of perinatal mortality, congenital anomalies, abruption, and cord prolapse

Vaginal Delivery of Breech Presentation
SOGC Clinical Practice Guidelines 2009;226;557-566
Objective: To discuss risks and benefits of trial of labour versus planned C/S, with selection criteria, management, and delivery techniques for trial of vaginal breech birth.
Evidence: Randomized trials, prospective cohort studies and select cohort studies from Medline search for long-term outcomes and epidemiology of vaginal breech delivery.
Summary: Higher risk of perinatal mortality and short-term neonatal morbidity can be associated with vaginal breech birth as compared to elective C/S. However, careful case selection (including term singleton breech fetuses and clinically adequate maternal pelvis) and labour management may achieve a similar safety level as elective C/S (~2 per 1,000 births perinatal mortality, ~2% short-term neonatal morbidity). Specific protocols for vaginal breech delivery should be followed: continuous fetal heart monitoring, assessment for adequate progress in labour, no induction of labour recommended, emergency C/S available, if required, and health care providers with requisite skills and experience. Informed consent for the preferred delivery method should be obtained.

Hypertensive Disorders of Pregnancy

Hypertension in Pregnancy

- hypertensive disorders of pregnancy are classified as either **pre-existing** or **gestational HTN**

PRE-EXISTING HYPERTENSION

Definition
- BP >140/90 prior to 20 wk GA, persisting >7 wk postpartum
- essential HTN is associated with an increased risk of gestational HTN, abruptio placentae, IUGR, and IUFD

GESTATIONAL HTN

Definition
- sBP >140 or dBP >90 developing after 20th wk GA in a woman known to be normotensive before pregnancy

Risk Factors
- maternal factors
  - primigravida (80-90% of gestational HTN)
  - first conception with a new partner
  - PMHx or FHx of gestational HTN
  - DM, chronic HTN, or renal insufficiency
  - antiphospholipid syndrome
  - extremes of maternal age (<18 or >35 yr)
  - previous stillbirth or IUFD
- fetal factors
  - IUGR or oligohydramnios
  - GTN
  - multiple gestation
  - fetal hydrops

Ominous Symptoms of HTN in Pregnancy
RUQ pain, headache, and visual disturbances

Clinical Evaluation of HTN in Pregnancy
- in general, clinical evaluation should include the mother and fetus
- **evaluation of mother**
  - body weight
  - central nervous system
    - presence and severity of headache
    - visual disturbances – blurring, scotomata
    - tremulousness, irritability, somnolence
    - hyperreflexia
  - hematologic
    - bleeding, petechiae
  - hepatic
    - RUQ or epigastric pain
    - severe N/V
  - renal
    - urine output and colour
  - non-dependent edema (i.e. hands and face)
- **evaluation of fetus**
  - fetal movement
  - fetal heart rate tracing – NST
  - ultrasound for growth
  - BPP
  - Doppler flow studies

Hypertension in Pregnancy

Adverse Maternal Conditions
- sBP >160 mmHg
- dBP >100 mmHg
- HELLP
- Cerebral hemorrhage
- Renal dysfunction: oliguria <500 mL/d
- Left ventricular failure, pulmonary edema
- Placental abruption, DIC
- **Symptoms**
  - Abdominal pain, N/V
  - Headaches, visual problems
  - SOB, chest pain
  - Eclampsia: convulsions

Adverse Fetal Conditions
- IUGR
- Oligohydramnios
- Absent/reversed umbilical artery end diastolic flow
- **Can result in:**
  - Fetal disability and/or death

Laboratory Evaluation of Gestational Hypertension
- CBC
- PTT, INR, fibrinogen – especially if surgery or regional anaesthetics are planned
- ALT, AST
- creatinine, uric acid
- 24 h urine collection for protein or albumin:creatinine ratio

Complications
- maternal
  - liver and renal dysfunction
  - seizure
  - abruptio placentae
  - left ventricular failure/pulmonary edema
  - DIC (release of placental thromboplastin consumptive coagulopathy)
  - HELLP syndrome
    - treat with FFP infusion or plasma exchange
  - hemorrhagic stroke (50% of deaths)
- fetal (2º to placental insufficiency)
  - IUGR, prematurity, abruptio placentae, IUFD

Management
- for non-severe HTN (149-159/90 to 105) target a BP of 130-155/80-105 in women without comorbidities or <140/90 in women with comorbidities
- for both pre-existing and gestational HTN, labetalol 100-400 mg PO bid-tid, nifedipine XL preparation 20-60 mg PO od, or α-methyldopa 250-500 mg PO bid-qid
- for severe HTN (BP>160/110), give one of:
  - labetalol 20 mg IV then 20-80 mg IV q30min (max 300 mg)(then switch to oral)
  - nifedipine 5-10 mg capsule q30min
    - hydralazine 5 mg IV, repeat 5-10 mg IV q30min or 0.5 to 10 mg/hr IV, to a maximum of 20 mg IV (or 30 mg IM)
- no ACEI, ARBs, diuretics, prazosin, or atenolol
- pre-existing HTN and gestational HTN without any deterioration can be followed until 37 wk then decide to induce shortly thereafter

I-A Evidence-Recommendation Highlights of SOGC Clinical Practice Guidelines
Diagnosis, Evaluation, and Management of the Hypertensive Disorders of Pregnancy
J Obstet Gynaecol Can 2014;36(5):416-438
- For BP measurement, Korotkoff phase V should be used to designate the dBP.
- Calcium supplementation (of at least 1g/d, orally) is recommended for women with low dietary intake of calcium (<600 mg/d). (I-A)
- For preeclampsia prevention among increased risk women, low-dose Aspirin® (75-100 mg/d) is recommended until delivery.
- Umbilical artery Doppler velocimetry should be part of the antenatal fetal surveillance in preeclampsia.
- Initial antihypertensive therapy for severe HTN (sBP >160 or dBP ≥110) should be with labetalol, nifedipine, or hydralazine.
- Initial antihypertensive therapy for non-severe HTN (BP 140-159/90-109 mmHg) should be with methyldopa, β-blockers, or calcium channel blockers.
- Antenatal corticosteroids for fetal lung maturation should be considered for all women with preeclampsia before 34 wk gestation.
- In a planned vaginal delivery with an unfavourable cervix, cervical ripening should be used.
- Oxytocin 5 units IV or 10 units IM should be used as part of the management during the third stage of labour, particularly in the presence of thrombocytopenia or coagulopathy.
- MgSO4 is the recommended first-line treatment for eclampsia.
- MgSO4 is the recommended eclampsia prophylaxis in severe preeclampsia.

PREECLAMPSIA

Definition
- pre-existing or gestational HTN with new onset proteinuria or adverse conditions

Risk Factors
- nulliparity
- preeclampsia in a previous pregnancy
- age >40 yr or <18 yr
- FHx of preeclampsia
- chronic HTN
- chronic renal disease
- antiphospholipid antibody syndrome or inherited thrombophilia
- vascular or connective tissue disease
- DM (pre-gestational and gestational)
- high BMI
- hydrops fetalis
- unexplained fetal growth restriction
- abruptio placentae
- there is a potential for further deterioration to severe preeclampsia as defined above
- the adverse conditions are many and include both maternal and fetal issues

Preeclampsia Investigations
- CBC
- AST, ALT
- INR and aPTT (if regional anesthesia planned)
- Cr
- Urine (24 h protein collection or albumin/creatinine ratio)
- Uric acid

Management
- depends on GA, possible threat of seizures (check reflexes)
- if stable and no adverse factors, may admit and follow, ± decide to deliver as approaching 34-36 wk (must weigh risks of fetal prematurity vs. risks of developing severe preeclampsia/eclampsia)
- for severe preeclampsia, stabilize and deliver
- if severe preeclampsia during labour, increase maternal monitoring: hourly input and output, urine dip q12h, hourly neurological vitals, and increase fetal monitoring (continuous FHR monitoring)
- antihypertensive therapy
  - labetalol 20 mg IV then 20-80 mg IV q30min (max 300 mg)(then switch to oral)
  - nifedipine 5-10 mg capsule q30min
    - hydralazine 5 mg IV, repeat 5-10 mg IV q30min or 0.5 to 10 mg/hr IV, to a maximum of 20 mg IV (or 30 mg IM)

- seizure prevention
  - $MgSO_4$
  - postpartum management
  - risk of seizure highest in first 24 h postpartum – continue $MgSO_4$ for 12-24 h after delivery
  - vitals q1h
  - consider HELLP syndrome in toxic patients
  - most return to a normotensive BP within 2 wk

HELLP Syndrome
Hemolysis
Elevated **L**iver enzymes
Low **P**latelets

ECLAMPSIA

Definition
- the occurrence of one or more generalized convulsions and/or coma in the setting of preeclampsia and in the absence of other neurologic conditions

Epidemiology
- an eclamptic seizure occurs in approximately 0.5% of mildly preeclamptic women and 2-3% of severely preeclamptic women

Note
Eclampsia prior to 20 wk of gestation is rare and should raise the possibility of an underlying molar pregnancy or antiphospholipid syndrome

Risk Factors
- same as risk factors for preeclampsia

Clinical Manifestations
- eclampsia is a clinical diagnosis
- typically tonic-clonic and lasting 60-75 s
- one of the signs of an impending seizure is hyperreflexia
- symptoms that may occur before the seizure include persistent frontal or occipital headache, blurred vision, photophobia, right upper quadrant or epigastric pain, and altered mental status
- in up to one third of cases, there is no proteinuria or blood pressure <140/90 mmHg prior to the seizure
- in general, women with typical eclamptic seizures who do not have focal neurologic deficits or prolonged coma do not require diagnostic evaluation including imaging

Differential Diagnosis of Cause for Seizure in a Pregnant Woman
- Stroke
- Hypertensive disease (hypertensive encephalopathy, pheochromocytoma)
- Space-occupying lesion of the CNS
- Metabolic disorders (hypoglycemia, SIADH)
- Infection (meningitis, encephalitis)
- Thrombotic thrombocytopenic purpura or thrombophilia
- Idiopathic epilepsy
- Use of illicit drugs
- Cerebral vasculitis

Management
- ABCs
- roll patient into LLDP
- supplemental O_2 via face mask to treat hypoxemia due to hypoventilation during convulsive episode
- aggressive antihypertensive therapy for sustained diastolic pressures ≥105 mmHg or systolic blood pressures ≥160 mmHg with hydralazine or labetalol
- prevention of recurrent convulsions: to prevent further seizures and the possible complications of repeated seizure activity (e.g. rhabdomyolysis, metabolic acidosis, aspiration pneumonitis, etc.)
- $MgSO_4$ is now the drug of choice, with previously used agents including diazepam and phenytoin
- the definitive treatment of eclampsia is DELIVERY, irrespective of gestational age, to reduce the risk of maternal morbidity and mortality from complications of the disease
- mode of delivery is dependent on clinical situation and fetal-maternal condition

$MgSO_4$ Toxicity
- Flushing
- Hyporeflexia
- Somnolence
- Respiratory and cardiac depression
- Weakness

Note: Increased risk of toxicity with concurrent calcium channel blocker use or renal disease

Treatment
- Stop $MgSO_4$
- Calcium gluconate 10% in 10 mL IV

Medical Complications of Pregnancy

Iron and Folate Deficiency Anemia

Table 14. Iron Deficiency and Folate Deficiency Anemia

	Iron Deficiency Anemia	Folate Deficiency Anemia
Etiology	See Hematology, H15	See Hematology, H25
Epidemiology	Responsible for 80% of causes of non-physiologic anemia during pregnancy	Incidence varies from 0.5-25% depending on region, population, diet
Clinical Features	See Hematology, H15	See Hematology, H25
Investigations	See Hematology, H15	See Hematology, H25

Table 14. Iron Deficiency and Folate Deficiency Anemia (continued)

	Iron Deficiency Anemia	Folate Deficiency Anemia
Management	Prevention (non-anemic): 30 mg elemental iron/d (met by most prenatal vitamins) Treatment (anemic): 30-120 mg elemental iron/d 325 mg ferrous fumarate = 106 mg elemental Fe^{2+}; 325 mg ferrous sulfate = 65 mg elemental Fe^{2+}; 325 mg ferrous gluconate = 36 mg elemental Fe^{2+} Polysaccharide-Iron Complex = 150 mg elemental Fe/capsule	Prevention: 0.4-1 mg folic acid PO daily for 1-3 mo preconceptually and throughout T1, or 5 mg folic acid per day with past history of oNTD, DM, or antiepileptic medication use
Complications	Maternal: angina, CHF, infection, slower recuperation, preterm labour Fetal: decreased oxygen carrying capacity leading to fetal distress, IUGR, and low birth weight	Maternal: decreased blood volume, N/V, anorexia Fetal: neural tube defects in T1, low birth weight, prematurity
Notes	Mother needs 1 g of elemental iron per fetus; this amount exceeds normal stores + dietary intake Iron requirements increase during pregnancy due to fetal/placental growth (500 mg), increased maternal RBC mass (500 mg), and losses (200 mg) – more needed for multiple gestations	Minimum daily requirement is 0.4 mg Most often associated with iron deficiency anemia Folic acid is necessary for closure of neural tube during early fetal development (by day 28 of gestation)

Diabetes Mellitus

Epidemiology
- 2-4% of pregnancies are complicated by DM

Classification of Diabetes Mellitus
- type 1 and type 2 DM (see Endocrinology, E7)
- GDM: onset of DM during pregnancy (usually around 24-28 wk GA)

Etiology
- type 1 and type 2 DM
- GDM: anti-insulin factors produced by placenta and high maternal cortisol levels create increased peripheral insulin resistance → leading to GDM and/or exacerbating pre-existing DM

MANAGEMENT

A. TYPE 1 and TYPE 2 DM

Preconception
- pre-plan and refer to high-risk clinic
- optimize glycemic control
- counsel patient on potential risks and complications
- evaluate for diabetic retinopathy, neuropathy, CAD

Pregnancy
- if already on oral medication, generally switch to insulin therapy
  - continuing glyburide or metformin controversial
  - teratogenicity unknown for other oral anti-hyperglycemics
- tight glycemic control
  - insulin dosage may need to be adjusted in T2 due to increased demand and increased insulin resistance
- monitor as for normal pregnancy plus initial 24 h urine protein and creatinine clearance, retinal exam, HbA1c
  - HbA1c: >140% of pre-pregnancy value associated with increased risk of spontaneous abortion and congenital malformations
- increased fetal surveillance (BPP, NST), consider fetal ECHO to look for cardiac abnormalities

Monitoring Glucose Levels
- Frequent measurements of blood glucose during pregnancy are advised for women with type 1 or 2 DM to help prevent or treat both hypoglycemia and hyperglycemia, and also improves neonatal outcome
- Aim for FPG ≤5.3 mmol/L (95 mg/dL), 1 h post prandial
- PG ≤7.8 mmol/L (140 mg/dL), 2 h post prandial PG ≤6.7 mmol/L (120 mg/dL)
- Most women can be followed with monthly HbA1c determinations

Labour
- timing of delivery depends on fetal and maternal health and risk factors (i.e. must consider size of baby, lung maturity, maternal blood glucose, and blood pressure control)
- can wait for spontaneous labour if blood glucose well-controlled and BPP normal
- induce by 38 wk

- type of delivery
  - increased risk of cephalopelvic disproportion (CPD) and shoulder dystocia with babies >4,000 g (8.8 lbs)
  - elective C/S for predicted birthweight >4,500 g (9.9 lbs) (controversial)
- monitoring
  - during labour monitor blood glucose q1h with patient on insulin and dextrose drip
  - aim for blood glucose between 3.5-6.5 mmol/L to reduce the risk of neonatal hypoglycemia

Postpartum

- insulin requirements dramatically drop with expulsion of placenta (source of insulin antagonists)
- no insulin is required for 48-72 h postpartum in most type 1 DM
- monitor glucose q6h, restart insulin at two-thirds of pre-pregnancy dosage when glucose >8 mmol/L

Post-prandial blood glucose values seem to be the most effective at determining the likelihood of macrosomia or other adverse pregnancy outcomes

B. GESTATIONAL DM

Screening and Diagnosis

- all pregnant women between 24-28 wk GA (or at any stage if high risk)
- 2 screening options
  - 1-step screening with fasting 75 g OGTT; GDM if ≥1 of:
    - FPG ≥ 5.1 mmol/L
    - 1h PG ≥ 10.0 mmol/L
    - 2h PG ≥ 8.5 mmol/L
  - 2-step screening
    - Step 1: Perform a random nonfasting 50 g OGCT
      - 1h PG < 7.8 mmol/L is normal
      - 1h PG ≥ 11.1 mmol/L is GDM
      - if 1h PG 7.8-11.0 mmol/L, proceed to Step 2
    - Step 2: Perform a fasting 75 g OGTT, GDM if ≥1 of:
      - FPG ≥ 5.3 mmol/L
      - 1h PG ≥ 10.6 mmol/L
      - 2h PG ≥ 9.0 mmol/L

Risk Factors for GDM

- Age >25 yr
- Obesity
- Ethnicity (Aboriginal, Hispanic, Asian, African)
- FHx of DM
- Previous history of GDM
- Previous child with birthweight >4.0 kg
- Polycystic ovarian syndrome
- Current use of glucocorticoids
- Essential HTN or pregnancy-related HTN

Management

- first line is management through diet modification and increased physical activity
- initiate insulin therapy if glycemic targets not achieved within 2 wk of lifestyle modification alone
- glycemic targets: FPG <5.3 mmol/L, 1h PG <7.8 mmol/L, 2h PG <6.7 mmol/L
- use of oral agents can be used in pregnancy but is off-label and should be discussed with patient
- stop insulin and diabetic diet postpartum
- 6 wk postpartum visit: follow up with 75 g OGTT, counsel about lifestyle modifications, and perform glucose challenge test q2 yr

Prognosis

- most maternal and fetal complications are related to hyperglycemia and its effects

Long-Term Maternal Complications

- type 1 and type 2 DM: risk of progressive retinopathy and nephropathy
- GDM: 50% risk of developing type 2 DM in next 20 yr

Table 15. Complications of DM in Pregnancy

Maternal	Fetal
Obstetric • HTN/preeclampsia (especially if pre-existing nephropathy/proteinuria): insulin resistance is implicated in etiology of HTN • Polyhydramnios: maternal hyperglycemia leads to fetal hyperglycemia, which leads to fetal polyuria (a major source of amniotic fluid)	**Growth Abnormalities** • Macrosomia: maternal hyperglycemia leads to fetal hyperinsulinism resulting in accelerated anabolism • IUGR: due to placental vascular insufficiency
Diabetic Emergencies • Hypoglycemia • Ketoacidosis • Diabetic coma	**Delayed Organ Maturity** • Fetal lung immaturity: hyperglycemia interferes with surfactant synthesis (respiratory distress syndrome)
End-Organ Involvement or Deterioration (occur in type 1 DM and type 2 DM, not in GDM) • Retinopathy • Nephropathy	**Congenital Anomalies (occur in type 1 DM and type 2 DM, not in GDM)** • 2-7x increased risk of cardiac (VSD), NTD, GU (cystic kidneys), GI (anal atresia), and MSK (sacral agenesis) anomalies due to hyperglycemia • Note: Pregnancies complicated by GDM do not manifest an increased risk of congenital anomalies because GDM develops after the critical period of organogenesis (in T1)

Table 15. Complications of DM in Pregnancy (continued)

Maternal	Fetal
Other • Pyelonephritis/UTI: glucosuria provides a culture medium for *E. coli* and other bacteria • Increased incidence of spontaneous abortion (in type 1 DM and type 2 DM, not in GDM): related to pre-conception glycemic control	**Labour and Delivery** • Preterm labour/prematurity: most commonly in patients with HTN/preeclampsia • Preterm labour is associated with poor glycemic control but the exact mechanism is unknown • Increased incidence of stillbirth • Birth trauma: due to macrosomia, can lead to difficult vaginal delivery and shoulder dystocia **Neonatal** • Hypoglycemia: due to pancreatic hyperplasia and excess insulin secretion in the neonate • Hyperbilirubinemia and jaundice: due to prematurity and polycythemia • Hypocalcemia: exact pathophysiology not understood, may be related to functional hypoparathyroidism • Polycythemia: hyperglycemia stimulates fetal erythropoietin production

Group B Streptococcus

Epidemiology
- 15-40% vaginal carrier rate

Risk Factors (for neonatal disease)
- GBS bacteriuria during current pregnancy even if treated
- previous infant with invasive GBS infection
- preterm labour <37 wk
- ruptured membranes >18 h before delivery
- intrapartum maternal temperature ≥38°C
- positive GBS screen during current pregnancy

Clinical Features
- not harmful to mother
- risk of vertical transmission (neonatal sepsis, meningitis or pneumonia, and death)

Investigations
- offer screening to all women at 35-37 wk with vaginal and anorectal swabs for GBS culture

Treatment
- treatment of maternal GBS at delivery decreases neonatal morbidity and mortality
- indications for antibiotic prophylaxis: positive GBS screen, GBS in urine, or previous infant with GBS disease or GBS status unknown and one of the other risk factors
- antibiotics for GBS prophylaxis
  - penicillin G 5 million units IV then 2.5 million units IV q4h until delivery
  - penicillin allergic but not at risk for anaphylaxis: cefazolin 2 g IV then 1 g q8h
  - penicillin allergic and at risk for anaphylaxis: vancomycin 1 g IV q12h until delivery
- if fever, broad spectrum antibiotic coverage is advised

Indications for Intrapartum Antibiotic GBS Prophylaxis
Centres for Disease Control and Prevention. Prevention of Perinatal Group B Streptococcal Disease. *MMWR* 2010;59(RR-10):14
- Previous infant with invasive GBS disease.
- GBS bacteriuria during any trimester of the current pregnancy.
- Positive GBS vaginal-rectal screening culture in late gestation during current pregnancy.
- Unknown GBS status at the onset of labour (culture not done, incomplete, or results unknown) and any of the following:
  - Delivery at <37 wk gestation.
  - Amniotic membrane rupture ≥18 h.
  - Intrapartum temperature ≥100.4°F (≥38.0°C).
  - Intrapartum nucleic-acid amplification test positive for GBS.

Urinary Tract Infection

Treat asymptomatic bacteriuria in pregnancy because of increased risk of progression to cystitis, pyelonephritis, and probable increased risk of preterm labour

Epidemiology
- most common medical complication of pregnancy
- asymptomatic bacteriuria in 2-7% of pregnant women, more frequently in multiparous women
- note: asymptomatic bacteriuria should be treated in pregnancy due to increased risk of pyelonephritis and preterm labour

Etiology
- increased urinary stasis from mechanical and hormonal (progesterone) factors
- organisms include GBS as well as those that occur in non-pregnant women

Clinical Features
- may be asymptomatic
- dysuria, urgency, and frequency in cystitis
- fever, flank pain, and costovertebral angle tenderness in pyelonephritis

Investigations
- urinalysis, urine C&S
- cystoscopy and renal function tests in recurrent infections

Management
- uncomplicated UTI
  - first line: amoxicillin (250-500 mg PO q8h x 7 d)
  - alternatives: nitrofurantoin (100 mg PO bid x 7 d)
  - follow with monthly urine cultures
- pyelonephritis
  - hospitalization and IV antibiotics

Prognosis
- complications if untreated: acute cystitis, pyelonephritis, and possible preterm labour
- recurrence is common

Infections During Pregnancy

Table 16. Infections During Pregnancy

Infection	Agent	Source of Transmission	Greatest Transmission Risk to Fetus	Effects on Fetus	Effects on Mother	Diagnosis	Management
Chicken Pox	Varicella zoster virus (herpes family)	To mom: direct, respiratory To baby: transplacental	13-30 wk GA, and 5 d pre- to 2 d post-delivery	Congenital varicella syndrome (limb aplasia, chorioretinitis, cataracts, cutaneous scars, cortical atrophy, IUGR, hydrops), preterm labour	Fever, malaise, vesicular pruritic lesions	Clinical, ± vesicle fluid culture, ± serology	VZIG for mother if exposed, decreases congenital varicella syndrome Note: do not administer vaccine during pregnancy (live attenuated vaccine)
***CMV**	DNA virus (herpes family)	To mom: blood/organ transfusion, sexual contact To baby: transplacental, during delivery, breast milk	T1-T3	5-10% develop CNS involvement (mental retardation, cerebral calcification, hydrocephalus, microcephaly, deafness, chorioretinitis)	Asymptomatic or flu-like	Serologic screen; isolate virus from urine or secretion culture	No specific treatment; maintain good hygiene and avoid high risk situations
Erythema Infectiosum (Fifth Disease)	Parvovirus B19	To mom: respiratory, infected blood products To baby: transplacental	10-20 wk GA	Spontaneous abortion (SA), stillbirth, hydrops *in utero*	Flu-like, rash, arthritis; often asymptomatic	Serology, viral PCR, maternal AFP; if IgM present, follow fetus with U/S for hydrops	If hydrops occurs, consider fetal transfusion
Hepatitis B	DNA virus	To mom: blood, saliva, semen, vaginal secretions To baby: transplacental, breast milk	T3 10% vertical transmission if asymptomatic and HBsAg +ve; 85-90% if HBsAg and HBeAg +ve	Prematurity, low birth weight, neonatal death	Fever, N/V, fatigue, jaundice, elevated liver enzymes	Serologic screening for all pregnancies	Rx neonate with HBIG and vaccine (at birth, 1, 6 mo); 90% effective
***Herpes Simplex Virus**	DNA virus	To mom: intimate mucocutaneous contact To baby: transplacental, during delivery	Delivery (if genital lesions present); less commonly *in utero*	Disseminated herpes (20%); CNS sequelae (35%); self-limited infection	Painful vesicular lesions	Clinical diagnosis	Acyclovir for symptomatic women, suppressive therapy at 36 wk controversial Suggested C/S if active genital lesions, even if remote from vulva
HIV	RNA retrovirus	To mom: blood, semen, vaginal secretions To baby: *in utero*, during delivery, breast milk	1/3 *in utero*, 1/3 at delivery, 1/3 breastfeeding	IUGR, preterm labour, PROM	See Infectious Diseases, ID28	Serology, viral PCR All pregnant women are offered screening	Triple anti-retroviral therapy decreases transmission to <1% Elective C/S: no previous antiviral Rx or monotherapy only, viral load unknown or >500 RNA copies/mL, unknown prenatal care, patient request

Table 16. Infections During Pregnancy (continued)

Infection	Agent	Source of Transmission	Greatest Transmission Risk to Fetus	Effects on Fetus	Effects on Mother	Diagnosis	Management
***Rubella**	ssRNA togavirus	To mom: respiratory droplets (highly contagious) To baby: transplacental	T1	SA or congenital rubella syndrome (hearing loss, cataracts, CV lesions, MR, IUGR, hepatitis, CNS defects, osseous changes)	Rash (50%), fever, posterior auricular or occipital lymphadenopathy, arthralgia	Serologic testing; all pregnant women screened (immune if titre >1:16); infection if IgM present or >4x increase in IgG	No specific treatment; offer vaccine following pregnancy Do not administer during pregnancy (live attenuated)
Syphilis	Spirochete (*Treponema pallidum*)	To mom: sexual contact To baby: transplacental	T1-T3	Risk of preterm labour, multisystem involvement, fetal death	See Infectious Diseases, ID25	VDRL screening for all pregnancies; if positive, requires confirmatory testing	Pen G 2.4 million U IM x 1 dose if early syphilis, 3 doses if late syphilis, monitor VDRL monthly If Pen G allergic: Clindamycin 900 mg IV q8h
***Toxoplasmosis**	Protozoa (*Toxoplasma gondii*)	To mom: raw meat, unpasteurized goat's milk, cat feces/urine To baby: transplacental	T3 (but most severe if infected in T1); only concern if primary infection during pregnancy	Congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial calcification, MR, microcephaly) NB: 75% initially asymptomatic at birth	Majority subclinical; may have flu-like symptoms	IgM and IgG serology; PCR of amniotic fluid	Self-limiting in mother; spiramycin decreases fetal morbidity but not rate of transmission

* Indicates TORCH infection

Venous Thromboembolism

Epidemiology

- incidence of 12.1/10, 000 (DVT), and 5.4/10,000 (PE)
- increased risk VTE throughout pregnancy with highest risk of DVT in third trimester and post-partum period and highest risk of PE post-partum (first 6 weeks)

Risk Factors

- previous VTE, age >35, obesity, infection, bedrest/immobility, shock/dehydration, thrombophilias (see Hematology, H35)

Table 17. Risk Factors for VTE Specific to Pregnancy

Hypercoagulability	Stasis	Endothelial
Increased factors: II, V, VII, VIII, IX, X, XII, fibrinogen Increased platelet aggregation Decreased protein S, tPA, factors XI, XIII	Increased resistance to activated protein C Antithrombin can be normal or reduced Increased venous distensibility Decreased venous tone 50% decrease in venous flow in lower extremity by T3 Uterus is mechanical impediment to venous return	Vascular damage at delivery (C/S or SVD) Uterine instrumentation Peripartum pelvic surgery

Clinical Features

- most DVTs occur in the iliofemoral or calf veins with a predilection for the left leg
- signs of a pulmonary embolism are non-specific (as in non-pregnant women)
- unexplained spontaneous fetal loss

Investigations

- duplex venous Doppler sonography for DVT
- CXR and V/Q scan or spiral CT for PE

Management

- before initiating treatment, obtain a baseline CBC including platelets, and aPTT
- warfarin is contraindicated during pregnancy due to its potential teratogenic effects
- unfractionated heparin
  - bolus of 5,000 IU followed by an infusion of ~30,000 IU/24h
  - measure aPTT 6 h after the bolus

  - maintain aPTT at a therapeutic level (1.5-2x normal)
  - repeat q24h once therapeutic
  - heparin-induced thrombocytopenia (HIT) uncommon (3%) but serious complication
  - LMWH can also be used in pregnancy
- compression stockings
- poor evidence to support a recommendation for or against avoidance of prolonged sitting
- VTE prophylaxis
  - women on long-term anticoagulation: full therapeutic anticoagulation throughout pregnancy and for 6-12 wk postpartum
  - women with a non-active PMHx of VTE: unfractionated heparin regimens suggested
  - insufficient evidence in pregnancy to recommend routine use of LMWH for all patients
  - current prophylaxis regimens for acquired thrombophilias (e.g. APS syndrome) include low dose Aspirin® in conjunction with prophylactic heparin

Virchow's Triad for VTE
- Hypercoagulable state
- Stasis
- Endothelial damage

Normal Labour and Delivery

Definition of Labour

- true labour: regular, painful contractions of increasing intensity associated with progressive dilatation and effacement of cervix and descent of presenting part, or progression of station
  - preterm (>20 to <36+6 wk GA)
  - term (37-41+6 wk GA)
  - postterm (>42 wk GA)
- false labour: Braxton-Hicks contractions
  - irregular contractions, with unchanged intensity and long intervals, occur throughout pregnancy and not associated with any cervical dilatation, effacement, or descent
  - often relieved by rest or sedation

Maternal Triage Assessment
ID: Age, GPA, EDC, GA, GBS, Rh, Ser
CC
HPI: 4 key questions:
- Contractions: Since when, how close (q x min), how long (x s), how painful
- Bleeding: Since when, how much (pads), colour (pinky mucous=show vs. brownish vs. bright red ± clots), pain?, last U/S, trauma/intercourse?
- Fluid (ROM): Since when, large gush vs. trickle, soaked pants?, clear vs. green vs. red?, continuous?
- FM: As much as usual?, When last movement?, Kick counts (lie still for 1-2 h, cold juice, feel FM – should have 6 movements in 2 h)

PregHx: Any complications (HTN, GDM, infections), IPS/FTS screening, last U/S (BPP score, growth/estimated fetal weight, position), last vaginal exam
POBHx: Every previous pregnancy and outcome: Year, SVD/CS/miscarriage/abortion, baby size, length of labour, use of vacuum or forceps, complications
PMHx, Meds, Allergies, SHx
O/E: Maternal vitals, fetal heart tracing (baseline, variability, presence of accelerations/decelerations), Leopold's, vaginal exam, U/S

The Cervix

- dilatation: latent phase: 0-4 cm (variable time); active phase: 4-10 cm
- effacement: thinning of the cervix by percentage or length of cervix (cm)
- consistency: firm vs. soft
- position: posterior vs. anterior
- application: contact between the cervix and presenting part (i.e. well or poorly applied)
- see Bishop score (Table 22, OB38)

The Fetus

- **fetal lie**
  - orientation of the long axis of the fetus with respect to the long axis of the uterus (longitudinal, transverse, oblique)

- **fetal presentation**
  - fetal body part closest to the birth canal
    - breech (complete, frank, footling) (see Figure 5, OB23)
    - cephalic (vertex/occiput, face, asynclitic, brow)
    - transverse (shoulder)
    - compound (fetal extremity prolapses along with presenting part)
  - all except vertex are considered malpresentations (see *Obstetrical Complications*, OB16)

- **fetal position**
  - position of presenting part of the fetus relative to the maternal pelvis
    - OA: most common presentation ("normal") – left OA most common
    - OP: most rotate spontaneously to OA; may cause prolonged second stage of labour
    - OT: leads to arrest of dilatation
  - normally, fetal head enters maternal pelvis and engages in OT position
  - subsequently rotates to OA position (or OP in a small percentage of cases)

Reference Point for Describing Fetal Position
- Occiput for cephalic presentation
- Sacrum for breech presentation
- Mentum for face presentation

- **attitude**
  - flexion/extension of fetal head relative to shoulders
    - brow presentation: head partially extended (requires C/S)
    - face presentation: head fully extended
      - mentum posterior always requires C/S, mentum anterior will deliver vaginally

- **station**
  - position of presenting part relative to ischial spines – determined by vaginal exam
    - at ischial spines = station 0 = engaged
    - -5 to -1 cm above ischial spines or
    - +1 to +5 cm below ischial spines
    - alternatively stations can be placed on a scale from -3 to +3

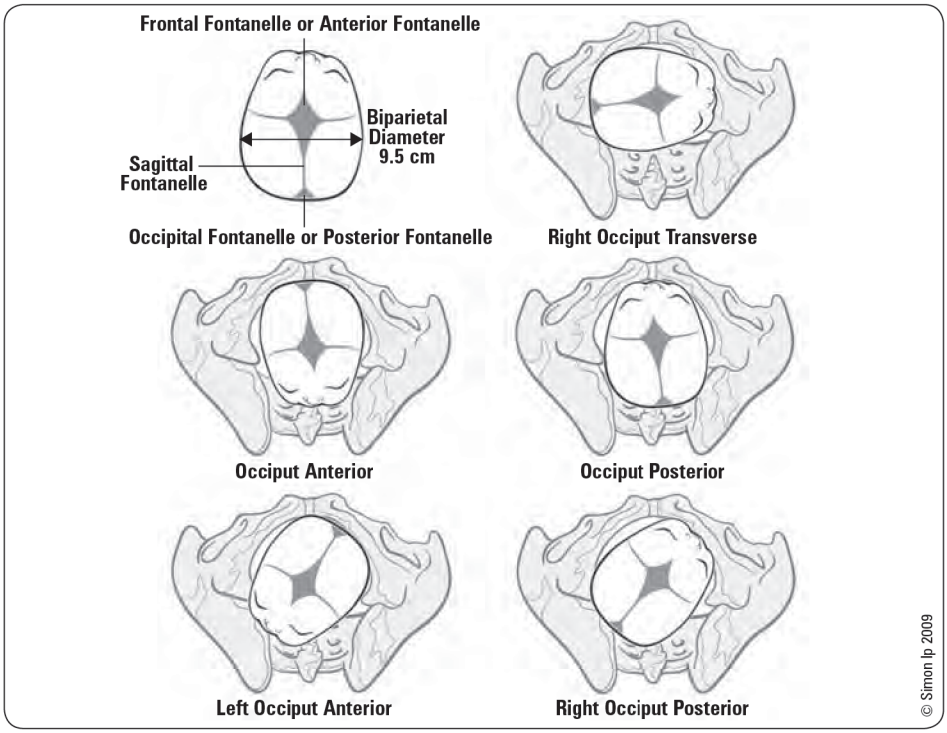


Figure 6. Fetal positions

Four Stages of Labour

First Stage of Labour

- latent phase
  - uterine contractions typically infrequent and irregular
  - slow cervical dilatation (usually to 4 cm) and effacement
- active phase
  - rapid cervical dilatation to full dilatation (nulliparous ≥1.0 cm/h, multiparous ≥1.2 cm/h)
  - phase of maximum slope on cervical dilatation curve
  - painful, regular contractions q2-3min, lasting 45-60 s
  - contractions strongest at fundus, weakest at lower segment

Second Stage of Labour

- from full dilatation to delivery of the baby, duration varies based on parity, contraction quality, and type of analgesia
- mother feels a desire to bear down and push with each contraction
- women may choose a comfortable position that enhances pushing efforts and delivery
  - upright (semi-sitting, squatting) and LLDP are supported in the literature
- progress measured by descent

Third Stage of Labour

- from baby's birth to separation and expulsion of the placenta
- can last up to 30 min before intervention indicated
- demonstrated by gush of fresh blood, umbilical cord lengthening, uterine fundus changing shape (firm and globular) and rising upward
- start oxytocin IV drip, or give 10 U IM or 5 mg IV push after delivery of anterior shoulder in anticipation of placental delivery, otherwise give after delivery of placenta
- routine oxytocin administration in third stage of labour can reduce the risk of PPH by >40%

Course of Normal Labour

Stage	Nulliparous	Multiparous
First	6-18 h	2-10 h
Second	30 min-3 h	5-30 min
Third	5-30 min	5-30 min

Signs of Placental Separation
- Gush of blood
- Lengthening of cord
- Uterus becomes globular
- Fundus rises

Continuous Support for Women During Childbirth
Cochrane DB Syst Rev 2011;16:CD003766
Study: Systematic review of 21 RCTs from 11 countries, 15,061 women in labour.
Intervention: Continuous support during labour vs. usual care.
Outcome: Effects on mothers and their babies.
Results: Continuous intrapartum support increased likehood of shorter labour, spontaneous vaginal birth, decrease in analgesia use, and a decrease in dissatisfaction with childbirth experience. Greatest benefit when provider is not a health care professional. Continuous support was also associated with decreased likelihood to have a Cesarean or instrumental vaginal birth, regional analgesia, or a baby with a low 5 min APGAR score.

Fourth Stage of Labour
- first postpartum hour
- monitor vital signs and bleeding, repair lacerations
- ensure uterus is contracted (palpate uterus and monitor uterine bleeding)
- inspect placenta for completeness and umbilical cord for presence of 2 arteries and 1 vein
- 3rd and 4th stages of labour most dangerous to the mother (i.e. hemorrhage)

The Cardinal Movements of the Fetus During Delivery

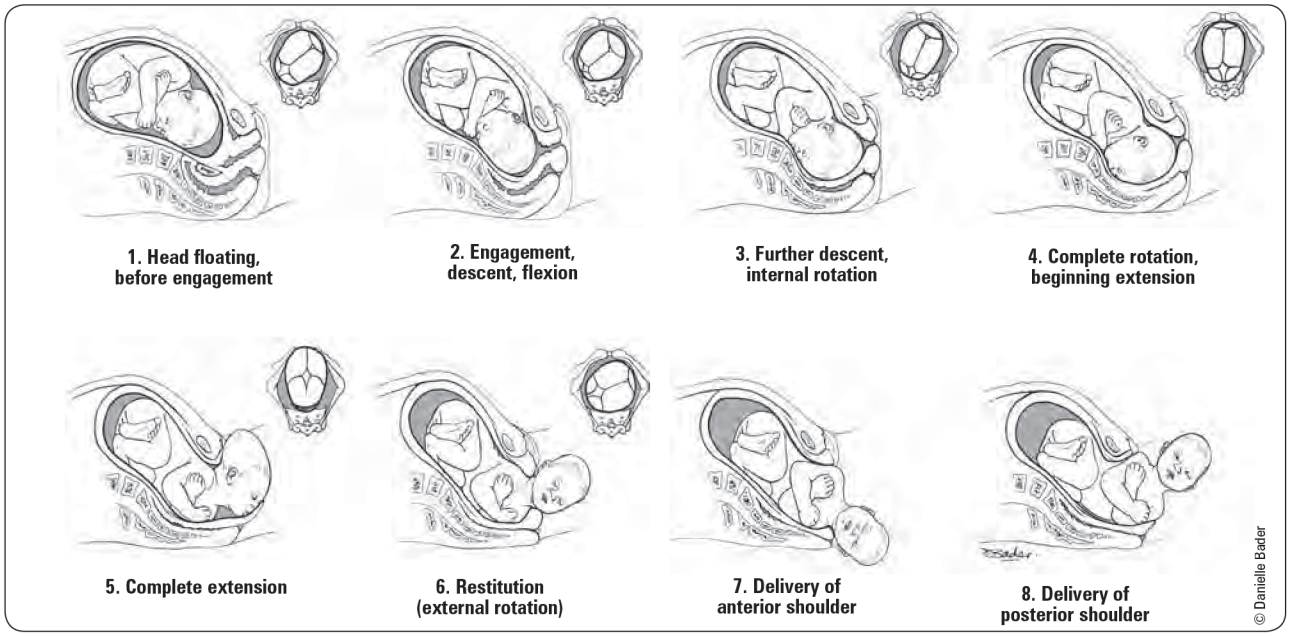


Figure 7. Cardinal movements of fetus during delivery
Adapted from illustration in Williams Obstetrics, 19th ed

Analgesic and Anesthetic Techniques in Labour and Birth

- pain or anxiety leads to high endogenous catecholamines, which produce a direct inhibitory effect on uterine contractility

Non-Pharmacologic Pain Relief Techniques
- reduction of painful stimuli
  - maternal movement, position change, counter-pressure, abdominal compression
- activation of peripheral sensory receptors
  - superficial heat and cold
  - immersion in water during labour
  - touch and massage, acupuncture, and acupressure
  - TENS
  - intradermal injection of sterile water
  - aromatherapy
- enhancement of descending inhibitory pathways
  - attention focusing and distraction
  - hypnosis
  - music and audio analgesia
  - biofeedback

Pharmacologic Methods (see Anesthesia and Perioperative Medicine, A2)
- nitrous oxide (e.g. self-administered Entonox®)
- narcotics (usually combined with anti-emetic)
- pudendal nerve block
- perineal infiltration with local anesthetic
- regional anesthesia (epidural block, combined spinal-epidural, spinal)

Fetal Monitoring in Labour

- see online **Fetal Heart Rate Tutorial**

Vaginal Exam

- membrane status
- cervical effacement (thinning), dilatation, consistency, position, application
- fetal presenting part, position, station
- bony pelvis size and shape
- monitor progress of labour at regular intervals and document in a partogram

Intrapartum Fetal Monitoring

- intermittent fetal auscultation with Doppler device q15-30min for 1 min in first stage active phase following a contraction, q5min during second stage when pushing has begun
- continuous electronic FHR monitoring reserved for abnormal auscultation, prolonged labour, and labour which is induced or augmented, meconium present, multiple gestation/fetal complication
  - use of continuous electronic monitoring shown to lead to higher intervention rates and no improvement in outcome for the neonate when used routinely in all patients (ie no risk factors)
  - techniques for continuous monitoring include external (Doppler) vs. internal (fetal scalp electrode) monitoring
- fetal scalp sampling should be used in conjunction with electronic FHR monitoring and contraction monitoring (CTG) to resolve the interpretation of abnormal or atypical patterns

Fetal Scalp Blood Sampling

- cervix must be adequately dilated
- indicated when atypical or abnormal fetal heart rate is suggested by clinical parameters including heavy meconium or moderately to severely abnormal FHR patterns, including unexplained low variability, repetitive late decelerations, complex variable decelerations, fetal cardiac arrhythmias

Done by measuring pH or more recently fetal lactate

- pH ≥7.25: normal, repeat if abnormal FHR persists
- pH 7.21-7.24: repeat assessment in 30 min or consider delivery if rapid fall since last sample
- pH ≤7.20: indicates fetal acidosis, delivery is indicated

- contraindications
- known or suspected fetal blood dyscrasia (hemophilia, von Willebrand disease)
- active maternal infection (HIV, genital herpes)

Electronic FHR Monitoring

- FHR measured by Doppler; contractions measured by tocometer
- described in terms of baseline FHR, variability (short-term, long-term), and periodicity (accelerations, decelerations)

- **Baseline FHR**
  - normal range is 110-160 bpm
  - parameter of fetal well-being vs. distress

- **Variability**
  - physiologic variability is a normal characteristic of FHR
  - variability is measured over a 15 min period and is described as: absent, minimal (<6 bpm), moderate (6-25 bpm), marked (>25 bpm)
  - normal variability indicates fetal acid-base status is acceptable
  - can only be assessed by electronic fetal monitoring (CTG)
  - variability decreases intermittently even in healthy fetus
  - see Table 19, OB36

- **Periodicity**
  - accelerations: increase of ≥15 bpm for ≥15 s, in response to fetal movement or uterine contraction (or ≥10 bpm for ≥10 s if <32 wk GA)
  - decelerations: 3 types, described in terms of shape, onset, depth, duration recovery, occurrence, and impact on baseline FHR and variability

Approach to the Management of Abnormal FHR

POISON – ER
Position (left lateral decubitus position)
O$_2$ (100% by mask)
IV fluids (corrects maternal hypotension)
Fetal **S**calp stimulation
Fetal **S**calp electrode
Fetal **S**calp pH
Stop **O**xytocin
Notify MD

Vaginal **E**xam to rule out cord prolapse
Rule out fever, dehydration, drug effects, prematurity

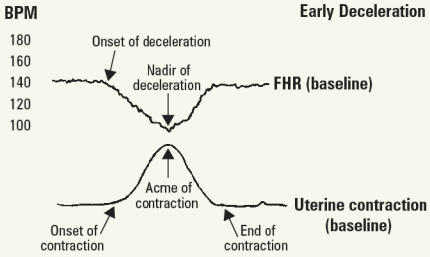
- If above fails, consider C/S

Table 18. Factors Affecting Fetal Heart Rate

	Fetal Tachycardia (FHR >160 bpm)	Fetal Bradycardia (FHR <110 bpm)	Decreased Variability
Maternal Factors	Fever, hyperthyroidism, anemia, dehydration	Hypothermia, hypotension, hypoglycemia, position, umbilical cord occlusion	Infection Dehydration
Fetal Factors	Arrhythmia, anemia, infection, prolonged activity, chronic hpoxemia, congenital anomalies	Rapid descent, dysrhythmia, heart block, hyopoxia, vagal stimulation (head compression), hypothermia, acidosis	CNS anomalies Dysrhythmia Inactivity/sleep cycle, preterm fetus
Drugs	Sympathomimetics	β-blockers Anesthetics	Narcotics, sedatives Magnesium sulphate, β-blockers
Uteroplacental	Early hypoxia (abruption, HTN) Chorioamnionitis	Late hypoxia (abruption, HTN) Acute cord prolapse Hypercontractility	Hypoxia

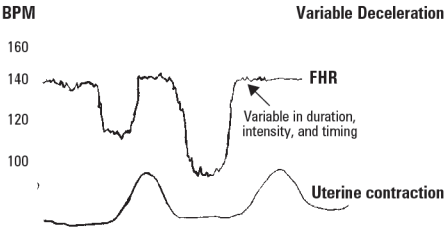
Table 19. Comparison of Decelerations

Early Decelerations
- Uniform shape with onset early in contraction, returns to baseline by end of contraction, mirrors contraction (nadir occurs at peak of contraction)
- Gradual deceleration and return to baseline
- Often repetitive; no effect on baseline FHR or variability
- Benign, due to vagal response to head compression



Variable Decelerations
- Variable in shape, onset, and duration
- Most common type of periodicity seen during labour
- Often with abrupt drop in FHR >15 bpm below baseline (>15 s, <2 min); usually no effect on baseline FHR or variability
- Due to cord compression or, in second stage, forceful pushing with contractions

Complicated Variable Decelerations
- FHR drop <70 bpm for >60 s
- Loss of variability or decrease in baseline after deceleration
- Biphasic deceleration
- Slow return to baseline
- Baseline tachycardia or bradycardia
- May be associated with fetal academia



Late Decelerations
- Uniform shape with onset, nadir, and recovery occurring after peak of contraction, slow return to baseline
- May cause decreased variability and change in baseline FHR
- Due to fetal hypoxia and acidemia, maternal hypotension, or uterine hypertonus
- Usually a sign of uteroplacental insufficiency (an ominous sign)

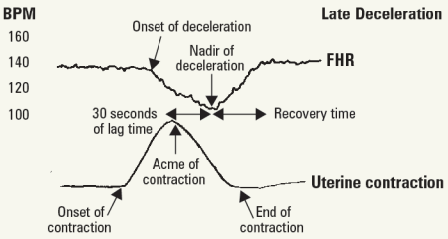


Table 20. Classification of Intrapartum EFM Tracings

	Normal Tracing (Category 1)	Atypical Tracing* (Category 2)	Abnormal Tracing* (Category 3)
Baseline	110-160 bpm	Bradycardia 100-110 bpm Tachycardia >160 for 30-80 min Rising baseline	Bradycardia <100 bpm Tachycardia >160 bpm for >80 min Erratic baseline
Variability	6-25 bpm ≤5 bpm for <40 min	≤5 bpm for 40-80 min	<5 bpm for >80 min ≥25 bpm for >10 min

Cochrane Review: CTG as a Form of EFM for Fetal Assessment During Labour
Cochrane DB Syst Rev 2013;5:CD006066
Purpose: To examine the effectiveness of continuous electronic fetal monitoring or cardiotocography during labour.
Selection Criteria: Randomized and quasi-randomized controlled trials comparing continuous CTG (with and without fetal blood sampling) to a) no fetal monitoring, b) intermittent auscultation, or c) intermittent CTG.
Results: 13 trials, 37,000 women. Continuous CTG compared with intermittent auscultation showed no difference in overall perinatal death rate or cerebral palsy rates. Nonetheless, neonatal seizures were halved (RR 0.50, 95% CI 0.31-0.80) and there was a significant increase in Cesarean sections with CTG (RR 1.63, 95% CI 1.29-2.07) and instrumental vaginal birth (RR 1.15, 95% CI 1.01-1.33).
Conclusion: Continuous CTG may reduce the incidence of neonatal seizures, but has no effect on cerebral palsy rates, infant mortality, or other measures of neonatal well-being. Continuous CTG was also associated with an increase in Cesarean sections and instrumental deliveries.

Rule of 60s Suggesting Severe Variable Decelerations
Deceleration to <60 bpm
>60 bpm below baseline
>60 s in duration with slow return to baseline

Table 20. Classification of Intrapartum EFM Tracings (continued)

	Normal Tracing (Category 1)	Atypical Tracing* (Category 2)	Abnormal Tracing* (Category 3)
Decelerations	None Early decelerations Occasional uncomplicated variable decelerations	Repetitive (≥3) uncomplicated variable decelerations Occasional late decelerations Any prolonged deceleration (2-3 min)	Repetitive (≥3) complicated variable decelerations Repetitive late decelerations Any prolonged deceleration (≥3 min)
Accelerations	Accelerations spontaneous or during scalp stimulation	Absent with scalp stimulation	Nearly absent
Action	EFM may be interrupted for ≤30 min if mother/fetus stable	Further assessment required	Action required: review clinical situation, obtain scalp pH, prepare for possible delivery

Adapted from SOGC Guidelines, September 2008
*Previous classification was "reassuring" vs. "non-reassuring", but distinction is now made between tracings that have some concerning changes but do not require immediate action (atypical) versus those with major concerns requiring immediate intervention (abnormal)

Fetal Oxygenation

- uterine contractions during labour decrease uteroplacental blood flow, which results in reduced oxygen delivery to the fetus
- most fetuses tolerate this reduction in flow and have no adverse effects
- distribution of oxygen to the fetus depends on maternal, uteroplacental, and fetal factors
- fetal response to hypoxia/asphyxia:
  - decreased movement, tone, and breathing activities
  - anaerobic metabolism (decreased pH)
  - transient fetal bradycardia followed by fetal tachycardia
  - redistribution of fetal blood flow
    - increased flow to brain, heart, and adrenals
    - decreased flow to kidneys, lungs, gut, liver, and peripheral tissues
    - increase in blood pressure

Table 21. Factors Affecting Fetal Oxygenation

Factor	Mechanism	Example
Maternal	Decreased maternal oxygen carrying capacity	Significant anemia (iron deficiency, hemoglobinopathies), carboxyhemoglobin (smokers)
	Decreased uterine blood flow	Hypotension (blood loss, sepsis), regional anesthesia, maternal positioning
	Chronic maternal conditions	Vasculopathies (SLE, type 1 DM, chronic HTN), antiphospholipid syndrome, cyanotic heart disease, COPD
Uteroplacental	Uterine hypertonus	Placental abruption, hyperstimulation secondary to oxytocin, prostaglandins, or normal labour
	Uteroplacental dysfunction	Placental abruption, placental infarction (dysfunction marked by IUGR, oligohydramnics, abnormal Doppler studies), chorioamnionitis, placental edema (DM, hydrops), placental senescence (post-dates)
Fetal	Cord compression	Oligohydramnios, cord prolapse, or entanglement
	Decreased fetal oxygen carrying capacity	Significant anemia (isoimmunization, feto-maternal bleed), carboxyhemoglobin (exposure to smokers)

Induction of Labour

Definition

- artificial initiation of labour in a pregnant woman prior to spontaneous initiation to deliver the fetus and placenta

Prerequisites for Labour Induction

- capability for C/S if necessary
- maternal
  - short, thin, soft, anterior cervix with open os ("inducible" or "ripe")
  - if cervix is not ripe, use prostaglandin vaginal insert (Cervidil®), prostaglandin gel (Prepidil®), or Foley catheter
- fetal
  - normal fetal heart tracing
  - cephalic presentation
  - adequate fetal monitoring available

Induction is indicated when the risk of continuing pregnancy exceeds the risks associated with induced labour and delivery

- likelihood of success determined by Bishop score
  - cervix considered unfavourable if <6
  - cervix favourable if ≥6
  - score of 9-13 associated with high likelihood of vaginal delivery

Table 22. Bishop Score

Cervical Characteristic	0	1	2	3
Position	Posterior	Mid	Anterior	–
Consistency	Firm	Medium	Soft	–
Effacement (%)	0-30	40-50	60-70	≥80
Dilatation (cm)	0	1-2	3-4	≥5
Station of Fetal Head	-3	-2	-1, 0	+1, +2, +3

Induction vs. Augmentation

Induction is the artificial initiation of labour

Augmentation promotes contractions when spontaneous contractions are inadequate

Indications
- post-dates pregnancy (generally >41 wk) = most common reason for induction
- maternal factors
  - DM = second most common reason for induction
  - gestational HTN
  - other maternal medical problems, e.g. renal or lung disease, chronic hypertension, cholestasis or pregnancy
  - maternal age over 40
- maternal-fetal factors
  - isoimmunization, PROM, chorioamnionitis, post-term pregnancy
- fetal factors
  - suspected fetal jeopardy as evidenced by biochemical or biophysical indications
  - fetal demise, IUGR, oligo/polyhydraminos, anomalies requiring surgical intervention, twins
  - previous still birth, low PAPP-A

Consider the Following Before Induction
- Indication for induction
- Contraindications
- GA
- Cervical favourability
- Fetal presentation
- Potential for CPD
- Fetal well-being/FHR
- Membrane status

Risks
- failure to achieve labour and/or vaginal birth
- uterine hyperstimulation with fetal compromise or uterine rupture
- maternal side effects to medications
- uterine atony and PPH

Contraindications
- maternal
  - prior classical or inverted T-incision C/S or uterine surgery (e.g. myomectomy)
  - unstable maternal condition
  - active maternal genital herpes
  - invasive cervical carcinoma
  - pelvic structure deformities
- maternal-fetal
  - placenta previa or vasa previa
  - cord presentation
- fetal
  - fetal distress, malpresentation /abnormal lie, preterm fetus without lung maturity

Induction Methods

CERVICAL RIPENING

Definition
- use of medications or other means to soften, efface, and dilate the cervix, increases likelihood of successful induction
- ripening of an unfavourable cervix (Bishop score <6) is warranted prior to induction of labour

Methods
- intravaginal prostaglandin PGE2 gel (Prostin® gel): long and closed cervix
  - recommended dosing interval of prostaglandin gel is every 6 to 12 h up to 3 doses
- intravaginal PGE2 (Cervidil®): long and closed cervix, may use if ROM
  - continuous release, can be removed if needed
  - controlled release PGE2
- Foley catheter placement to mechanically dilate the cervix

Evidence for Cervical Ripening Methods (SOGC Guidelines)
- Meta-analysis of five trials has concluded that the use of oxytocin to ripen the cervix is not effective
- Since the best dose and route of misoprostol for labour induction with a live fetus are not known and there are concerns regarding hyperstimulation, the use of misoprostol for induction of labour should be within clinical trials only (Level Ib evidence) or in cases of intrauterine fetal death to initiate labour

INDUCTION OF LABOUR

Amniotomy

- artificial rupture of membranes (amniotomy) to stimulate prostaglandin synthesis and secretion; may try this as initial measure if cervix is open and soft, the membranes can be felt, and if the head is present at the cervix
- few studies address the value of amniotomy alone for induction of labour
- amniotomy plus intravenous oxytocin: more women delivered vaginally at 24 h than amniotomy alone (relative risk = 0.03) and had fewer instrumental vaginal deliveries (relative risk = 5.5)

Oxytocin

- oxytocin (Pitocin®): 10 U in 1L NS, run at 0.5-2 mU/min IV increasing by 1-2 mU/min q20-60min to a max of 36-48 mU/min
  - reduces rate of unsuccessful vaginal deliveries within 24 h when used alone (8.3% vs. 54%, RR 0.16)
  - ideal dosing regime of oxytocin is not known
  - current recommendations: use the minimum dose to achieve active labour and increase q30min as needed
  - reassessment should occur once a dose of 20 mU/min is reached
- potential complications
  - hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus)
  - uterine muscle fatigue, uterine atony (may result in PPH)
  - vasopressin-like action causing anti-diuresis

Oxytocin $t_{1/2}$ = 3-5 min

Intravaginal PGE2 (Cervidil®) Compared to Intravaginal Prostaglandin Gel
4 RCTs have compared the two with varying results, depending on the dosing regime of gel used.
Theoretical advantages of Cervidil®:
- Insertion without a speculum
- Slow, continuous release
- Only one dose required
- Ability to use oxytocin 30 min after removal
- Ability to remove insert if required (i.e. excessive uterine activity)

Augmentation of Labour

- augmentation of labour is used to promote adequate contractions when spontaneous contractions are inadequate and cervical dilatation or descent of fetus fails to occur
- oxytocin (0.5-2 mU/min IV increasing by 1-2 mU/min q20-60min to a max of 36-48 mU/min)

Provided there are no contraindications, oxytocin is utilized to improve uterine contraction strength and/or frequency

Abnormalities and Complications of Labour and Delivery

Meconium in Amniotic Fluid

Epidemiology

- present early in labour in 10% of pregnancies
- in general, meconium may be present in up to 25% of all labours; usually NOT associated with poor outcome, but extra care is required at time of delivery to avoid aspiration. Concern is fluid changes from clear to meconium stained. Always abnormal if seen in preterm patient

Etiology

- likely cord compression ± uterine hypertonia
- may indicate undiagnosed breech
- increasing meconium during labour may be a sign of fetal distress

Features

- may be watery or thicker
- light yellow/green or dark green-black in colour

Dark green or black meconium is associated with lower APGARs and increased risk of meconium aspiration

Treatment

- call respiratory therapy, neonatology, or pediatrics to delivery room
- oropharynx suctioning upon head expulsion or immediately after delivery if baby not breathing spontaneously (do NOT stimulate infant before)
- consider amnioinfusion of ~800 mL of IV NS over 50-80 min during active stage of labour and a maintenance dose of ~3 mL/min until delivery
- closely monitor FHR for signs of fetal distress

Abnormal Progression of Labour (Dystocia)

Definition
- expected patterns of descent of the presenting part and cervical dilatation fail to occur in the appropriate time frame; can occur in all stages of labour
- during active phase: >4 h of <0.5 cm/h
- during 2nd phase: >1 h with no descent during active pushing

Etiology
- Power (leading cause): contractions (hypotonic, incoordinate), inadequate maternal expulsive efforts
- Passenger: fetal position, attitude, size, anomalies (hydrocephalus)
- Passage: pelvic structure (CPD), maternal soft tissue factors (tumours, full bladder or rectum, vaginal septum)
- Psyche: hormones released in response to stress may contribute to dystocia; psychological and physiological stress should be evaluated as part of the management once dystocia has been diagnosed

The 4 Ps of Dystocia

Power
Passenger
Passage
Psyche

Management
- confirm diagnosis of labour (rule out false labour)
- search for factors of CPD
- diagnosed if adequate contractions measured by intrauterine pressure catheter (IUPC) with no descent/dilatation for >2 h
- management: if CPD ruled out, IV oxytocin augmentation ± amniotomy

Risks of Dystocia
- inadequate progression of labour is associated with an increased incidence of:
  - maternal stress
  - maternal infection
  - postpartum hemorrhage
  - need for neonatal resuscitation
  - fetal compromise (from uterine hyperstimulation)
  - uterine rupture
  - hypotension

Shoulder Dystocia

Definition
- fetal anterior shoulder impacted above symphysis pubis after fetal head has been delivered
- life threatening emergency

Etiology/Epidemiology
- incidence 0.15-1.4% of deliveries
- occurs when breadth of shoulders is greater than biparietal diameter of the head

Risk Factors
- maternal: obesity, DM, multiparity, previous shoulder dystocia
- fetal: prolonged gestation, macrosomia
- labour
  - prolonged 2nd stage
  - instrumental midpelvic delivery

Clinical Features
- "turtle sign": head delivered but retracts against inferior portion of pubic symphysis
- complications
  - fetal:
    - hypoxic ischemic encephalopathy (chest compression by vagina or cord compression by pelvis can lead to hypoxia)
    - brachial plexus injury (Erb's palsy: C5-C7; Klumpke's palsy: C8-T1), 90% resolve within 6 mo
    - fracture (clavicle, humerus, cervical spine)
    - death
  - maternal:
    - perineal injury
    - PPH (uterine atony, lacerations)
    - uterine rupture

Treatment
- goal: to displace anterior shoulder from behind symphysis pubis; follow a stepwise approach of maneuvers until goal achieved

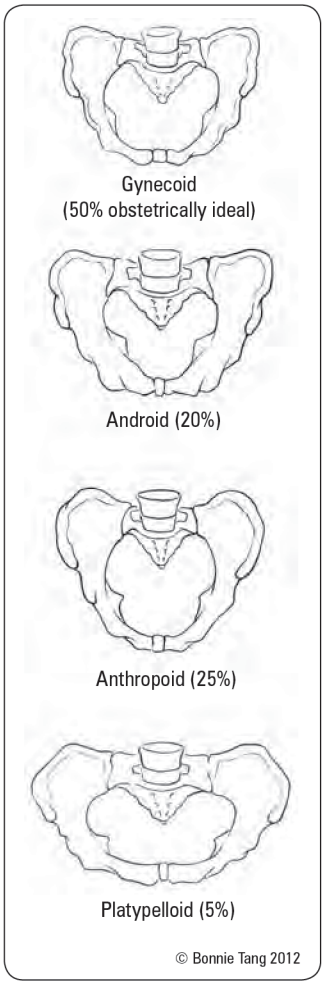


Figure 8. Types of pelvis

- other options
  - cleidotomy (deliberate fracture of neonatal clavicle)
  - Zavanelli maneuver: replacement of fetus into uterine cavity and emergent C/S
  - symphysiotomy

Prognosis
- 1% risk of long-term disability for infant

Approach to the Management of Shoulder Dystocia

ALARMER
Apply suprapubic pressure and ask for help
Legs in full flexion (McRobert's maneuver)
Anterior shoulder disimpaction (suprapubic pressure)
Release posterior shoulder by rotating it anteriorly with hand in the vagina under adequate anesthesia
Manual corkscrew i.e. rotate the fetus by the posterior shoulder until the anterior shoulder emerges from behind the maternal symphysis
Episiotomy
Rollover (on hands and knees)

*Note that suprapubic pressure and McRobert's maneuver together will resolve 90% of cases

Umbilical Cord Prolapse

Definition
- descent of the cord to a level adjacent to or below the presenting part, causing cord compression between presenting part and pelvis

Etiology/Epidemiology
- increased incidence with prematurity/PROM, fetal malpresentation (~50% of cases), low-lying placenta, polyhydramnios, multiple gestation, CPD
- incidence: 1/200 – 1/400 deliveries

Clinical Features
- visible or palpable cord
- FHR changes (variable decelerations, bradycardia, or both)

Treatment
- emergency C/S
- O2 to mother, monitor fetal heart
- alleviate pressure of the presenting part on the cord by placing digit in vagina (maintain this position until C/S)
- keep cord warm and moist by replacing it into the vagina ± applying warm saline soaks
- roll mom onto all fours
- position mother in Trendelenburg or knee-to-chest position
- if fetal demise or too premature (<22 wk), allow labour and delivery

- 1/3 of protraction disorders develop into 2° arrest of dilatation due to CPD
- 2/3 of protraction disorders progress through labour to vaginal delivery

Umbilical Cord Accident Causes
- Nuchal cord
  - Type A (looped)
  - Type B (hitched)
- Body loop
- Single artery
- True knot
- Torsion
- Velamentous
- Short cord <35 cm
- Long cord >80 cm

Uterine Rupture

Etiology/Epidemiology
- associated with previous uterine scar (in 40% of cases), hyperstimulation with oxytocin, grand multiparity, and previous intrauterine manipulation
- generally occurs during labour, but can occur earlier with a classical incision
- 0.5-0.8% incidence, up to 12% with classical incision

Clinical Features
- prolonged fetal bradycardia – most common presentation
- acute onset of constant lower abdominal pain, may not have pain if receiving epidural analgesia
- hyper or hypotonic uterine contractions
- vaginal bleeding
- intra-abdominal hemorrhage

Risk Factors
- uterine scarring (i.e. previous uterine surgeries including Cesarean, perforation with D&C, myomectomy)
- excessive uterine stimulation (i.e. protracted labour, oxytocin, prostaglandins)
- uterine trauma (i.e. operative equipment, ECV)
- multiparity
- uterine abnormalities
- placenta accreta

Treatment
- rule out placental abruption
- immediate delivery for fetal survival
- maternal stabilization (may require hysterectomy), treat hypovolemia

Complications
- maternal mortality 1-10%
- maternal hemorrhage, shock, DIC
- amniotic fluid embolus
- hysterectomy if uncontrollable hemorrhage
- fetal distress, associated with 50% fetal mortality

Maternal Mortality Causes
- Thromboembolism
- Cardiac event
- Suicide
- Sepsis
- Ectopic pregnancy
- HTN
- Amniotic fluid embolism
- Hemorrhage

* In Canada (2013), lifetime risk of maternal death is 1 in 5,200

Amniotic Fluid Embolus

Definition
- amniotic fluid debris in maternal circulation triggering an anaphylactoid immunologic response

Etiology/Epidemiology
- rare intrapartum or immediate postpartum complication
- 60-80% maternal mortality rate, accounts for 10% of all maternal deaths
- leading cause of maternal death in induced abortions and miscarriages
- 1/8,000-1/80,000 births

Risk Factors
- placental abruption
- rapid labour
- multiparity
- uterine rupture
- uterine manipulation

Differential Diagnosis
- pulmonary embolus, drug-induced anaphylaxis, septic shock, eclampsia, HELLP syndrome, abruption, chronic coagulopathy

Clinical Features
- sudden onset of respiratory distress, cardiovascular collapse (hypotension, hypoxia), and coagulopathy
- seizure in 10%
- ARDS and left ventricular dysfunction seen in survivors

Management
- supportive measures (high flow O_2, ventilation support, fluid resuscitation, inotropic support, ± intubation), coagulopathy correction
- ICU admission

Chorioamnionitis

Definition
- infection of the chorion, amnion, and amniotic fluid typically due to ascending infection by organisms of normal vaginal flora

Etiology/Epidemiology
- incidence 1-5% of term pregnancies and up to 25% in preterm deliveries
- ascending from vagina
- predominant microorganisms include: GBS, *Bacteroides* and *Prevotella* species, *E. coli*, and anaerobic *Streptococcus*

Risk Factors
- prolonged ROM, long labour, multiple vaginal exams during labour, internal monitoring
- bacterial vaginosis and other vaginal infections

Clinical Features of Chorioamnionitis
- Temperature
- Tachycardia (maternal or fetal)
- Tenderness (uterine)
- Foul discharge

Clinical Features
- maternal fever, maternal or fetal tachycardia, uterine tenderness, foul, and purulent cervical discharge

Investigations
- CBC: leukocytosis
- amniotic fluid: leukocytes or bacteria

Treatment
- IV antibiotics
  - ampicillin (2 g IV q6h) and gentamicin (1.5 mg/kg q8h)
  - anaerobic coverage (i.e. clindamycin if C/S)
- expedient delivery regardless of gestational age

Complications
- bacteremia of mother or fetus, wound infection if C/S, pelvic abscess, infant meningitis

Operative Obstetrics

Operative Vaginal Delivery

Definition
- forceps or vacuum extraction

Indications
- fetal
  - atypical or abnormal fetal heart rate tracing, evidence of fetal compromise
  - consider if second stage is prolonged as this may be due to poor contractions or failure of fetal head to rotate
- maternal
  - need to avoid voluntary expulsive effort (e.g. cardiac/cerebrovascular disease)
  - exhaustion, lack of cooperation, and excessive analgesia may impair pushing effort

Contraindications
- non-cephalic presentation
- unengaged head
- cervix incompletely dilated

Prerequisites for Operative Vaginal Delivery

ABCDEFGHIJK
Anesthesia (adequate)
Bladder empty
Cervix fully dilated and effaced with ROM
Determine position of fetal head
Equipment ready (including facilities for emergent C/S)
Fontanelle (posterior fontanelle midway between thighs)
Gentle traction
Handle elevated
Incision (episiotomy)
Once **J**aw visible remove forceps
Knowledgeable operator

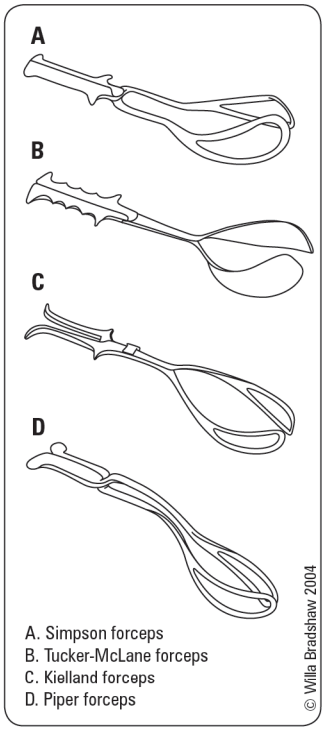
Forceps

Outlet Forceps Position
- head visible between labia in between contractions
- sagittal suture in or close to AP diameter
- rotation cannot exceed 45°

Low Forceps Position
- presenting part at station +2 or greater
- subdivided based on whether rotation less than or greater than 45 degrees

Mid Forceps Position
- presenting part below spines but above station +2

Types of Forceps
- Simpson or Tucker-McLane forceps for OA presentations
- Kielland (rotational) forceps when rotation of head is required
- Piper forceps for breech



A. Simpson forceps
B. Tucker-McLane forceps
C. Kielland forceps
D. Piper forceps

Figure 9. Types of forceps

Vacuum Extraction

- traction instrument used as alternative to forceps delivery; aids maternal pushing
- contraindications: <34 wk GA, fetal head deflexed, fetus requires rotation, fetal condition (e.g. bleeding disorder)

Table 23. Advantages and Disadvantages of Forceps versus Vacuum Extraction

	Forceps	Vacuum Extraction
Advantages	Higher overall success rate for vaginal delivery Decreased incidence of fetal morbidity	Easier to apply Less anesthesia required Less maternal soft-tissue injury compared to forceps
Disadvantages	Greater incidence of maternal injury	Contraindicated if fetus at risk for coagulation defect Suitable only for vertex presentations Maternal pushing required Contraindicated in preterm delivery
Complications	Maternal: anesthesia risk, lacerations, injury to bladder, uterus, or bone, pelvic nerve damage, PPH, infections Fetal: fractures, facial nerve palsy, trauma to face/scalp, intracerebral hemorrhage, cephalohematoma, cord compression	Increased incidence of cephalohematoma and retinal hemorrhages compared to forceps Subgaleal hemorrhage, Subaponeurotic hemorrhage, Soft tissue trauma

Limits for Trial of Vacuum
- After 3 pulls over 3 contractions with no progress
- After 3 pop-offs with no obvious cause
- 20 min and delivery is not imminent

Lacerations

- first degree: involves skin and vaginal mucosa but not underlying fascia and muscle
- second degree: involves fascia and muscles of the perineal body but not the anal sphincter
- third degree: involves the anal sphincter but does not extend through it
- fourth degree: extends through the anal sphincter into the rectal mucosa

Muscles of Perineal Body
- Superficial transverse perineal
- Bulbocavernous
- External anal sphincter

Episiotomy

Definition
- incision in the perineal body at the time of delivery
- essentially a controlled second degree laceration
- midline: incision through central tendinous portion of perineal body and insertions of superficial transverse perineal and bulbocavernosus muscle
  - heals better, but increases risk of 3rd/4th degree tears
- mediolateral: incision through bulbocavernosus, superficial transverse perineal muscle, and levator ani
  - reduced risk of extensive tear but more painful
  - easier to repair

Indications
- to relieve obstruction of the unyielding perineum
- instrumental delivery
- controversial between practitioners as to whether it is preferable to make a cut or let the perineum tear as needed
- current evidence suggests letting perineum tear and then repair as needed (restricted use)

Complications
- infection, hematoma, extension into anal musculature or rectal mucosa, fistula formation, incontinence

Risk Factors for Primary and Subsequent Anal Sphincter Lacerations
Am J Obstet Gynecol 2007;196:344
Objective: Assess effects of pregnancy, delivery method, and parity on risk of primary and secondary anal sphincter laceration in women with 1st vaginal delivery (VD), VBAC, or 2nd VD.
Methods: Retrospective cohort study of all deliveries at one hospital from 1995-2002.
Conclusion: 20,674 live singleton deliveries were included. Women with first VD and VBAC both had OR 5.1 for laceration compared to 2nd VD. Forceps and midline episiotomy significantly increased risk of laceration for all 3 groups. Second stage of labour >2 h only increased risk for 1st VD. Factors that had no significant increase in risk: infant birth weight >3,500 g and vacuum delivery. Women with prior anal sphincter laceration are at 3x increased risk for subsequent sphincter laceration, compared with women with prior vaginal delivery without sphincter laceration.

Restrictive vs. Routine Episiotomies with Vaginal Births
Cochrane DB Syst Rev 2009;1:CD000081
Study: This systematic review and meta-analysis of 8 RCTs assessed the effects of restrictive (only done for fetal indications or if severe perineal trauma was judged to be imminent) and routine (liberally done to prevent any tear) use of episiotomy during vaginal birth.
Patients: Of the 2,709 patients in the routine episiotomy group, 2,035 (75%) women had episiotomies. In the restrictive episiotomy group, 776 (28%) of the 2,733 women had episiotomies.
Results: Restrictive episiotomies appear to have less severe perineal trauma (RR 0.67), less suturing (RR 0.71), and fewer healing complications at 7 d (RR 0.69) compared to routine episiotomies. There is no difference for pain measures, dyspareunia, urinary incontinence, and severe vaginal or perineal trauma, but there was an increased risk of anterior perineal trauma (RR 1.84) with restrictive episiotomy. Similar results were obtained when comparing restrictive versus routine mediolateral versus midline episiotomy.
Conclusions: Compared to routine use, restrictive use of episiotomy during vaginal delivery appears to be more beneficial.

Cesarean Delivery

Epidemiology
- incidence 20-25%

Indications
- maternal: obstruction, active herpetic lesion on vulva, invasive cervical cancer, previous uterine surgery (past C/S is most common), underlying maternal illness (eclampsia, HELLP syndrome, heart disease)
- maternal-fetal: failure to progress, placental abruption or previa, vasa previa
- fetal: abnormal fetal heart tracing, malpresentation, cord prolapse, certain congenital anomalies

Types of Cesarean Incisions
- skin
  - transverse (i.e. Pfannensteil)
    - decreased exposure and slower entry
    - improved strength and cosmesis
  - vertical midline
    - rapid peritoneal entry and increased exposure
    - increased dehiscence
- uterine
  - low transverse (most common): in noncontractile segment
    - decreased chance for rupture in subsequent pregnancies
  - low vertical
    - used for very preterm infants, poorly developed maternal lower uterine segment
  - classical (rare): in thick, contractile segment
    - used for transverse lie, fetal anomaly, >2 fetuses, lower segment adhesions, obstructing fibroid, morbidly obese patients

Risks/Complications
- anesthesia
- hemorrhage (average blood loss ~1,000 cc)
- infection (UTI, wound, endometritis)
  - single dose prophylactic antibiotic should be used (e.g. cefazolin 1-2 g)
- injury to surrounding structures (bowel, bladder, ureter, uterus)
- thromboembolism (DVT, PE)
- increased recovery time/hospital stay
- maternal mortality (<0.1%)

Common OR Questions

7 Layers to Dissect
Skin, fatty layer, fascia, muscle separation (rectus abdominus), peritoneum, bladder flap, uterus

Layers of the Rectus Sheath
Above the arcuate line: external oblique, internal oblique, rectus abdominis, internal oblique, transversus abdominis
Below the arcuate line: external oblique, internal oblique, transversus abdominis, rectus abdominis

Name of the Obliterated Umbilical Ligament
Urachus

Vaginal Birth After Cesarean (Trial of Labour After Cesarean)

- recommended after previous low transverse incision
- success rate varies with indication for previous C/S (generally 60-80%)
- risk of uterine rupture (<1% with low transverse incision)

Contraindications
- previous classical, inverted T, or unknown uterine incision, or complete transection of uterus (6% risk of rupture)
- history of uterine surgery (e.g. myomectomy) or previous uterine rupture
- multiple gestation
- non-vertex presentation or placenta previa
- inadequate facilities or personnel for emergency C/S

VBAC
- Rate of VBAC ranges from 60-82%
- No significant difference in maternal deaths or hysterectomies between VBAC or C/S
- Uterine rupture more common in VBAC group
- Evidence regarding fetal outcome is lacking

Safety of vaginal birth after Cesarean section: A systematic review. *Obstet Gynecol* 2004;103:420-9

Puerperal Complications

- puerperium: 6 wk period of adjustment after pregnancy when pregnancy-induced anatomic and physiologic changes are reversed

Postpartum Hemorrhage

Definition
- loss of >500 mL of blood at the time of vaginal delivery, or >1,000 mL with C/S
- early (immediate) – within first 24 h postpartum
- late (delayed) – after 24 h but within first 6 wk

Epidemiology
- incidence 5-15%

Etiology (4 Ts)
1. Tone
- uterine atony
  - most common cause of PPH
  - avoid by giving oxytocin with delivery of the anterior shoulder or placenta
  - occurs within first 24 h
- due to
  - overdistended uterus (polyhydramnios, multiple gestations, macrosomia)
  - uterine muscle exhaustion (prolonged or rapid labour, grand multiparity, oxytocin use, general anaesthetic)
  - uterine distortion (fibroids, placenta previa, placental abruption)
  - intra-amniotic infection (fever, prolonged ROM)

2. Tissue
- retained placental products (membranes, cotyledon or succenturiate lobe)
- retained blood clots in an atonic uterus
- gestational trophoblastic neoplasia
- abnormal placentation

3. Trauma
- laceration (vagina, cervix, uterus), episiotomy, hematoma (vaginal, vulvar, retroperitoneal), uterine rupture, uterine inversion

4. Thrombin
- coagulopathy (pre-existing or acquired)
  - most identified prior to delivery (low platelets increases risk)
  - includes hemophilia, DIC, Aspirin® use, ITP, TTP, vWD (most common)
  - therapeutic anti-coagulation

Uterine atony is the most common cause of PPH

DDx of Early PPH – 4 Ts
Tone (atony)
Tissue (retained placenta, clots)
Trauma (laceration, inversion)
Thrombin (coagulopathy)

DDx of Late PPH
Retained products
± endometritis
Sub-involution of uterus

Investigaions
- assess degree of blood loss and shock by clinical exam
- explore uterus and lower genital tract for evidence of tone, tissue, or trauma
- may be helpful to observe red-topped tube of blood – no clot in 7-10 min indicates coagulation problem

Management
- ABCs , call for help
- 2 large bore IVs, run crystalloids wide open
- CBC, coagulation profile, cross and type 4 units pRBCs
- treat underlying cause
- Foley catheter to empty bladder and monitor urine output

Medical Therapy
- oxytocin 5U IV bolus with delivery of anterior shoulder
  - 20-40 U/250 mL in crystalloid
  - in addition can give 10 U IM if CV collapse or IV access not possible
- methylergonavine maleate (ergotamine) 0.25 mg IM/IMM q5min up to 1.25 mg; can be given as IV bolus of 0.125 mg (may exacerbate HTN)
- carboprost (Hemabate®), a synthetic PGF-1α analog 250 μg IM/IMM q15min to max 2 mg (major prostaglandin side effects and contraindicated in cardiovascular, pulmonary, renal, and hepatic dysfunction)
- misoprostol 600-800 μg po/sl (faster) or pr/pv (side effect: pyrexia if >600 μg)
- tranexamic acid (Cyklokapron®) 1 g IV, an antifibrinolytic

Local Control
- bimanual compression: elevate the uterus and massage through patient's abdomen
- uterine packing (mesh with antibiotic treatment)
- Bakri Balloon for tamponade: may slow hemorrhage enough to allow time for correction of coagulopathy or for preparation of an OR

Surgical Therapy (Intractable PPH)
- D&C (beware of vigorous scraping which can lead to Asherman's syndrome)
- embolization of uterine artery or internal iliac artery by interventional radiologist
- laparotomy with bilateral ligation of uterine artery (may be effective), internal iliac artery (not proven), ovarian artery, or hypogastric artery
- hysterectomy last option with angiographic embolization if post-hysterectomy bleeding

Retained Placenta

Definition
- placenta undelivered after 30 min postpartum

Etiology
- placenta separated but not delivered
- abnormal placental implantation (placenta accreta, placenta increta, placenta percreta)

Risk Factors
- placenta previa, prior C/S, post-pregnancy curettage, prior manual placental removal, uterine infection

Clinical Features
- risk of postpartum hemorrhage and infection

Investigations
- explore uterus
- assess degree of blood loss

Management
- 2 large bore IVs, type and screen
- Brant maneuver (firm traction on umbilical cord with one hand applying suprapubic pressure to avoid uterine inversion by holding uterus in place)
- oxytocin 10 IU in 20 mL NS into umbilical vein
- manual removal if above fails
- D&C if required

Uterine Inversion

Definition
- inversion of the uterus through cervix ± vaginal introitus

Etiology/Epidemiology
- often iatrogenic (excess cord traction with fundal placenta)
- excessive use of uterine tocolytics
- more common in grand multiparous (lax uterine ligaments)
- 1/1,500-1/2,000 deliveries

Clinical Features
- can cause profound vasovagal response with bradycardia, vasodilation, and hypovolemic shock
- shock may be disproportionate to maternal blood loss

Management
- urgent management essential, call anesthesia
- ABCs: initiate IV crystalloids
- can use tocolytic drug (see 'Management' of *Preterm Labour*, OB16) or nitroglycerin IV to relax uterus and aid replacement
- replace uterus without removing placenta
- remove placenta manually and withdraw slowly
- IV oxytocin infusion (only after uterus replaced)
- re-explore uterus
- may require general anesthetic ± laparotomy

Postpartum Pyrexia

Definition
- fever >38°C on any 2 of the first 10 d postpartum, except the first day

Etiology
- endometritis
- wound infection (check C/S and episiotomy sites)
- mastitis/engorgement
- UTI
- atelectasis
- pneumonia
- DVT, pelvic thombophlebitis

Etiology of Postpartum Pyrexia

B-5W
Breast: engorgement, mastitis
Wind: atelectasis, pneumonia
Water: UTI
Wound: episiotomy, C/S site infection
Walking: DVT, thrombophlebitis
Womb: endometritis

Investigations
- detailed history and physical exam, relevant cultures
- for endometritis: blood and genital cultures

Treatment
- depends on etiology
  - infection: empiric antibiotics, adjust when sensitivities available
    - endometritis: clindamycin + gentamycin IV
    - mastitis: cloxacillin or cephalexin
    - wound infection: cephalexin, frequent sitz baths for episiotomy site infection
  - DVT: anticoagulants
- prophylaxis against post-C/S endometritis: begin antibiotic immediately after cord clamping and administer only 1-3 doses – cefazolin is most common choice

ENDOMETRITIS
- definition: infection of uterine myometrium and parametrium
- clinical features: fever, chills, abdominal pain, uterine tenderness, foul-smelling discharge, or lochia
- treatment: depends on infection severity; oral antibiotics if well, IV with hospitalization in moderate to severe cases

Risk Factors for Endometritis
C/S, intrapartum chorioamnionitis, prolonged labour, prolonged ROM, multiple vaginal examinations

VENOUS THROMBOEMBOLISM
- see *Venous Thromboembolism*, OB31

Mastitis

- definition: inflammation of mammary glands
- must rule out inflammatory carcinoma, as indicated
- differentiate from mammary duct ectasia: mammary duct(s) beneath nipple clogged and dilated ± ductal inflammation ± nipple discharge (thick, grey to green), often postmenopausal women

Table 24. Lactational vs. Non-Lactational Mastitis

	Lactational	Non-Lactational
Epidemiology	More common than non-lactational Often 2-3 wk postpartum	Periductal mastitis most common Mean age 32 yr
Etiology	*S. aureus*	May be sterile May be infected with *S. aureus* or other anaerobes Smoking is risk factor May be associated with mammary duct ectasia
Symptoms	Unilateral localized pain Tenderness Erythema	Subareolar pain May have subareolar mass Discharge (variable colour) Nipple inversion
Treatment	Heat or ice packs Continued nursing/pumping Antibiotics (cloxacillin/cephalexin) (Erythromycin if pen-allergic)	Broad-spectrum antibiotics and I&D Total duct excision (definitive)
Abscess	Fluctuant mass Purulent nipple discharge Fever, leukocytosis Discontinue nursing, IV antibiotics (nafcillin/oxacillin), I&D usually required	If mass does not resolve, FNA to exclude cancer and U/S to assess presence of abscess Treatment includes antibiotics, aspiration, or I&D (tends to recur) May develop mammary duct fistula A minority of non-lactational abscesses may occur peripherally in breast with no associated periductal mastitis (usually *S. aureus*)

Postpartum Mood Alterations

POSTPARTUM BLUES

- 40-80% of new mothers, onset day 3-10; extension of the "normal" hormonal changes and adjustment to a new baby
- self-limited, should resolve by 2 wk
- manifested by mood lability, depressed affect, increased sensitivity to criticism, tearfulness, fatigue, irritability, poor concentration/despondency, anxiety, insomnia

POSTPARTUM DEPRESSION

- definition: major depression occurring in a woman within 6 mo of childbirth (see Psychiatry, PS12)
- epidemiology: 10-15%, risk of recurrence 50%
- risk factors
  - personal or family history of depression (including PPD)
  - prenatal depression or anxiety
  - stressful life situation
  - poor support system
  - unwanted pregnancy
  - colicky or sick infant
- clinical features: suspect if the "blues" last beyond 2 wk, or if the symptoms in the first 2 wk are severe (e.g. extreme disinterest in the baby, suicidal or homicidal/infanticidal ideation)
- assessment: Edinburgh Postnatal Depression Scale or other
- treatment: antidepressants, psychotherapy, supportive care, ECT if refractory
- prognosis: interferes with bonding and attachment between mother and baby so it can have long-term effects

POSTPARTUM PSYCHOSIS

- definition: onset of psychotic symptoms over 24-72 h within first month postpartum, can present in the context of depression
- epidemiology: rare (0.2%)

Postpartum Care

Postpartum Office Visit at 6 Weeks

Care of Mother (The 10 Bs)

- **B**e careful: do not use douches or tampons for 4-6 wk post-delivery
- **B**e fit: encourage gradual increases in walking, Kegel exercises
- **B**irth control: assess for use of contraceptives; breastfeeding is NOT an effective method of birth control (see Gynecology, GY18, for more detail about different contraceptive options postpartum)
- **B**ladder: assess for urinary incontinence, maintain high fluid intake
- **B**lood pressure: especially if gestational HTN
- **B**lood tests: glucose, CBC (for anemia as sign of hematomas, retained placenta)
- **B**lues: (see *Postpartum Mood Alterations*)
- **B**owel: fluids and high-fibre foods, bulk laxatives; for hemorrhoids/perineal tenderness: pain meds, doughnut cushion, Sitz baths, ice compresses
- **B**reast and pelvic exam: watch for Staphylococcal or Streptococcal mastitis/abscess, ± Pap smear at 6 wk

The acronym "**BUBBLES**" for what to ask about when rounding on postpartum care. Modify this for C/S or vaginal delivery.

Baby care and breastfeeding (latch, amount)
Uterus – firm or boggy?
Bladder function – Voiding well? Dysuria?
Bowel function – Passing gas or stool? Constipated?
Lochia or discharge – Any blood?
Episiotomy/laceration/incision – Pain controlled?
Symptoms of VTE – Dyspnea? Calf pain?

Physiological Changes Postpartum

- uterus weight rapidly diminishes through catabolism, cervix loses its elasticity and regains firmness
  - should involute ~1 cm below umbilicus per day in first 4-5 d, reaches non-pregnant state in 4-6 wk postpartum
- ovulation resumes in ~45 d for non-lactating women and within 3-6 mo for lactating women
- lochia: normal vaginal discharge postpartum, uterine decidual tissue sloughing
  - decreases and changes in colour from red (lochia rubra; presence of erythrocytes, 3-4 d) → pale (lochia serosa) → white / yellow (lochia alba; residual leukorrhea) over 3-6 wk
  - foul-smelling lochia suggests endometritis

Breastfeeding Problems

- inadequate milk: consider domperidone
- breast engorgement: cool compress, manual expression/pumping
- nipple pain: clean milk off nipple after feeds, moisture cream, topical steroid if needed
- mastitis: treat promptly (see *Postpartum Pyrexia*, OB47)
- inverted nipples: makes feeding difficult
- maternal medications: may require pediatric consultation (see *Breastfeeding and Drugs*)

Bladder Dysfunction

- pelvic floor prolapse can occur after vaginal delivery
- stress or urge urinary incontinence common
- increased risk with instrumental delivery or prolonged second stage
- conservative management: pelvic floor retraining with Kegel exercises, vaginal cone, or pessaries, lifestyle modifications (e.g. limit fluid, caffeine intake)
- surgical management: minimally invasive procedures (tension-free vaginal tape, transobturator tape, midurethral sling)

Puerperal Pain

- "after pains" common in first 3 d due to uterine contractions; encourage simple analgesia
- ice packs can be used on perineum if painful
- encourage regular analgesia and stool softener

Breastfeeding and Drugs

Table 25. Drug Safety During Breastfeeding

Safe During Breastfeeding	Contraindicated When Breastfeeding
Analgesics (e.g. acetaminophen, NSAIDs)	Chloramphenicol (bone marrow suppression)
Anticoagulants (e.g. heparin)	Cyclophosphamide (immune system suppression)
Antidepressants (e.g. sertraline, fluoxetine, TCAs)	Sulphonamides (in G6PD deficiency, can lead to hemolysis)
Antiepileptics (e.g. phenytoin, carbamazepine, valproic acid)	Nitrofurantoin (in G6PD deficiency, can lead to hemolysis)
Antihistamines	Tetracycline
Antimicrobials (e.g. penicillins, aminoglycosides, cephalosporins)	Lithium
β-adrenergics (e.g. propanolol, labetalol)	Cocaine
Insulin	Phenindione
Steroids	Bromocriptine
OCP (low dose) – although may decrease breast milk production	Anti-neoplastics and immunosuppresants
	Psychotropic drugs (relative contraindication)

Breastfeeding: Contraindicated Drugs

BREAST
Bromocriptine/**B**enzodiazepines
Radioactive isotopes/**R**izatriptan
Ergotamine/**E**thosuximide
Amiodarone/**A**mphetamines
Stimulant laxatives/**S**ex hormones
Tetracycline/**T**retinoin

Common Medications

Table 26. Common Medications

Drug Name (Brand Name)	Dosing Schedule	Indications/Comments
betamethasone valerate (Celestone®)	12 mg IM q24h x 2 doses	Enhancement of fetal pulmonary maturity for PTL
carboprost (Hemabate®)	0.25 mg IM/IMM q15min; max 2 mg	Treatment of uterine atony
cefazolin	2 g IV then 1 g q8h	GBS prophylaxis (penicillin allergic and not at risk for anaphylaxis)
clindamycin	900 mg IV q8h	Used in endometritis
dexamethasone	6 mg IM q12h x 4 doses	Enhancement of fetal pulmonary maturity for PTL
dinoprostone (Cervidil®: PGE_2 impregnated thread)	10 mg PV (remove after 12 h) max 3 doses	Induction of labour Advantage: can remove if uterine hyperstimulation
doxylamine succinate (Diclectin®)	2 tabs qhs + 1 tab qAM + 1 tab qPM max 8 tabs/d	Each tablet contains 10 mg doxylamine succinate with vitamin B_6 Used for hyperemesis gravidarum
erythromycin	500 mg IV q6h	GBS prophylaxis (penicillin allergic and at risk for anaphylaxis)
folic acid	0.4-1 mg PO OD x 1-3 mo preconception and T1 5 mg PO OD with past Hx of NTD	Prevention of oNTD
methotrexate	50 mg/m^2 IM or 50 mg PO x 1 dose	For ectopic pregnancy or medical abortion
methylergonavine maleate (Ergotamine®)	0.25 mg IM/IMM q5min up to 1.25 mg or IV bolus 0.125 mg	Treatment of uterine atony
misoprostol (Cytotec®)	600-1000 μg PR x 1 dose 400 μg PO/SL x 1 dose or 800 μg PV x 1 dose, 3-7 d after methotrexate	For treatment of PPH For medical abortion/retained products of conception Also used for NSAID-induced ulcers (warn patients of contraindications)
oxytocin (Pitocin®)	0.5-2.0 mU/min IV, or 10 U/L NS increase by 1-2 mU/min q20-60min max 36-48 mU/min 10 U IM at delivery of anterior shoulder and of placenta 20 U/L NS or RL IV continuous infusion	Augmentation of labour (also induction of labour) Prevention of uterine atony Treatment of uterine atony
Penicillin G	5 million U IV then 2.5 million U IV q4h until delivery	GBS prophylaxis
PGE_2 gel (Prostin® gel)	0.5 mg PV q6-12h; max 3 doses	Induction of labour
Rh IgG (Rhogam®)	300 μg IM x 1 dose	Given to Rh negative women • Routinely at 28 wk GA • Within 72 h of birth of Rh+ fetus • Positive Kleihauer-Betke test • With any invasive procedure in pregnancy • Ectopic pregnancy • Antepartum hemorrhage • Miscarriage or therapeutic abortion (dose: 50 μg IM only)

Common Discharge Medications
Oxycodone IR 5-10 mg PO q4-6h PRN
Docusate sodium 100 mg PO bid

Misoprostol (Cytotec®) is also indicated to protect against NSAID-induced gastric ulcers in non-pregnant individuals. The use of misoprostol for cytoprotection is contraindicated in pregnancy; warn female patients of this contraindication

References

American College of Obstetricians and Gynecologists. Available from: www.acog.org.
The Society of Obstetricians and Gynaecologists of Canada. Available from: www.sogc.org.
Alfirevic Z, Devane D, Gyte GM. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. Cochrane DB Syst Rev 2013;5:CD006066.
Antenatal Corticosteroid Therapy for Fetal Maturation. SOGC clinical practice guidelines policy statement. December 1995:53.
Arsenault M, Lane CA. Guidelines for the management of nausea and vomiting in pregnancy. SOGC Clinical Practice Guidelines Committee Opinion 2002;120:1-7.
Banti S et al. From the third month of pregnancy to 1 year postpartum. Prevalence, incidence, recurrence, and new onset of depression. Results from the perinatal depression-research&screening unit study. Compr Psychiatry. 2011 Jul;52(4):343-51. Epub 2010 Sep 23.
Baskett T. Essential management of obstetric emergencies, 3rd ed. Bristol: Clinical Press, 1999.
Bastian LA, Piscitelli JT. Is this patient pregnant? Can you reliably rule in or rule out early pregnancy by clinical examination? JAMA 1997;278:586-591.
Berghella V, Odibo AO, Tolosa JE. Cerclage for prevention of preterm birth in women with a short cervix found on transvaginal examination: a randomized trial. Am J Obstet Gynecol 2004;191:1311-1317.
Berghella V, Rafael TJ, Szychowski JM, et al. Cerclage for short cervix on ultrasonography in women with singleton gestations and previous preterm birth: a meta-analysis. Obstet Gynecol 2011;117:663-671.
Blenning CE, Paladine H. An approach to the postpartum office visit. Am Fam Physician 2005;72(12):2491-2496.
Boucher M. Mode of delivery for pregnant women infected by the human immunodeficiency virus. Clinical Practice Guidelines Policy Statement 2001:101.
Boucher M, Gruslin A. The reproductive care of women living with hepatitis C infection. Clinical Practice Guidelines Policy Statement 2000:96.
Bricker L, Luckas M. Amniotomy alone for induction of labour. Cochrane DB Syst Rev 2000;(4):CD002862.
Carroli G, Mignini L. Episitotomy for vaginal birth. Cochrane DB Syst Rev 2009;1:CD000081.
Chamberlain G, Zander L. Induction. BMJ 1999;318:995-998.
Chamberlain G, Steer P. Labour in special circumstances. BMJ 1999;318:1124-1127.
Chamberlain G, Steer P. Obstetric emergencies. BMJ 1999;318:1342-1345.
Chamberlain G, Steer P. Operative delivery. BMJ 1999;318:1260-1264.
Chamberlain G, Steer P. Unusual presentations and positions and multiple pregnancy. BMJ 1999;318:1192-1194.
Chodirker BN, Cadrin C, Davies GAL, et al. Canadian guidelines for prenatal diagnosis. Techniques of prenatal diagnosis. SOGC Clinical Practice Guidelines 2001:105.
Chyu JK, Strassner HT. Prostaglandin E2 for cervical ripening: a randomized comparison of cervidil vs. prepidil. Am J Obstet Gynecol 1997;177:606-611.
Cohen-Kerem R, Nulman I, Abramow-Newerly M, et al. Diagnostic radiation in pregnancy: perception versus true risks. JOGC 2005;28:43-48.
Crane J. Induction of labour at term. SOGC Clinical Practice Guidelines 2001;107:1-12.
Farrell S, Chan MC, Schulz JA. Midurethral minimally invasive sling procedures for stress urinary incontinence. SOGC Clinical Practice Guidelines 2008;213:728-733.
Findley I, Chamberlain G. Relief of pain. BMJ 1999;318:927-930.
Ford HB, Schust DJ. Recurrent pregnancy loss: etiology, diagnosis, and therapy. Rev Obstet Gynecol 2009;2:76-83.
Gagnon A, Wilson R. Obstetrical complications associated with abnormal maternal serum markers analytes. SOGC Clinical Practice Guidelines 2008;216:918-932.
Gavin NI et al. Perinatal depression: a systematic review of prevalence and incidence.Obstet Gynecol. 2005 Nov;106(5 Pt 1):1071-83.
Goldenberg RL, Culhane JF, Iams JD, et al. Epidemiology and causes of preterm birth. Lancet 2008;371;75-84.
Guise JM, Berlin M, McDonagh M, et al. Safety of vaginal birth after cesarean: a systematic review. Obstet Gynec 2004;103:420-429.
Hajenius PJ, Mol F, Mol BW, et al. Interventions for tubal ectopic pregnancy. Cochrane DB Syst Rev 2007;1:CD000324.
Hamilton P. Care of the newborn in the delivery room. BMJ 1999;318:1403-1406.
Hennessey MH, Rayburn WF, Stewart JD, et al. Preeclampsia and induction of labour: a randomized comparision of prostaglandin E2 as an intracervical gel, with oxytocin immediately, or as a sustained-release vaginal insert. Am J Obstet Gynecol 1998;179:1204-1209.
Hod M, Bar J, Peled Y, et al. Antepartum management protocol. Timing and mode of delivery in gestational diabetes. Obstet Gynecol 2009;113:206-217.
Hodnett ED, Gates S, Hofmeyr GJ, et al. Continuous support for women during childbirth. Cochrane DB Syst Rev 2011;2:CD003766.
Howarth GR, Botha DJ. Amniotomy plus intravenous oxytocin for induction of labour. Cochrane DB Syst Rev 2001;3:CD003250.
Kelly AJ, Tan B. Intravenous oxytocin alone for cervical ripening and induction of labour. Cochrane DB Syst Rev 2001;3;CD003246.
Kent N. Prevention and treatment of venous thromboembolism (VTE) in obstetrics. SOGC Clinical Practice Guidelines 2000;95:2-8.
Koren G. Motherisk update: Caffeine during pregnancy? Canadian Family Phys 2000;46:801-803.
Kotaska A, Menticoglou S, Gagnon R, et. al. Vaginal delivery of breech presentation. SOGC Clinical Practice Guidelines 2009;226;557-566.
Langlois S, Ford J, Chitayat D. Carrier screening for thalassemia and hemoglobinopathies in Canada. J Obstet Gynaecol Can 2008;217;950-959.
Langlois S, Wilson R. Carrier screening for genetic disorders in individuals of Ashkenazi Jewish descent. SOGC Clinical Practice Guidelines 2006;177;324-332.
Ling F, Duff P. Obstetrics and Gynecology, Principles for practice. USA: McGraw-Hill Companies, 2002.
Liston R, Sawchuck D, Young D. Fetal health surveillance: antepartum and intrapartum consensus guideline. SOGC Clinical Practice Guidelines 2007;197:S1-60.
Lowder JL, Burrows LJ, Krohn MA, et al. Risk factors for primary and subsequent anal sphincter lacerations: a comparison of cohorts by parity and prior mode of delivery. Am J Obstet Gynecol 2007;196:344.e1-5.
Luckas M, Bricker L. Intravenous prostaglandin for induction of labour. Cochrane DB Syst Rev 2000;4:CD002864.
Mackeen AD, Seibel-Seamon J, Muhammad J, et al. Tocolytics for preterm premature rupture of membranes. Cochrane DB Syst Rev 2014;2:CD007062.
Magee LA, Helewa M, Moutquin J-M, et al. Diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. J Obstet Gynaecol Can 2008;30:S1-48.
Menezes EV, Yakoob MY, Soomro T, et al. Reducing stillbirths: prevention and management of medical disorders and infections during pregnancy. BMC Pregnancy Childbirth 2009;9(Suppl 1):S4.
Menticoglou S, Gagnon R, Kotaska A. Vaginal delivery of breech presentation. SOGC Clinical Practice Guidelines 2009;226;557-566.
Ministry of Health and Long Term Care and Canadian Medical Association. Antenatal record 1. Ontario.
Ministry of Health and Long Term Care and Canadian Medical Association. Antenatal record 2. Ontario.
Money D, Dobson S. The prevention of early-onset group B streptococcal disease. SOGC Clinical Practice Guidelines 2004;149:826-832.
Morgan S, Koren G. Is caffeine consumption safe during pregnancy? Can Fam Physician 2013;59(4):361-362.
Mottola MF, Wolfe LA, MacKinnon K, et al. Exercise in pregnancy and the postpartum period. SOGC Clinical Practice Guidelines 2003;129:1-7.
Mount Sinai Hospital. First trimester combined screening program. 2001.
Nicolaides KH, Syngelaki A, Ashoor G, et al. Noninvasive prenatal testing for fetal trisomies in a routinely screened first-trimester population. Am J Obstet Gynecol 2012;207(5):374.
North York General Hospital Genetics Program. Integrated prenatal screening. 1999.
Ottinyer WS, Menara MK, Brost BC. A randomized control trial of prostaglandin E2 intracervical gel and a slow release vaginal pessary for preinduction cervical ripening. Am J Obstet Gynecol 1998;179:349-353.
Ross S, Robert M. Conservative management of urinary incontinence. SOGC Practice Guidelines 2006;186:1113-1118.
Schrag SJ, Zell ER, Lynfield R, et al. A population-based comparison of strategies to prevent early-onset group B streptococcal disease in neonates. NEJM 2002;347:233-239.
Schuurmans N, Gagne G, Ezzat A, et al. Healthy beginnings: guidelines for care during pregnancy and childbirth. Clinical Practice Guidelines Policy Statement 1998:71:1-65.
Steer P, Flint C. Physiology and management of normal labour. BMJ 1999;318:793-796.
Steer P, Flint C. Preterm labour and premature rupture of membranes. BMJ 1999;318:1059-1062.
Stewart D. A broader context for maternal mortality. CMAJ 2006;74;302-303.
Stewart JD, Rayburn WF, Farmer KC, et al. Effectiveness of prostaglandin E2 intracervical gel (prepidil) with immediate oxytocin vs. vaginal insert (cervidil) for induction of labour. Am J Obstet Gynecol 1998;179:1175-1180.
Summers A, Langlois S, Wyatt P, et al. Prenatal screening for fetal aneuploidy. SOGC Clinical Practice Guidelines 2007;187:146-161.
Thompson D, Berger H, Feig D, et al. Diabetes and Pregnancy. Can J Diabetes 2013;37:S168-S183.
Van den Hof M, Crane J. Ultrasound cervical assessment in predicting preterm birth. SOGC Clinical Practice Guidelines 2001;102:1-4.
Verani JR, McGee L, Schrag SJ. Prevention of perinatal group B streptococcal disease. MMWR 2010;59(No.RR-10):1-32.
Zander L, Chamberlain G. Place of birth. BMJ 1999;318:721-723.
Zhang J, Bowes WA Jr, Fortney JA. Efficacy of external cephalic version: a review. Obstet Gynecol 1993;82(2):306-312.