NOTES
FEMALE REPRODUCTIVE SYSTEM

ANATOMY & PHYSIOLOGY OF THE FEMALE REPRODUCTIVE SYSTEM

osms.it/female-reproductive-system

EXTERNAL ORGANS

- Labia minora, labia majora, clitoris (erectile tissue), mons pubis
  - *Vulvar vestibule*: space between labia minora; includes vaginal, urethral opening

INTERNAL ORGANS

Ovaries (female gonads)

- Epithelial, follicular, granulosa, theca, oocyte cells
- Secrete estrogen, progesterone
- Located superior, lateral to uterus
- Held in place by ovarian, broad, suspensory ligaments
  - Suspensory ligaments contain ovarian artery, vein, nerve plexus
- Made up of outer cortex, inner medulla
  - Cortex contains ovarian follicles (oocytes surrounded by granulosa cells); medulla contains blood vessels, nerves

Fallopian tubes (uterine tubes)

- Two tubes, each associated with one ovary, on side of uterus
- Flattened mesothelial, epithelial, secretory, intercalary cells
- Fimbriae around ovary → infundibulum → ampulla (where fertilization most commonly occurs) → isthmus region opens into uterine cavity
- Covered by peritoneum, supported by mesosalpinx
- Lined with smooth muscle, cilia to sweep zygote towards uterus; inner mucosa provides nutrients for oocyte

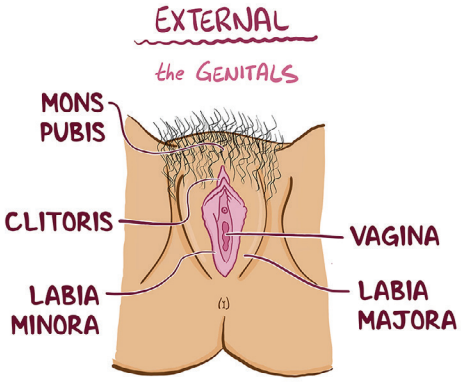


Figure 8.1 External organs of the female reproductive system.

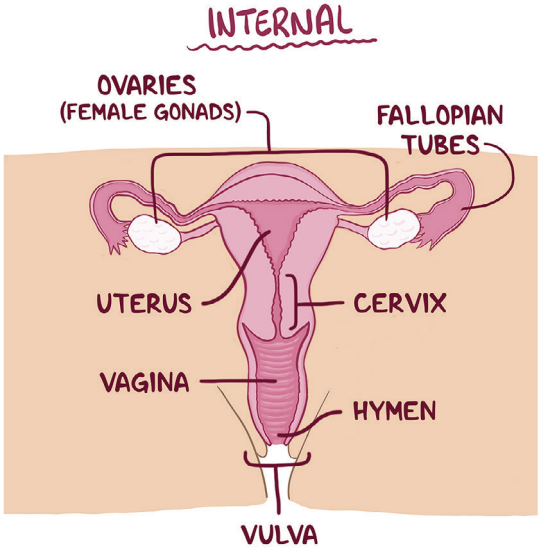


Figure 8.2 External organs of the female reproductive system.

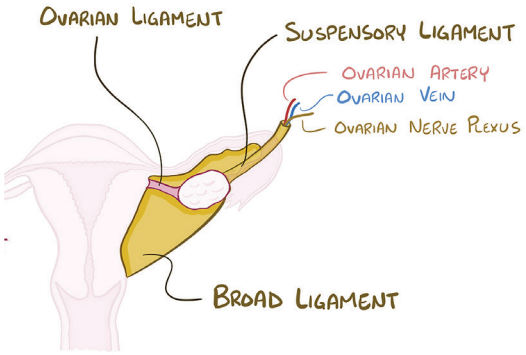


Figure 8.3 The locations of the ovarian, suspensory, and broad ligaments.

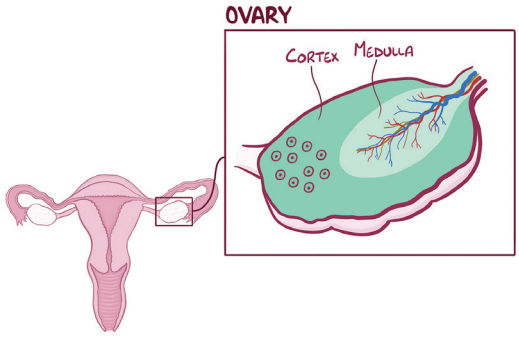


Figure 8.4 Outer cortex of ovary containing follicles and inner medulla containing blood vessels, nerves.

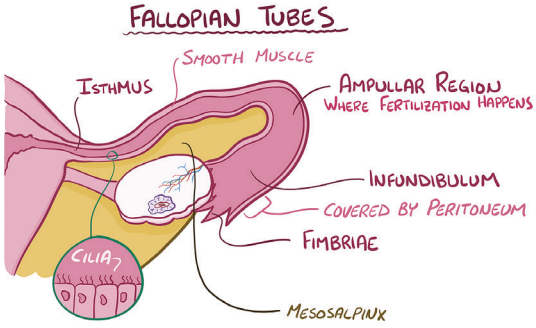


Figure 8.5 Features of the fallopian tubes.

Uterus

- Located posterior to bladder, anterior to rectum
- Fundus (top) → uterine body → uterine isthmus → cervix (neck of uterus)
  - *Cervical opening to vagina*: external os; thins, dilates during childbirth
  - *Cervical opening into uterine cavity*: internal os
- Anchored to sacrum (uterosacral ligaments) → anterior body wall (round ligaments)
- Supported by cardinal ligaments, mesometrium
- Three layers of uterine wall
  - Perimetrium, myometrium (smooth muscle), endometrium (highly vascular mucosal layer)

Vagina

- Extends from uterus, opens into vulva (covered by hymen in childhood)
- Outer muscular wall containing rugae; inner mucous membrane of stratified squamous epithelium
- Fornix (superior, domed area) connects to sides of cervix

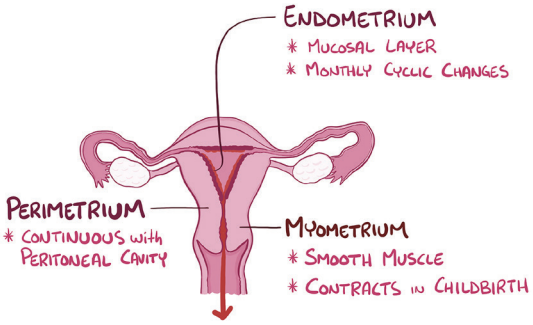


Figure 8.6 The three layers of the uterine wall. External to internal: perimetrium → myometrium → endometrium.

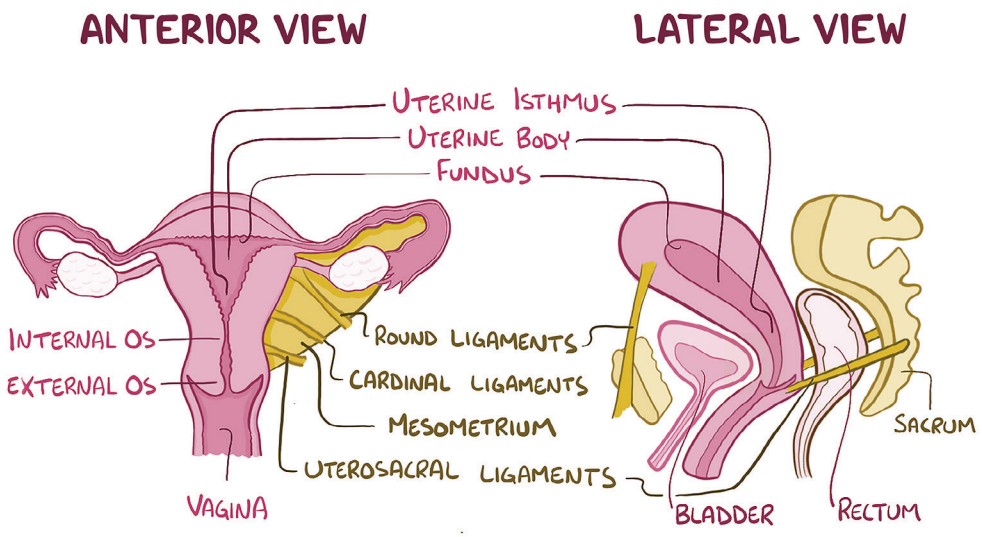


Figure 8.7 Anterior view of the uterus and lateral view of the uterus in relationship to surrounding structures.

OOGENESIS

Fetal development

- Oogonia (primordial oocyte cell) undergo mitotic division → ↑ oogonia (diploid cells)
- 7 months
  - Oogonia begin meiotic division, become primary oocytes (diploid cells)

Follicular development

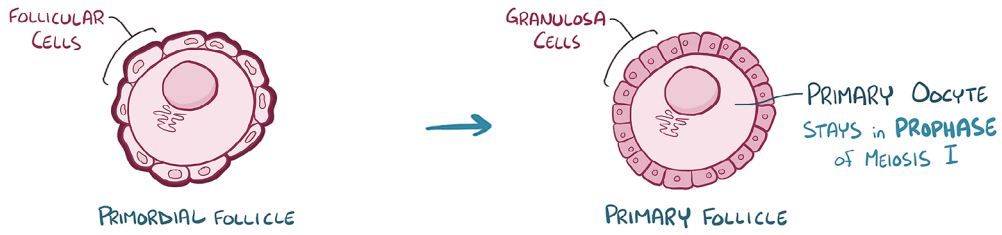
- Infancy to puberty
  - Primary oocyte surrounded by granulosa cells form primary (primordial) follicle
- Menstrual cycle (approx. every 28 days)
  - Primary follicle → secondary follicle → tertiary (Graafian) follicle
- Antrum (fluid-filled cavity) forms in Graafian follicles; granulosa cells secrete nourishing fluid for primary oocyte
- Theca cells produce androstenedione (sex hormone precursor) → converted into estradiol in granulosa cells
- *Follicular phase of menstrual cycle*: Graafian follicles grow
  - Follicle with most follicle-stimulating hormone (FSH) receptors becomes dominant follicle; primary oocyte → meiosis I completed, secondary oocyte (haploid cell with 23 chromosomes) formed
- *Ovulation*: dominant follicle ruptures → secondary oocyte released → peritoneal cavity → pulled inside fallopian tube
- *Luteal phase*: follicle remains → corpus luteum (luteinized granulosa, theca cells)
  - Luteinized granulosa cells secrete inhibin → ↓ FSH → ↓ estrogen → ↓ luteinizing hormone (LH)
  - *Luteinized theca cells*: ↑ progesterone → dominant hormone

Fertilization

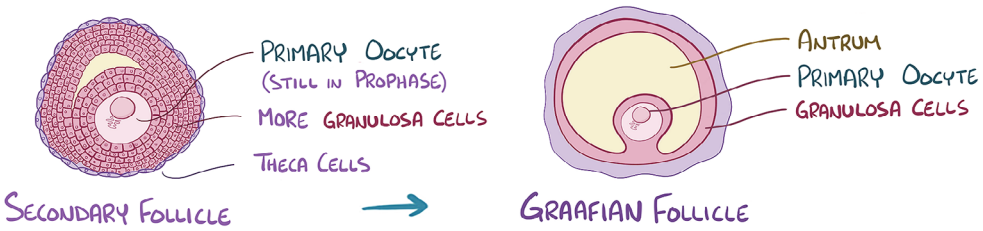
- If fertilization occurs → oocyte becomes mature ovum → progesterone produced until placenta forms
- If fertilization does not occur → corpus luteum → corpus albicans

FOLLICULAR DEVELOPMENT

FIRST STAGE: INFANCY to PUBERTY

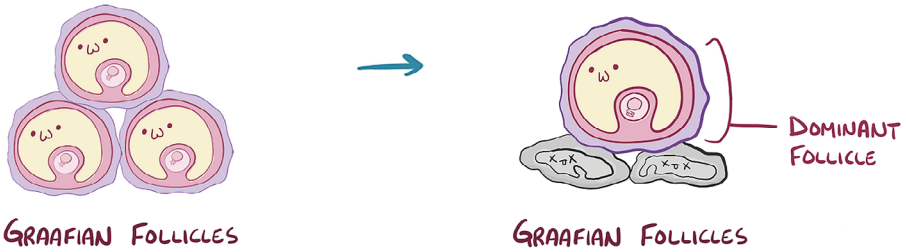


SECOND STAGE: a FEW PRIMARY FOLLICLES ENTER EACH MENSTRUAL CYCLE

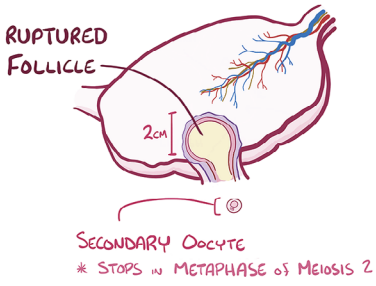


THIRD STAGE: BEGINS WHEN GRAAFIAN FOLLICLES ARE READY

OCCURS DURING FOLLICULAR PHASE



OVULATION



LUTEAL PHASE (WEEKS 3 & 4)

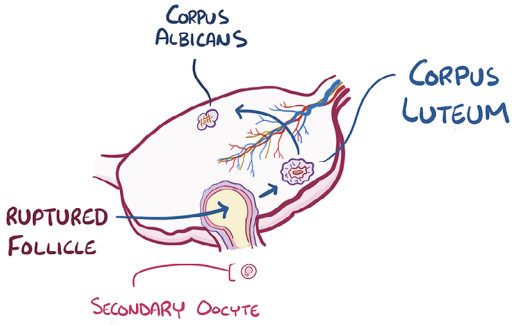


Figure 8.8 Stages of follicular development. *Stage one:* primordial follicles → primary follicles, meaning that the follicular cells surrounding the primary oocyte develop into granulosa cells. *Stage two:* primary follicles → secondary follicles → teritary (Graafian) follicles. This stage results in a few fast-growing Graafian follicles. *Stage three:* dominant follicle is established. *Ovulation:* dominant follicle ruptures, releases secondary oocyte into fallopian tube. The secondary oocyte stops in metaphase of meiosis II. *Luteal phase:* weeks 3 to 4 of menstrual cycle. The remains of the follicle turn into the corpus luteum. If fertilization occurs, the corpus luteum keeps making progesterone until the placenta forms. If not, the corpus luteum stops making hormones after about ten days, becomes fibrotic → corpus albicans.

OXYTOCIN & PROLACTIN

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- Peptide hormones involved in production, release of milk → stored in Herring bodies → released into blood → target tissues (e.g. breasts, uterus)

OXYTOCIN

- Essential for progression of labor, control of postpartum bleeding, return of uterus to pre-pregnancy state (involution)
- Synthesized, secreted by hypothalamus → travels down axons to posterior pituitary

PROLACTIN (PL)

- Synthesized by lactotrophs in anterior pituitary → target tissue (breasts)
- Synthesis inhibited by dopamine during non-pregnant/non-breastfeeding state

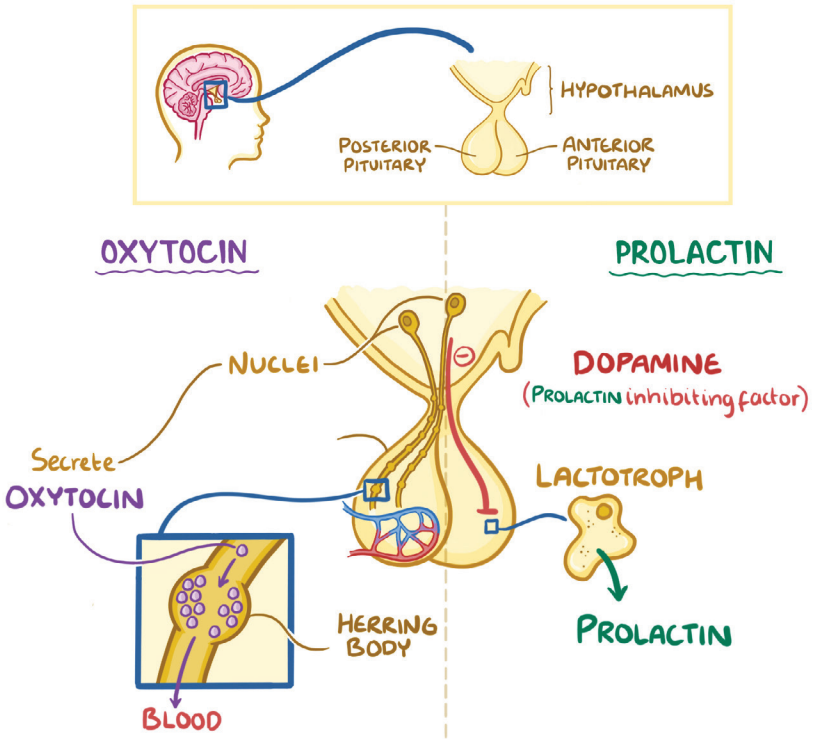

Figure 8.9 Synthesis and secretion of oxytocin and prolactin.

FUNCTIONS DURING LACTATION

- *Neuroendocrine reflex:* suckling by infant at breast → stimulates mechanoreceptors in nipple, areola → action potential travels up spinal cord to hypothalamus
- First, burst of oxytocin released from posterior pituitary → enters bloodstream → breasts, uterus
  - Myoepithelial cells surrounding alveoli in breasts contract → milk ejection from alveolus (let-down reflex)
  - Stimulates contractile activity of uterine myometrium → ↓ postpartum bleeding; promotes uterine involution
- Second, thyrotropin-releasing hormone (TRH) from hypothalamus → PL released from anterior pituitary → enters bloodstream → breasts → ↑ milk production, secretion by alveolar epithelial cells
- ↑ PL inhibits release of GnRH from hypothalamus → ↓ LH, FSH from anterior pituitary → ↓ development of ovarian follicles, ovulation, menstrual periods

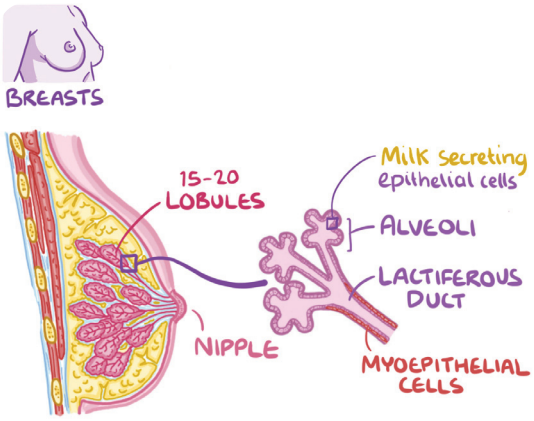

Figure 8.10 Anatomy of the breast.

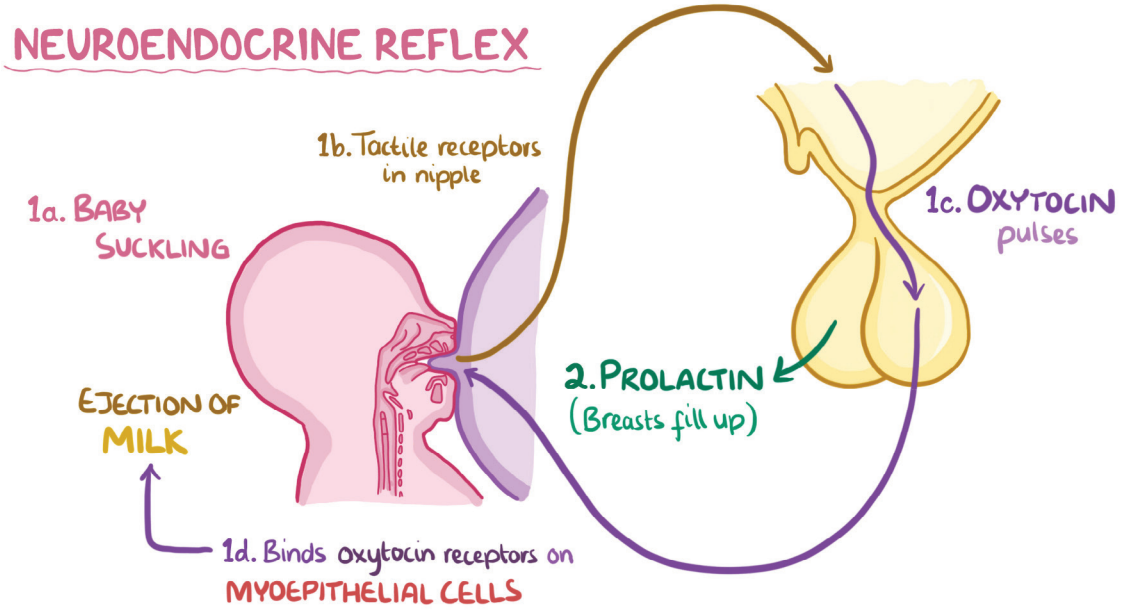

Figure 8.11 Illustration of the neuroendocrine reflex. In response to the suckling of a baby, oxytocin released from the posterior pituitary stimulates ejection of milk, and prolactin released from the anterior pituitary increases milk production.

FUNCTIONS DURING & AFTER LABOR

- Oxytocin (powerful uterine muscle stimulant) produced during pregnancy, does not stimulate uterine contractions due to
  - Rapid degradation by placental oxytocinase
  - Progesterone-induced inhibition of oxytocin receptors on myometrium
- Estrogen-induced oxytocin receptor expression + ↑ myometrial sensitivity to oxytocin promotes uterine contractions during labor
  - *Positive feedback loop*: ↑ uterine contractions → fetal head pushes against cervix → neural signal travels to spinal cord → hypothalamus → ↑ oxytocin release from posterior pituitary → ↑ uterine contractions → cycle continues until delivery (baby, placenta)
- After labor, milder contractions continue
  - Clamp down on placental arteries at placental attachment site → ↓ bleeding
  - Gradually ↓ size of uterus (involution)
  - Additional oxytocin released during breastfeeding → speeds involution

MENSTRUAL CYCLE

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- *Menstruation (menses)*: shedding of uterine functional endometrium
- Occurs approx. every 28 days

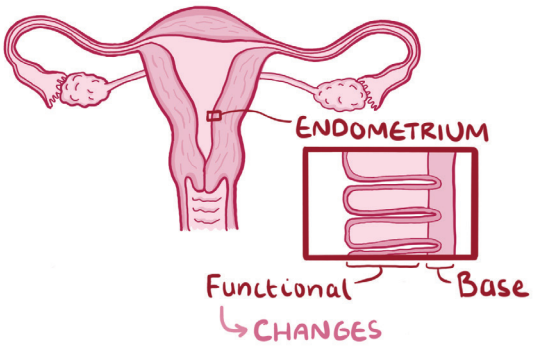

Figure 8.12 The uterine endometrium consists of a thin base layer and a functional layer. The functional layer is subject to the changes (thickening and shedding) that occur during the menstrual cycle.

FOLLICULAR PHASE

- *Ovulation (days 1–14)*: maturing follicles, proliferation of uterine mucosa, dominated by estrogen

Day 1

- Hypothalamus releases gonadotropin-releasing hormone (GnRH) → anterior pituitary releases FSH, LH → one oocyte dominates → develops within primary follicle
- *Primary (primordial) follicle*: oocyte surrounded by single layer of granulosa cells (nourish oocyte)

Days 1–13

- Granulosa cells proliferate → follicle grows → develops outer layer of cells (theca layer) → respond to LH by producing estrogen → mature follicle
  - Estrogen acts on uterine endometrium to prepare for fertilized egg → initiates uterine proliferative phase → endometrial lining grows
  - Estrogen also feeds back to hypothalamus, pituitary → turns off GnRH, FSH, LH

Day 14

- Brief LH surge stimulates ovulation → follicle ruptures → oocyte ejected out of follicle

LUTEAL PHASE

- After ovulation, empty follicle collapses → turns into corpus luteum → produces progesterone (approx. 14 days)
  - Endometrium becomes highly vascularized, glycogen-filled tissue (secretory phase)

Days 15–24

- Egg travels through fallopian tube

Day 25

- If fertilization does not occur → corpus luteum undergoes apoptosis → progesterone levels fall
- If fertilization does occur → embryonic tissue secretes human chorionic gonadotropin (hCG) → signals corpus luteum to continue production of estrogen, progesterone to support pregnancy

PREGNANCY

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- Obstetric history (GTPAL)
  - *G (gravida)*: number of pregnancies, regardless of duration (including current pregnancy)
  - *T*: number of term infants born
  - *P*: number of preterm infants born
  - *A*: number of spontaneous/induced abortions
  - *L*: number of currently living children
  - Example: G3P1202 (3 pregnancies, 1 term birth, 2 preterm births, 0 abortions, 2 living children)
- Pregnancy lasts approx. 280 days (40 weeks); divided into three trimesters

SIGNS & SYMPTOMS

Presumptive

- Amenorrhea; breast fullness, tenderness; nausea/vomiting ("morning sickness"); urinary frequency; fatigue; fetal movement (16–20 weeks of gestation)

Probable

- Uterine enlargement; softening of uterine isthmus (Hegar sign); vaginal, cervical purplish-blue discoloration (Chadwick sign); positive urine/serum hCG

Positive

- Auscultation of fetal heart tones (7–8 weeks of gestation); "quickening" (fetal movements); fetal sac visualized by ultrasound (5–6 weeks); fetal cardiac activity (6–8 weeks)

ESTIMATED DATE OF DELIVERY (EDD)

- Calculated from last menstrual period (LMP) to estimated date of delivery (EDD)
- *Naegele's rule*: add 7 days to 1st day of LMP, subtract 3 months, add 7 days, add 1 year
- Ultrasonic examination
  - Measurement of crown-to-rump length in first trimester
- Measurement of fundal height estimates pregnancy progression
  - *Symphysis*: 12–14 weeks
  - *Umbilicus*: 20 weeks
  - Rises above umbilicus 1 cm/week until 36 weeks

PHYSIOLOGICAL CHANGES IN THE REPRODUCTIVE SYSTEM

Uterus

- ↑ size, capacity due to hypertrophy, hyperplasia, mechanical stretching
- 20 times larger
- ↑ strength, distensibility, contractile proteins, number of mitochondria
- ↑ volume capacity (10 mL–5 L)
- Softening of uterine isthmus (Hegar's sign)

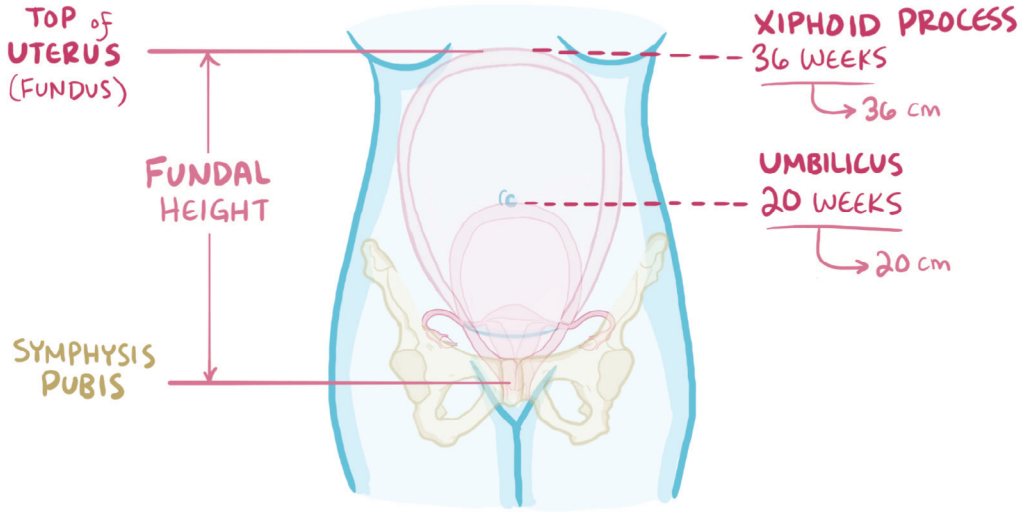

Figure 8.13 Fundal height = distance from symphysis pubis to top of uterus (fundus). Fundal height is a good estimate of gestational age.

Cervix

- Formation of mucus plug; seals endocervical canal
- ↑ vascularity → purplish-blue color
- Mild softening due to edema, hyperplasia (Goodell's sign); ↑ softening in third trimester

Placenta

- Develops where embryo attaches to uterine wall
- Expands to cover 50% internal uterine surface
- Functions as maternal-fetal organ for metabolic, nutrient exchange
- Secretes estrogen, progesterone, relaxin, hCG

Vagina

- ↑ vascularity → bluish-purple color
- Loosening of connective tissue → ↑ distensibility
- Leukorrhea
  - pH of 3.5–6.0 → protects against bacterial infections

Breasts

- ↑ size, weight, nodularity, blood flow, vascular prominence
- Areola, nipples are a darker pigmentation due to ↑ melanocyte activity
- ↑ activity of Montgomery's tubercles (sebaceous glands)
- Progesterone
  - ↑ alveolar-lobular development; prevents milk production during pregnancy (inhibits prolactin)
- Estrogen
  - ↑ growth of lactiferous ducts
- Secretion of colostrum begins week 16

PHYSIOLOGICAL CHANGES IN OTHER BODY SYSTEMS

Cardiovascular

- Mild hypertrophy
- S2, S3 more easily auscultated, split exaggerated
- Heart displaced upward, forward, slightly to left
- ↑ heart rate by 15–20 beats/minute
- Stroke volume ↑ 30%, cardiac output (CO) ↑ 30-50% (by term); ↓ blood pressure (BP) despite ↑ CO due to progesterone-induced vasodilation; BP = CO × systemic vascular resistance (SVR)
- Supine hypotensive syndrome caused by gravid uterus pressing on inferior vena cava (left lateral recumbent position optimal for CO, uterine perfusion)
- Gravid uterus elevates pressure veins draining legs, pelvic organs → slowed venous return, dependent edema, varicose veins, hemorrhoids

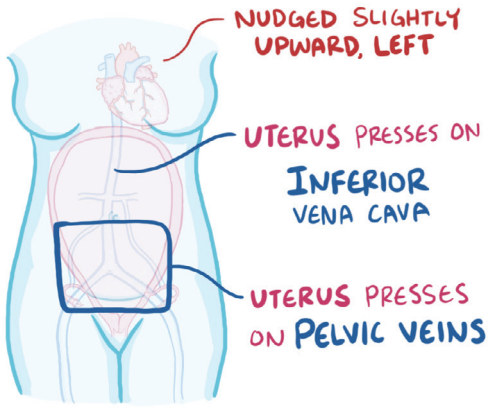

Figure 8.14 Cardiovascular changes during pregnancy. When lying down, uterus presses on inferior vena cava → less blood to right atrium → hypotension. The uterus also presses on pelvic veins → varicose veins, swelling in lower legs, ankles.

Hematologic

- ↑ blood volume (approx. 1500 mL)
  - Related to sodium, water retention due to changes in osmoregulation, secretion of vasopressin by anterior pituitary, renin-angiotensin-aldosterone system (RAAS)
- ↑ total red blood cell (RBC) volume (approx. 30%), with iron supplementation
  - ↑ volume, oxygen-carrying capacity needed for ↑ basal metabolic rate (BMR), needs of uterine-placental unit (offsets blood loss at delivery)
  - Plasma > RBC volume → hemodilution, ↓ hematocrit (physiologic anemia)
- ↑ white blood cell (WBC) count (approx. 5,000–12,000/mm3)
- ↑ clotting factors (fibrin, fibrinogen): hypercoagulable state of pregnancy

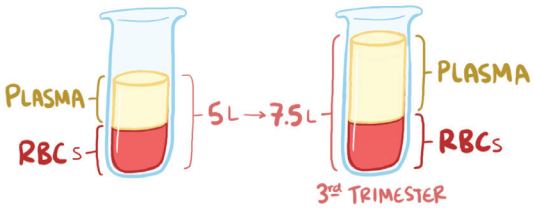

Figure 8.15 Pregnancy is a high volume state. Plasma volume ↑ > RBC volume ↑ → ↓ hematocrit (physiologic anemia).

Respiratory

- ↑ oxygen consumption, subcostal angle, anteroposterior diameter, tidal volume (30–50%), minute ventilatory volume, minute oxygen uptake
- Gravid uterus places upward pressure on diaphragm → elevates approx. 4 cm
- Hyperventilation → mild respiratory alkalosis (renal compensation → maternal blood pH 7.40–7.45)
- Nasal congestion, epistaxis due to estrogen-induced edema

Gastrointestinal

- Gums bleed easily due to estrogen-induced hyperemia, friability
- Progesterone-induced smooth muscle relaxation, delayed gastric emptying, ↓ peristalsis → nausea, vomiting (AKA "morning sickness"); constipation; heartburn (pyrosis), esophageal reflux; intrahepatic cholestasis of pregnancy due to ↓ gallbladder emptying time → ↑ risk of cholelithias
- ↑ saliva production (ptyalism)

Urinary & renal

- Bladder
  - *First trimester*: gravid uterus presses on bladder → urinary frequency, nocturia, stress incontinence
  - *Second trimester*: uterus occupies abdominal space → ↓ urinary frequency
  - *Third trimester*: presenting part descends into pelvis → urinary frequency, nocturia, stress incontinence
- ↑ glomerular filtration rate (GFR)
  - 40–50% by second trimester; ↑ urinary output (25%)
- ↑ size of kidneys (1–1.5 cm)
- Dilation of urinary collecting system → physiologic hydronephrosis
- Urinalysis
  - Glycosuria (due to ↑ glucose load), ↑ protein excretion (due to altered proximal tubule function + ↑ GFR)

Integumentary

- Hyperpigmentation (due to estrogen, ↑ melanocyte activity) → melasma (chloasma) brownish "mask of pregnancy"; linea nigra formation on abdomen; darkening of

nipples, areolae, vulva
- ↑ cutaneous blood flow → ↑ heat dissipation → pregnancy "glow"
- ↓ connective tissue strength secondary to ↑ adrenal steroid levels → stretch marks (striae gravidarum) in breasts, abdomen, thighs, inguinal area
- Estrogen-induced vascular permeability → spider nevi, angiomas, palmar erythema

Musculoskeletal
- Abdominal distension + shift in center of gravity → lordosis
- Enlarging uterus → separation of abdominal rectus muscles (diastasis recti)
- ↑ progesterone, relaxin → ↑ joint mobility, "waddling" gait
  - Widening of symphysis pubis
  - Facilitates accommodation of fetus into pelvis
- High bone turnover, remodeling

Endocrine
- ↑ size of pituitary gland; mostly due to proliferation of lactotroph cells
  - ↑ intrasellar pressure → ↑ risk of postpartum infarction (Sheehan syndrome) in setting of postpartum hemorrhage
- ↑ parathyroid hormone (meets calcium need of developing fetal skeleton)
- Physiologic hypercortisolism
  - ↑ need for estrogen, cortisol → ↑ glucocorticoids from adrenal glands → supports fetal somatic, reproductive growth
- "Diabetogenic state" of pregnancy
  - ↑ need for glucose, insulin production → hypertrophy, hyperplasia of pancreatic beta cells
- ↓ thyroid-stimulating hormone (TSH); thyroid gland enlarges; ↑ total T3, T4
- Reproductive hormones
  - hCG from placenta; estrogen, progesterone from corpus luteum (first, second trimesters), placenta (second, third trimesters)
  - Suppressed FSH, LH due to feedback from estrogen, progesterone, inhibin
  - ↓ oxytocin levels throughout pregnancy → ↑ labor onset → ↑↑ second stage of labor

NUTRITIONAL NEEDS
- Recommendation of additional 300 kcal/day, weight gain of 25–35 pounds (11.5–16 kg)
  - *11 lb (5 kg)*: placenta, amniotic fluid, fetus
  - *2 lb (0.9 kg)*: uterus
  - *4 lb (1.8 kg)*: ↑ blood volume
  - *3 lb (1.4 kg)*: breast tissue
  - *5–10 lb (2.3–4.5 kg)*: maternal reserves
- 600 mcg folic acid/day → RBC synthesis, placental/fetal growth, ↓ risk of neural tube defects
- 1,000–1,300 mg calcium/day supports pregnancy, lactation
- 60g protein daily supports tissue growth
- 27 mg iron/day supports ↑ RBCs

LABOR

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- *Labor (parturition)*: uterine contractions → cervical changes → delivery of baby, placenta
- Begins at term (37–42 weeks of gestation)
- Duration of three stages varies with gravidity (nulliparas typically longer than multiparas)

PREMONITORY SIGNS
- Cervical changes
  - Remodeling of cervix by enzymatic collagen dissolution, ↑ water content → softening, ↑ distensibility
- Cervical softening → expulsion of mucus plug → "bloody show" (pink-tinged mucus)

- Spontaneous rupture of amniotic membranes (ROM)

False labor

- AKA Braxton-Hicks contractions
- *True labor*: regular, increase in frequency, duration, intensity; produce cervical changes (e.g. dilation/opening up, effacement/getting thinner); pain begins in lower back, radiates to abdomen, not relieved by ambulation
- *False labor*: irregular, intermittent contractions; no cervical changes; pain in abdomen; walking may decrease pain

FIRST STAGE OF LABOR

Early/latent

- 8–12 hours
- Mild contractions every 5–30 minutes
- Duration 30 seconds each
- Gradually increase in frequency, intensity, duration
- Cervical dilation 0–3 cm
- Effacement 0–30%
- Spontaneous ROM

Active phase

- 3–5 hours
- Contractions every 3–5 minutes
- Duration ≥ 1 minute
- Cervical dilation 3–7 cm
- Effacement 80%
- Progressive fetal descent

Transition phase

- 30 minutes–2 hours
- Intense contractions every 1.5–2 minutes
- Duration 60–90 seconds
- Cervical dilation 7–10cm
- Effacement 100%

SECOND STAGE

- AKA pushing stage
- Begins with full dilation
- Navigation through maternal pelvis dictated by 3 Ps
  - Power, passenger, passage

Power

- Frequency, duration, intensity of uterine contractions
- Physiology of contractions
  - Stimulation of uterine myometrium
  - Alpha-receptors stimulate uterine contractions
  - Numerous oxytocin receptors, mostly on uterine fundus
- Contraction steps
  - Wave begins in fundus, proceeds downward to rest of uterus → muscle shortens in response to stimulus → increment (build up) → acme (peak) → decrement (gradual letting up) → relaxation → fetal descent, cervical effacement, dilation → amount of pressure exerted by uterine contractions (intrauterine pressure) measured in millimeters of mercury (mm Hg)

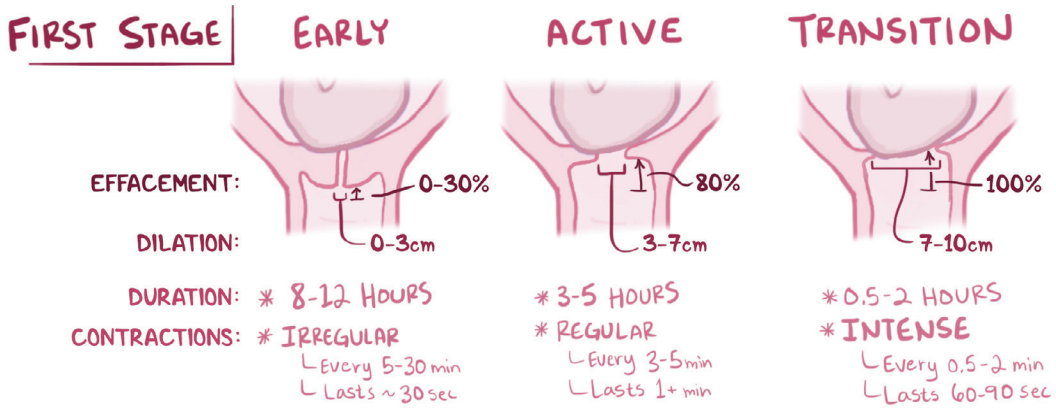

Figure 8.16 Features of the phases of the first stage of labor.

Passenger

- Fetal size
  - Fetal head most critical; cephalopelvic disproportion → labor dystocia (difficult/obstructed)
  - Macrosomia (birth weight ≥ 90th percentile for gestational age/> 4500 g) associated with shoulder dystocia (fetal shoulder unable to pass below maternal pubic symphysis), birth injuries
- *Fetal attitude*: relationship of fetal parts to one another
  - Full flexion (chin on chest; rounded back with flexed arms, legs); smallest diameter of head (suboccipitobregmatic diameter) presents at pelvic inlet
- *Fetal lie*: relationship of fetal cephalocaudal axis (spinal column) to maternal cephalocaudal axis
  - *Longitudinal (ideal)*: fetal spine lies along maternal
  - *Transverse*: fetal spine perpendicular to maternal
  - *Oblique*: fetus at slight angle
- *Fetal presentation*: fetal/presenting part enters pelvic inlet first
- *Cephalic*: head first
  - *Vertex (most common)*: optimal for easy delivery; head completely flexed onto chest → occiput (part of fetal skull covered by occipital bone) is presenting
  - *Brow*: fetal head partially extended; sinciput (part of fetal skull covered by frontal bone, anterior fontanelle to orbital ridge) presenting part
  - *Face*: fetal head hyperextended; fetal face from forehead to chin presenting part
- *Breech*: head up; bottom, feet, knees present first
  - *Frank breech*: hips flexed, knees extended; bottom presents
  - *Complete breech*: hips, knees flexed; bottom presents
  - *Incomplete breech*: one/both hips not completely flexed; feet present
  - *Shoulder*: transverse lie; shoulders present first

Passage

- Route through bony pelvis
- Size, type of pelvis
  - *Gynecoid*: rounded pelvic inlet, midpelvis, outlet capacity adequate; optimal for vaginal delivery
  - *Android*: heart-shaped pelvic inlet; ↓ midpelvis diameters, outlet capacity; associated with labor dystocia
  - *Anthropoid*: oval-shaped pelvic inlet; midpelvis diameters, outlet capacity adequate; favorable for vaginal delivery
  - *Platypelloid*: oval-shaped pelvic inlet, ↓ midpelvis diameters, outlet capacity adequate; not favorable for vaginal delivery
- Cardinal movements (mechanisms of labor)
  - *Descent*:: presenting part reaches pelvic inlet (engagement) before onset of labor → degree of descent (fetal station), relationship of presenting part to maternal ischial spines → fetus moves from pelvic inlet (-5 station) down to ischial spines (0 station) to pelvic outlet (+4 station) to crowning at vaginal opening (+5 station)
  - *Flexion*: fetal chin presses against chest, head meets resistance from pelvic floor
  - *Internal rotation*: fetal shoulders internally rotate 45°; widest part of shoulders in line with widest part of pelvic inlet
  - *Extension*: fetal head passes under symphysis pubis (+4 station), moves (+5 station), emerges from vagina
  - *Restitution (external rotation)*: head externally rotates as shoulders pass through pelvic outlet, under symphysis pubis, turns to align with back
  - *Expulsion*: anterior shoulder slips under symphysis pubis, followed by posterior shoulder, rest of the body; marks end of second stage

THIRD STAGE

- Delivery of placenta, umbilical cord, fetal membranes; uterus contracts firmly, placenta begins to separate from uterine wall

FOURTH STAGE

- Physiological adaptation to blood loss, initiation of uterine involution

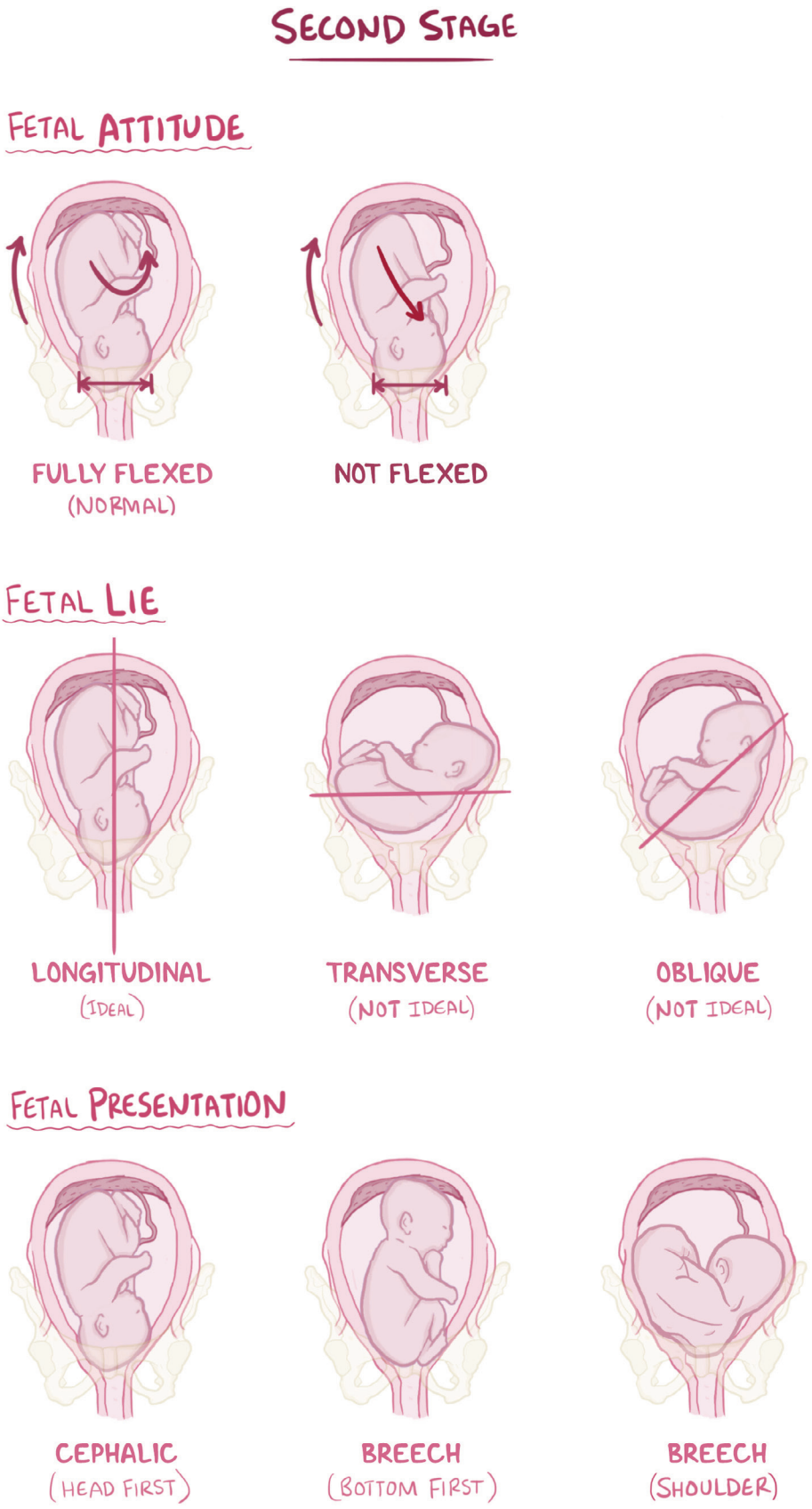

Figure 8.17 Fetal attitude, lie, and presentation are all critical factors in determining the fetus' ease of passage through the maternal pelvis.

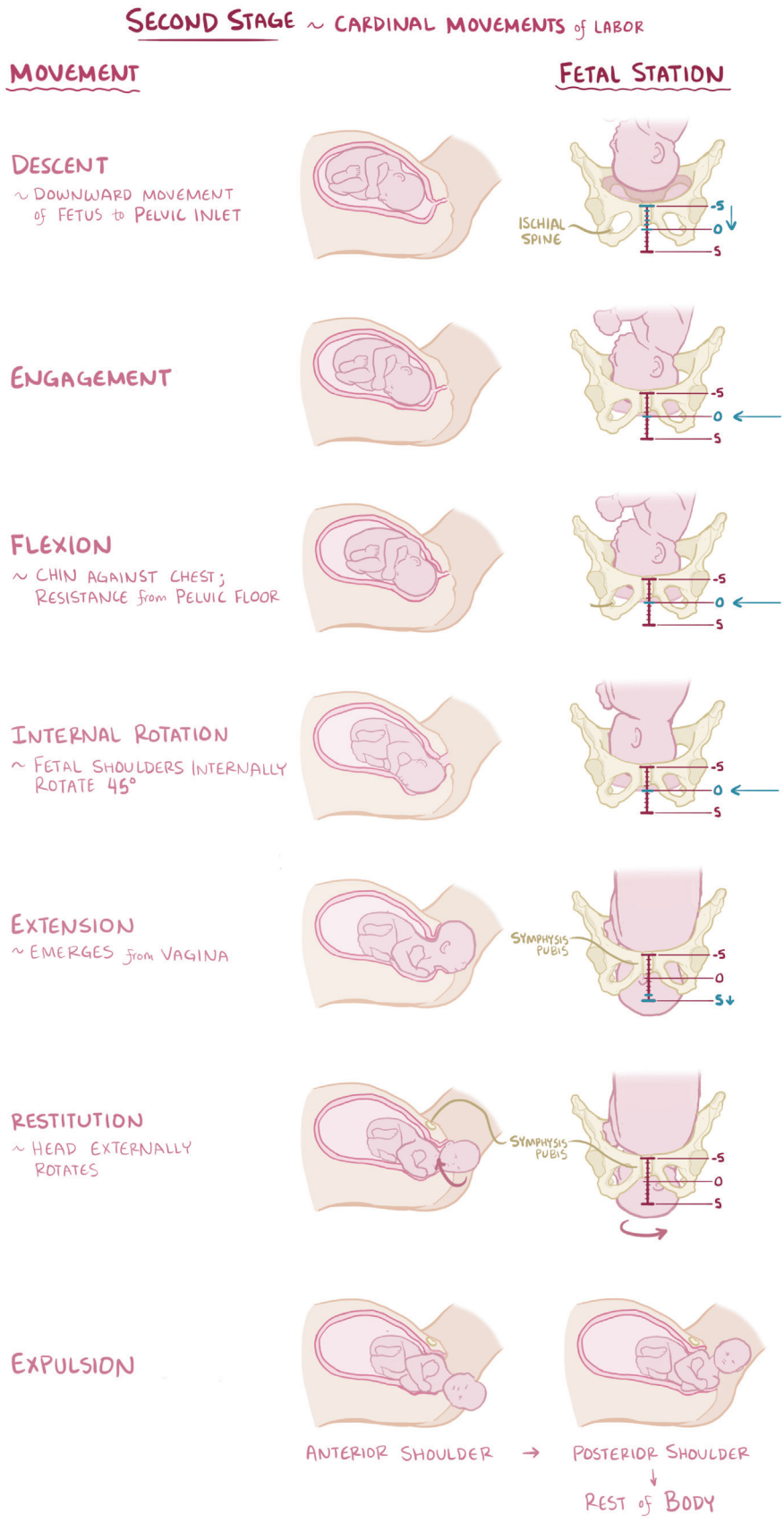

Figure 8.18 Second stage cardinal movements: the fetal position changes that occur during labor.

BREASTFEEDING

osms.it/breastfeeding

- Provision of breast milk from lactating breast; involves breast tissue development, initiation of milk secretion lactogenesis
- Pregnancy, human placental lactogen (hPL), progesterone released from placenta, + PL released from anterior pituitary gland → stimulates growth of breast glandular tissue → prepares epithelial cells lining alveoli to produce milk
  - Progesterone prevents lactation until after delivery of placenta
- Delivery of baby, placenta → ↓↓ progesterone → milk synthesized in alveoli

INFANT SUCKLING

- Stimulates release of oxytocin, PL

Oxytocin

- Required for milk to be released from alveoli
- Neuroendocrine reflex → let-down reflex (milk ejection)
  - Myoepithelial cells contract → milk ejection from alveolus → drained by milk-collecting ducts → transported to nipple
- Milk ejection continues as long as infant continues suckling
- Other triggers for oxytocin release, let-down reflex
  - Sounds/sights/smells connected to infant (e.g. infant crying)

PL

- Continues milk production
- Amount of milk produced depends on amount removed at feeding (supply meets demand)
- Milk extraction facilitated by good latch of baby onto nipple, frequent emptying of breast
  - *Good latch*: baby's mouth wide open, covering areola, lips flanged out, nipple up against roof of mouth, baby's tongue up against bottom of areola
  - Feedings every 1–2 hours at first, then every 3 hours
- If milk not removed, builds up → ↑ intramammary pressure → ↓ capillary blood flow → glandular tissue involutes → ↓ milk production

BIOCHEMICAL COMPOSITION OF BREAST MILK

Benefits for baby

- ↑ whey to casein ratio, enzymes, hormones → ↑ absorption, digestion of milk
- Immunoglobulins
  - ↓ risk of infection; esp. respiratory, gastrointestinal, otitis media; ↓ risk of necrotizing enterocolitis in premature infants
- Long-chain polyunsaturated fatty acids (PUFAs)
  - Aids neural. visual development
- ↑ beneficial bacteria (Lactobacillus, Bifidobacterium) in gut microflora
- Cytokines
  - Anti-inflammatory properties
- Ideal source of nutrition for newborns, including premature infants
- Milk composition transitions from early postpartum period to mature milk to meet infant needs

Benefits for mother

- Accelerated uterine involution, ↓ risk of chronic disease (e.g. diabetes Type II, arthritis, heart disease; cancers of breast, ovaries, uterus)

Colostrum

- Small amounts of milk produced during second half of pregnancy
- Thick, yellowish fluid (due to beta-carotene) rich in immune cells, antibodies, antioxidants, protein, fat-soluble vitamins, minerals; low in fat, lactose
- Protects newborn from infection; laxative effect → passage of first stool (meconium),

formed in fetal gastrointestinal tract
- Helps establish healthy gut microbiome

Transitional milk
- Produced 7–10 days postpartum; thinner than colostrum; light yellow color

Mature milk
- Produces 2 weeks postpartum
- Watery, slight bluish color; fat content increases during feeding
- Biologically complex
  - Protein, fat, sugars (e.g. lactose, oligosaccharides), vitamins, minerals, immunoglobulins, antibodies (esp. secretory IgA), immune cells (e.g. macrophages, neutrophils), immune-modulating factors (e.g. lactoferrin, lysozyme, lactoperoxidase)
- Low in vitamin D; supplementation often recommended
- Continues to be produced until lactation ceases
- Healthy maternal diet supports breast milk production

CONTRAINDICATIONS & CAUTIONS TO BREASTFEEDING

Contraindications
- Certain maternal medications (e.g. chemotherapy), illicit drugs (e.g. cannabis, heroin)
- HIV infection (in high-income settings)
- Herpes zoster, herpes simplex
  - If lesions on breast
- Tuberculosis
  - Until approx. 2 weeks of maternal pharmacotherapy

Cautions
- Smoking discouraged (↑ risk of SIDS, respiratory problems)
- Minimize alcohol; if consumed, wait two hours before breastfeeding
- Limit caffeine

BREASTFEEDING PROBLEMS

Engorgement
- *Cause*: milk accumulation in breast tissue, vascular congestion, resulting in pain
- *Presentation*: firm, tender breast; may have ↑ vascular markings
- *Treatment*: empty breasts (↑ breastfeeding, pumping); warm shower/compresses before feeding (enhances let-down), cool compresses after feeding; nonsteroidal anti-inflammatory drugs (NSAIDs); application of cool green cabbage leaves
- *Prevention*: frequent feedings, good latch to ensure emptying breast

Sore, cracked nipples
- *Cause*: improper latch, positioning
- *Presentation*: pain; blister/bleb on nipple if pores plugged
- *Treatment*: cool/warm compresses; apply expressed breast milk to nipple; mild analgesics (e.g. acetaminophen)
- *Prevention*: good breastfeeding technique

Mastitis
- *Cause*: bacterial infection
- *Presentation*: usually unilateral, localized warmth, tenderness/pain, edema, erythema, firmness; acute onset of flu-like symptoms (e.g. fever, fatigue)
- *Treatment*: continued breastfeeding, NSAIDs, antibiotics
- *Prevention*: good hygiene

Yeast infections
- *Cause*: Candida albicans; history of infant oral/diaper candidal infection/maternal vaginal candidal infection
- *Presentation*: infant may have white plaques in oral area; mother may experience pain, red/sore nipples
- *Treatment*: for mother, topical antifungal applied after feeding; infant, nystatin solution swabbed into oral mucosa after feeding
- *Prevention*: good hygiene; avoid excessive moisture by keeping breasts dry between feedings

MENOPAUSE

osms.it/menopause

- Diagnosed when menstrual cycles have stopped for entire year, no identified pathological cause
- Caused by natural effects of ovarian follicular depletion during aging process
- Usually begins age 50
- Preceded by perimenopause
  - 4 years before final menstrual period; missed/irregular menstrual cycles, changes in bleeding patterns (heavy, prolonged, light)

HORMONAL CHANGES

- ↓ estrogen, progesterone → ↓ hypothalamic inhibition → ↑ bursts of GnRH → ↑ FSH, LH

PHYSIOLOGICAL EFFECTS OF ESTROGEN WITHDRAWAL

Hot flashes

- Caused by hypothalamus-associated thermoregulatory dysfunction → vasomotor instability
- Sensation of heat (centered on chest, face → generalized), diaphoresis, palpitations, anxiety
- Night sweats
  - Hot flashes occur at night → trouble sleeping
- Avoid triggers (e.g. hot drinks, spicy foods); maintain cool ambient temperature; dress in lighter clothing
- Stops within few years of onset

Vulvovaginal atrophy

- Vaginal dryness, loss of vaginal rugae → dyspareunia
- Vaginal estrogen creams, lubricants helpful

↓ protective effects from estrogen

- ↑ risk of cardiovascular disease
- ↓ bone marrow density → ↑ risk of osteoporosis, bone fractures
  - ↑ vitamin D, calcium (diet, supplements) helpful

Others

- Urinary tract dysfunction → dysuria, urinary urgency
- Mood instability → depression, anxiety
- Decline in cognitive function, difficulty concentrating
- ↓ collagen content in skin → ↑ skin wrinkling
- ↓ lean body mass
- Individualized approach for menopausal hormone therapy (MHT)
  - Estrogen/estrogen + progestin helpful in some cases

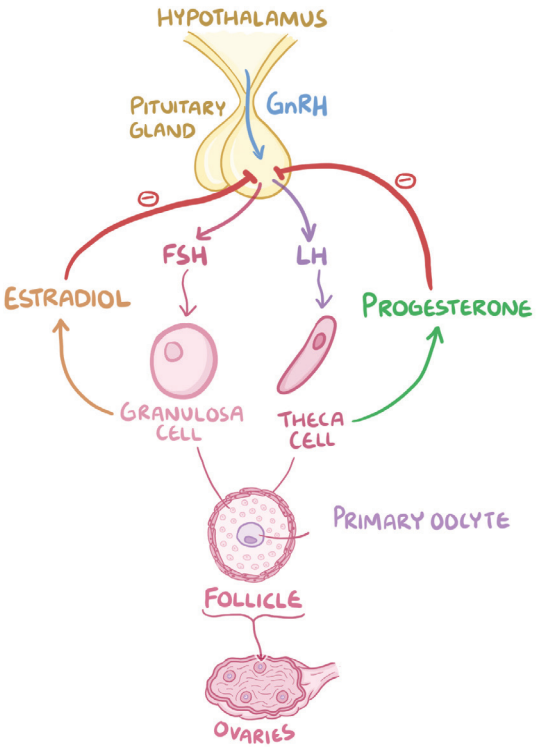

Figure 8.19 Hormone activity in a regular menstrual cycle. Estrogen and progesterone levels ↓ during menopause because the ovaries run out of functional follicles → no theca or granulosa cells to produce more hormones. So ↓ estrogen, progesterone → ↓ hypothalamic inhibition → ↑ bursts of GnRH → ↑ FSH, LH.

ESTROGEN & PROGESTERONE

osms.it/estrogen-progesterone

- Female steroid hormones, produced mainly by ovaries
  - Some estrogen produced in adrenal cortex, adipose tissue; secreted by placenta during pregnancy
  - Corpus luteum secretes estrogen, progesterone
- Three types
  - Estradiol (most biologically active), estrone, estriol

SYNTHESIS

- Cholesterol → theca cells → converted to pregnenolone via cholesterol desmolase → pregnenolone converted into progesterone via 3-beta-hydroxysteroid dehydrogenase (HSD) → released into blood → binds to plasma proteins (e.g. albumin) → transported to target tissues
- Remainder of pregnenolone converted to 17-hydroxypregnenolone → converted into dehydroepiandrosterone (DHEA) → finally converted into androstenedione (testosterone precursor) by 3-beta-HSD
- Androstenedione diffuses to nearby granulosa cells → androstenedione converted to testosterone by 17-beta-hydroxysteroid → testosterone converted to 17-beta-estradiol dehydrogenase aromatase (most biologically active type of estrogen during reproductive period)
- 17-beta-estradiol released into blood → binds to sex hormone-binding globulin (SHBG)
  - Plasma protein, carries 17-beta-estradiol to target tissues (e.g. uterus, vagina, bones)

SECRETION

- Regulated by hypothalamic-pituitary-ovarian axis through feedback loops
- At puberty, pulsatile release of GnRH from hypothalamus → anterior pituitary secretes FSH, LH → ovarian follicles differentiate into theca, granulosa cells → secrete estrogen, progesterone

EFFECTS OF ESTROGEN

- Maturation of female reproductive organs (e.g. uterus, fallopian tubes, vagina)
- Secondary sexual characteristics (e.g. breast growth, fat distribution)
- ↑ estrogen (pre-ovulation) → prepares uterine epithelium for implantation (endometrial proliferation); endometrial secretion in collaboration with progesterone
- Dominant hormone during the follicular phase of ovarian cycle; follicle maturation; initiates ovulation via FSH, LH surge

Pregnancy

- Secreted by placenta to support uterus; stimulates development of myometrium
- ↑ melanin-stimulating hormones → hyperpigmentation
- ↑ vascularity of upper respiratory tract; hypersecretion of mucus
- Preparation for labor
  - Stimulates development of myometrial gap junctions, promotes coordinated contractions
  - Promotes cervical ripening
  - ↑ uterine responsiveness to oxytocin (↑ oxytocin receptors), triggering parturition
- Breasts
  - Stimulates growth of duct cells

Systemic

- Required for closure of epiphyseal plates (both sexes)
- Anabolic effect on bones
- ↓ low-density lipoprotein (LDL), ↑ high-density lipoproteins (HDL)
- Maintains flexibility of blood vessels
- Promotes skin elasticity, fat deposition
- ↓ estrogen during perimenopausal/menopausal years → ↑ risk of cardiovascular morbidity, osteoporosis, sexual dysfunction

EFFECTS OF PROGESTERONE

- Dominant hormone during luteal phase of ovarian cycle
- ↑ progesterone (secretory phase of menstrual cycle) → forms decidual tissue for implantation

Pregnancy

- *Maintains pregnancy*: ↓ irritability of myometrium → ↓ risk of spontaneous abortion
- *Cervis*: forms mucus plug
- *Breasts*: ↑ alveolar-lobular development, prevents milk production during pregnancy (inhibits prolactin)
- *Respiratory*:: ↑ sensitivity to CO2, mild hyperventilation, ↓ airway resistance
- ↑ vasodilation

Systemic

- Works with estrogen to promote bone remodeling → ↑ bone density
- Promotes skin elasticity

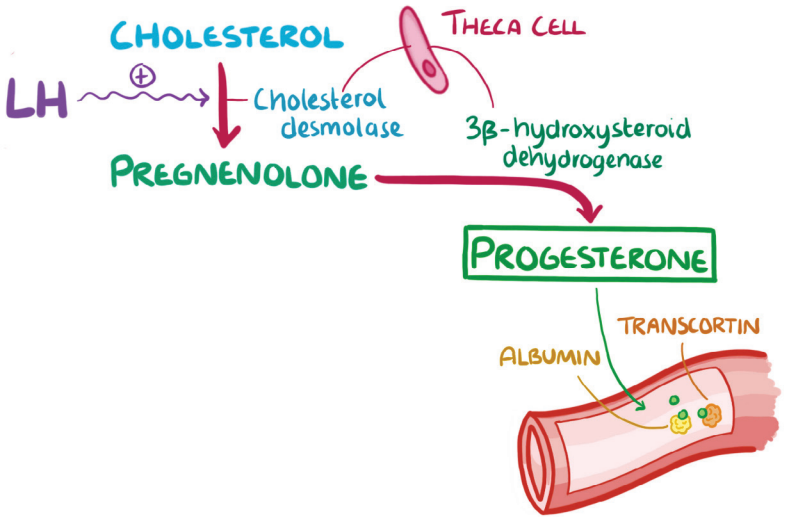

Figure 8.20 The steps of progesterone synthesis. LH stimulates proliferation of theca cells → cholesterol desmolase converts more cholesterol into pregnenolone.

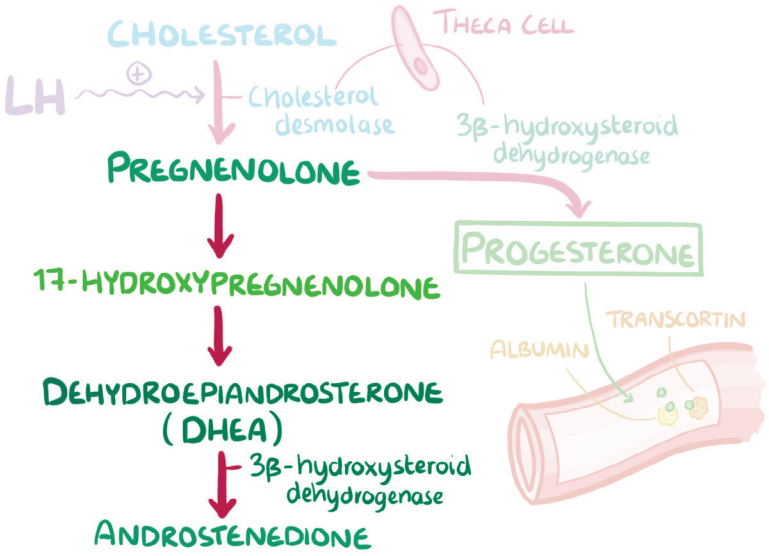

Figure 8.21 Synthesis of androstenedione from pregnenolone. Androstenedione will be used in the next steps to synthesize 17-beta-estradiol.

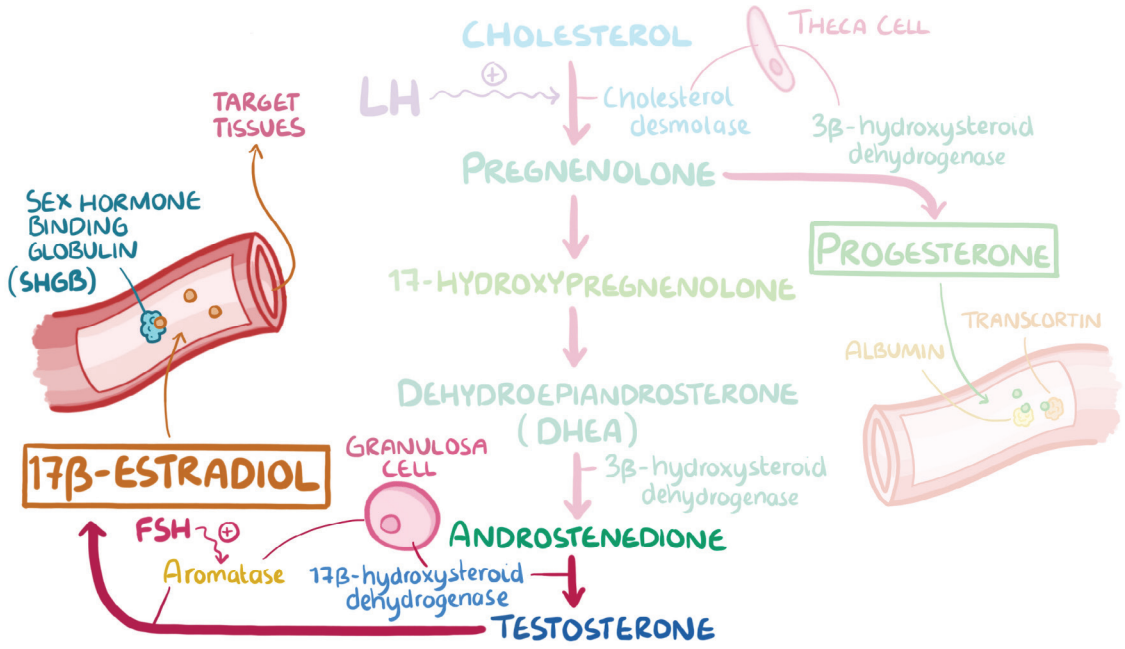

Figure 8.22 Synthesis of 17-beta-estradiol from androstenedione. FSH increases the activity of aromatase. Some target tissues for 17-beta-estradiol include the uterus and vagina, bones, and blood vessels.