OBSTETRICS

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NORMAL OBSTETRICS

DEFINITIONS

Gravidity
❑ the total number of pregnancies of any gestation
- includes current pregnancy, abortions, ectopic pregnancies, and hydatidiform moles
- twins count as one pregnancy

Parity
❑ the number of pregnancies that have been carried to > 20 weeks
- twins count as one
- grand multiparity is parity of 4 or more

❑ four letter description (**T P A L**)
- **T**: number of term infants delivered (> 37 weeks)
- **P**: number of premature infants delivered (20 to 37 weeks)
- **A**: number of abortions (< 20 weeks)
- **L**: number of living children

Trimesters
❑ T1 (first trimester): 0 to 12 weeks
❑ T2 (second trimester): 12 to 28 weeks
❑ T3 (third trimester): 28 to 40 weeks
❑ normal pregnancy term: 37 to 42 weeks

Abortion
❑ loss of intrauterine pregnancy prior to viability of fetus
- < 20 weeks and/or < 500 g fetal weight
- includes induced (therapeutic) and spontaneous (miscarriage)

Stillbirth
❑ loss of intrauterine pregnancy after 20 weeks and/or > 500 g fetal weight

Stillbirth Rate
❑ the annual number of stillbirths per 1,000 total births

Perinatal Mortality Rate
❑ the annual number of stillbirths and early neonatal deaths (in the first seven days of life) per 1,000 total births

❑ causes
- prematurity
- congenital anomalies

Neonatal Mortality Rate
❑ the annual number of deaths of liveborn infants within 28 days per 1,000 live births

Infant Mortality Rate
❑ the annual number of deaths of liveborn infants in the first year of life per 1,000 live births (includes neonatal mortality)

Maternal Mortality Rate
❑ the annual number of deaths of women while pregnant or within 90 days of pregnancy per 100,000 live births
- direct: from obstetrical causes such as ectopic, pregnancy induced hypertension (PIH), post partum hemorrhage (PPH), infection, pulmonary embolus (PE)
- indirect: from pre-existing illness or by accident

Birth Rate
❑ the annual number of live births per 1,000 population

Fertility Rate
❑ the annual number of live births per 1,000 women aged 15-44 years

DIAGNOSIS OF PREGNANCY

Symptoms
❑ amenorrhea
❑ nausea and/or vomiting
❑ breast tenderness
❑ urinary frequency
❑ fatigue

Signs
- ❑ softening of the cervix (Goodell's sign): 4-6 weeks
- ❑ bluish discoloration of the cervix and vagina due to engorgement of pelvic vasculature (Chadwick's sign): 6 weeks
- ❑ uterine enlargement
- ❑ softening of the isthmus (Hegar's sign): 6-8 weeks

INVESTIGATIONS
- ❑ βhCG
  - positive in the serum at 9 days post-conception
  - positive in the urine 28 days after LMP (last menstrual period)
- ❑ transvaginal U/S
  - 5 weeks: gestational sac visible (βhCG = 1,200-1,500 mIU/mL)
  - 6 weeks: fetal pole seen
  - 7-8 weeks: fetal heart tones visible
- ❑ transabdominal U/S
  - 6-8 weeks: intrauterine pregnancy visible (βhCG = 6,500 mIU/mL)

MATERNAL PHYSIOLOGY

General Principles
- ❑ progesterone induces relaxation of smooth muscle, among other effects
- ❑ physiologic changes are more pronounced in multiple gestations

Cardiovascular System (CVS)
- ❑ increased cardiac output, heart rate, and blood volume (hyperdynamic circulation)
- ❑ decreased blood pressure (especially diastolic, maximal in T2) due to decreased peripheral vascular resistance (PVR)
- ❑ blood flow to the uterus, kidneys, breasts, and skin increases with gestational age
- ❑ enlarging uterus compresses inferior vena cava (IVC) and pelvic veins leading to risk of hypotension (by decreasing venous return) as well as varicose veins, hemorrhoids and leg edema (because of increased venous pressure)

Hematologic System
- ❑ apparent decrease in hemoglobin and hematocrit due to hemodilution
  - plasma volume increases more than RBC mass
- ❑ increased risk of deep vein thrombus (DVT) and pulmonary embolus (PE) secondary to hypercoagulable state
  - increase in factors I, VII, VIII, IX, X, XII
  - decrease in factors XI, XIII and antithrombin III activity
  - venous stasis from uterine compression of veins
- ❑ increased leukocyte count but impaired function
  - 5,000 to 12,000/uL in pregnancy
  - up to 25,000/uL in labour/postpartum
  - often have improvement in autoimmune conditions
- ❑ gestational thrombocytopenia
  - occurs in 8% of gestations, possibly due to accelerated platelet consumption
  - mild thrombocytopenia (> 70,000/uL), asymptomatic, no history of prior thrombocytopenia
  - platelet counts normalize within 2-12 weeks following delivery

Respiratory System
- ❑ increased oxygen consumption by 20%
- ❑ increased sensitivity to carbon dioxide (progesterone effect on respiratory centre) results in hyperventilation and respiratory alkalosis compensated by increased renal excretion of serum bicarbonate
- ❑ 50% increase in minute ventilation
- ❑ decreased total lung capacity (TLC), functional residual capacity (FRC) and residual volume (TV) (elevated diaphragms)
- ❑ vital capacity (VC) unchanged
- ❑ increased tidal volume (TV) by 35-50%
- ❑ increased alveolar ventilation by 65%

Gastrointestinal (GI) System
- ❑ increased gastroesophageal reflux
  - decreased sphincter tone
  - delayed gastric emptying
  - increased intraabdominal pressure
- ❑ increased stasis in gallbladder
- ❑ decreased GI motility and constipation
- ❑ upward displacement of appendix (appendicitis may have atypical presentation)
- ❑ hemorrhoids caused by constipation and elevated venous pressure

Genitourinary (GU) System
- ❑ increased glomerular filtration rate (GFR) by 50% (therefore decreased BUN and serum creatinine) but no change in urine output because of increased tubular reabsorption
- ❑ glycosuria can be physiologic; with increase in GFR the threshold for glucose reabsorption can be surpassed
- ❑ increased urinary frequency
- ❑ physiologic dilatation of ureters and renal pelvis (R > L) due to progesterone-induced smooth muscle relaxation and uterine enlargement
- ❑ increased incidence of UTI and pyelonephritis (see Medical Conditions in Pregnancy section)

Endocrine System
- ❑ estrogen
  - main estrogen is estradiol
  - production involves an intricate pathway, requiring maternal, placental and fetal contributions
  - sudden decline may indicate fetal compromise
- ❑ progesterone
  - produced by corpus luteum during first 7 weeks, thereafter synthesized by the placenta
  - maintains the endometrium
  - absolutely necessary for continuation of pregnancy
- ❑ human chorionic gonadotropin (hCG)
  - produced by placental trophoblastic cells
  - peptide hormone composed of two subunits: alpha (common to all glycoproteins) and beta (specific to hCG)
  - has LH-like action: maintains the corpus luteum
  - serum βhCG positive 8-9 days after ovulation
  - plasma levels double every 1-2 days, peak at 8-10 weeks and then fall to a plateau until delivery
  - rule of 10s
    - 10 IU at time of missed menses
    - 100,000 IU at 10 weeks (peak)
    - 10,000 IU at term
  - levels below expected by dates suggest an ectopic pregnancy, abortion, or wrong dates
  - levels higher than expected suggest multiple gestation, molar pregnancy, trisomy 21, or wrong dates
- ❑ 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)
- ❑ prolactin
  - produced by maternal pituitary in response to increasing estrogen in pregnancy
  - stimulates lactation
- ❑ relaxin
  - produced by the corpus luteum/ovary
  - relaxes symphysis pubis and other pelvic joints
  - helps soften and dilate the cervix
  - inhibits uterine contraction
- ❑ calcium metabolism
  - total maternal Ca^{2+} decreased due to decreased albumin
  - free ionized (i.e. active) proportion remains the same due to increased parathyroid hormone (PTH) which results in increased bone resorption and gut absorption
  - bone turnover increased but no loss of bone density because estrogen counteracts PTH by inhibiting resorption

Neurologic System
- ❑ increased incidence of carpal tunnel syndrome and Bell's palsy

Integumentary System
- ❑ pigmentation changes (fade after delivery)
  - increased pigmentation of perineum and areola
  - chloasma (pigmentation changes under eyes and on bridge of nose)
  - linea nigra (midline abdominal pigmentation)
  - spider angiomas
  - palmar erythema
- ❑ striae gravidarum (fade but seldom disappear)

PRENATAL CARE

PRECONCEPTION COUNSELING

❑ medication
- folic acid to prevent neural tube defects (NTDs) (0.4 to 1 mg daily in all women, 4 mg if past NTD); start 12 wks preconception
- iron supplementation in 2nd and 3rd trimester

❑ proper nutrition and physical fitness
❑ genetic history and risk factors
❑ viral screening
- HBsAg, rubella, +/– HIV

❑ social history
- alcohol, smoking, drug use
- domestic violence (50% of domestic violence begins in pregnancy)
- impact on family and occupation (maternity/paternity leave)

INITIAL VISIT

❑ fill out antenatal forms 1 and 2

History

❑ determine gestational age (GA) by dates from the first day of the LMP (if regular periods and sure dates)
❑ if LMP unsure, get a dating ultrasound (best between weeks 8-12)
❑ determine estimated date of conception (EDC) using Nägele's Rule
- first day of LMP + 7 days – 3 months
- e.g. LMP = 1 Apr. 2001, EDC = 8 Jan. 2002
- modify appropriately for longer or shorter cycles

❑ obtain obstetrical history of all previous pregnancies: GTPAL/year/sex/weight/gestational age/mode of delivery/length of labour/complications/uterine surgery (myomectomy, D&C's, cone biopsies)
❑ obtain relevant medical, social, and family history
❑ prenatal classes

Physical

❑ complete physical exam
❑ baseline blood pressure (BP) (important for interpreting subsequent changes)
❑ baseline weight
❑ pelvic exam

Investigations

❑ bloodwork
- CBC, blood group and type, Rh antibodies
- rubella titre, VDRL, HBsAg routine; HIV serology should be offered to all (if viral screen not already done preconception)

❑ urine
- R&M, C&S
- asymptomatic bacteriuria in 5% of pregnant women
- if untreated, 25-30% will get a UTI in pregnancy (increased risk of preterm labour)

❑ pelvic exam
- Pap smear (if none within 6 months), culture for GC and *chlamydia*

Counselling

❑ exercise
- exercise should be maintained at approximately the same level as before pregnancy
- muscle strength and flexibility improve posture and muscle tone and reduce common discomforts of pregnancy
- aggressive exercise (prolonged jogging and skiing) should be avoided as the developing fetus can affect balance

❑ nutrition
- in general, a daily caloric increase of 300 kcal throughout pregnancy is recommended
- for women who do not consume an adequate diet, a daily multivitamin should be continued in the second trimester; otherwise routine multivitamin supplementation is not necessary (only one vitamin per day is recommended to avoid excess vitamin A)
- iron is the only known nutrient for which requirements during pregnancy cannot be met by diet alone (see Iron Deficiency Anemia section)

❑ work
- for women who work, rest periods are recommended to avoid the likelihood of fatigue
- stressful work during pregnancy is associated with a greater risk of preterm delivery and poor fetal growth
- heavy forms of work should be discouraged

- ❑ travel
  - in general, travel by car, plane, or train is not harmful during pregnancy
  - however, stress related to travel may be associated with preterm labour
- ❑ sexual intercourse
  - sexual intercourse may continue throughout pregnancy except in patients at risk for abortion, preterm labour, or those with placenta previa
  - breast stimulation may induce uterine activity and is discouraged in high-risk patients near term
  - labour may follow coitus due to the effect of prostaglandins in the seminal fluid

SUBSEQUENT VISITS

- ❑ for low-risk, uncomplicated pregnancy
  - q monthly until 28 weeks
  - q 2 weeks from 28 to 36 weeks
  - q weekly from 36 weeks until delivery

With Every Visit

- ❑ estimate GA
- ❑ ask about fetal movements, bleeding, leaking, cramping
- ❑ urine dip for glucose and protein
- ❑ weight gain
  - expect gain of roughly 1 lb/month in first half of pregnancy, 1 lb/week in second half of pregnancy
  - average weight gain 25-35 lbs (40% of weight gain is due to products of conception)
- ❑ BP
- ❑ symphysis-fundal height (SFH) measurement: SFH should be within 2 cm of gestational age in weeks between 20 and 37 weeks, i.e. SFH = 20 cm @ 20 weeks
  - 12 weeks: fundus at pubic symphysis
  - 20 weeks: fundus at umbilicus
  - 37 weeks: fundus at sternum
- ❑ differential diagnosis of uterus (SFH) incorrect size for dates (accurate dates essential)
  - maternal: diabetes mellitus
  - maternal-fetal: poly/oligo-hydramnios, multiple gestation
  - fetal: abnormal karyotype, IUGR, fetal anomaly, abnormal lie
- ❑ examination of abdomen for lie, position and presentation (Leopold maneuvers) in T3
- ❑ fetal heart tones starting at ~ 12 weeks using doppler U/S

Table 1. Gestation-Dependent Management

Gestational Age (weeks)	Management Issues
10-12	CVS U/S for nuchal translucency (if available)
15-16 or up to term	Amniocentesis
16	MSS (maternal serum screen)
16-18	U/S for dates and structural assessment Quickening (fetal movement felt by mother)
26 -28	50 g oral glucose challenge test (OGCT)
28	Repeat CBC Rhogam to Rh negative woman
36	Rh antibody screen if indicated GBS screen
6 (postpartum)	Follow-up visit • discuss contraception • breast exam and pelvic exam, Pap smear • depression/mental health

Maternal Serum Screen (MSS or Triple Screen)
- ❑ offered to all mothers at 16 weeks
- ❑ provides a risk estimate of whether the fetus may be affected with Down Syndrome, trisomy 18, or an open neural tube defect (oNTD)
- ❑ other chromosomal abnormalities not detected; therefore still offer amniocentesis/chorionic villous sampling (CVS) to high risk women
- ❑ results are highly dependent on gestational age, so accurate dating is important
- ❑ to make accurate diagnosis, positive MSS should be followed up with U/S and/or amniocentesis
- ❑ three markers: maternal serum alpha-feto protein (MSAFP), βhCG, unconjugated estrogen (uE3)
    - Trisomy 21: low MSAFP, high βhCG, low uE3
    - Trisomy 18: low MSAFP, low βhCG, low uE3
- ❑ differential diagnosis of high MSAFP
    - wrong gestational age
    - > 1 fetus (e.g. twins)
    - fetal demise
    - oNTD
    - abdominal wall defects (e.g. omphalocele)
- ❑ differential diagnosis of low MSAFP
    - gestational trophoblastic neoplasia (GTN)
    - incorrect GA
    - missed abortion
    - chromosomal anomalies (e.g. Trisomy 18, 21)
- ❑ 80% of Down's babies born to women under 35 years, so MSS is a valuable screening tool
    - sensitivity for Down Syndrome detection: 60%
    - sensitivity for oNTD: 80-90%
- ❑ MSS has a 9.5% false positive rate if maternal age >35; lower false positive rate for oNTD and T18

Group B Streptococcus (GBS)
- ❑ not harmful to mother
    - 15-40% vaginal carrier rate
- ❑ danger of vertical transmission (neonatal sepsis, meningitis or pneumonia); treatment of maternal GBS at time of delivery decreases morbidity and mortality for neonates
- ❑ guidelines controversial: screening with vaginal cultures often done at 34-36 weeks, but this does not always reflect colonization status at delivery; some physicians screen all while others treat empirically only
- ❑ indications for antibiotic prophylaxis:
    - positive GBS screen OR
    - GBS status unknown and one of the following risk factors
        - previous GBS bacteriuria even if treated
        - previous infant with GBS infection
        - preterm labour
        - premature rupture of membranes (PROM) > 12 hours
        - intrapartum maternal temperature > 37.7ºC
        - fetal tachycardia
- ❑ treat with ampicillin 2 g IV load, then 1 g IV q4h until delivery (if pen-allergic, use clindamycin)

PRENATAL DIAGNOSIS

Indications
- ❑ maternal age > 35 (increased risk of chromosomal anomalies)
- ❑ risk factors in current pregnancy
    - teratogenic exposure
    - abnormal MSS or U/S
- ❑ past history/family history of
    - previous pregnancy with chromosomal anomaly or genetic disease
    - either parent a known carrier of a genetic disorder or balanced translocation
    - family history of chromosomal anomaly, genetic disorder, birth defect, or undiagnosed mental retardation
    - consanguinity
    - three or more spontaneous abortions

Amniocentesis
- ❑ U/S-guided transabdominal extraction of amniotic fluid
- ❑ indications
  - identification of genetic problems such as trisomies (15-16 weeks gestation)
  - assessment of fetal lung maturity (third trimester)
  - L/S ratio (lecithin:sphingomyelin): if > 2:1, fetal lungs are mature enough that respiratory distress syndrome (RDS) is less likely to occur
  - assessment of amniotic fluid bilirubin concentration in Rh-isoimmunized pregnancies
- ❑ advantages
  - also screens for oNTD (acetylcholinesterase and amniotic AFP)
  - more accurate genetic testing
- ❑ disadvantages
  - 0.5% risk of spontaneous abortion
  - results take 10-14 days; FISH available in 72 hours
- ❑ in women over 35 years, the risk of chromosomal anomaly (1/180) is greater than the increased risk of miscarriage from the procedure, so it is offered routinely

Chorionic Villus Sampling (CVS)
- ❑ needle through abdomen or catheter through cervix at 10-12 weeks for CVS
- ❑ the chorion is of fetal origin and cells obtained by villus biopsy can be examined using the same techniques as for amniocytes
- ❑ advantages
  - enables pregnancy to be terminated earlier
  - more rapid karyotyping, DNA tests, chromosome status, biochemical assay (results in 48 hours; do not have to wait for culture)
  - increasing availability of probes to allow diagnosis of genetic abnormalities (i.e. FISH)
  - high sensitivity and specificity
- ❑ disadvantages
  - 1-2% risk of spontaneous abortion
  - does not screen for neural tube defects (NTD)
  - risk of limb injury
  - 1-2% incidence of genetic mosaicism —> false negative results

Table 2. Amniocentesis versus CVS

	Amniocentesis	CVS
Accuracy of prenatal cytogenetic diagnosis	99.8%	97.5%
Detection of cytogenetic abnormality	3.4%	5.6%
Laboratory failure	0.1%	2.3%

FETAL MONITORING

ANTENATAL MONITORING

Fetal Movements
- ❑ assessed by
  - maternal perception (quickening)
    - choose a time when baby is normally active to count movements
    - if < 6 movements in 2 hours, notify MD
    - 10 movements in 12 hour period is lower limit of normal (32 weeks and over)
    - false positive rate = 30-60%, false negative rate is < 5%
  - palpation
  - U/S

Ultrasound
- ❑ routinely done at 16-20 weeks to assess fetal growth and anatomy
- ❑ earlier or subsequent U/S only when medically indicated
  - confirmation of intrauterine pregnancy
  - identification of multiple pregnancy
  - past history of early fetal losses
  - bleeding or other complications
  - measurement of fetal growth and identifiation of IUGR
  - placental localization
  - determining gestational age (most accurately determined through measurement of crown-rump length prior to 11-12 weeks gestational age)
  - assessment of amniotic fluid index (AFI): total of largest amniotic fluid pocket measurements in 4 quadrants of gestational sac, normally 8-24 cm

Non-Stress Test (NST)
- ❑ constant fetal heart rate (FHR) tracing using an external doppler to assess FHR and its relationship to fetal movement (see Intrapartum Fetal Cardiotocography section)
- ❑ indicated when there is any suggestion of uteroplacental insufficiency or suspected fetal distress
- ❑ false positive rate depends on duration; false negative rate = 0.2-0.3%
- ❑ reactive NST (normal/negative result)
  - at least 2 accelerations of FHR > 15 bpm from the baseline lasting ≥ 15 seconds, in 20 minutes
- ❑ nonreactive NST (abnormal/positive result)
  - < 2 FHR accelerations > 15 bpm and 15 seconds duration associated with fetal movement in 40 minutes
  - if no observed accelerations or fetal movement in the first 20 minutes, stimulate fetus (fundal pressure, acoustic/vibratory stimulation) and continue monitoring for 30 minutes
  - if NST nonreactive, then perform biophysical profile (BPP)

Biophysical Profile (BPP)
- ❑ consists of NST and 30 minute U/S assessment of the fetus
- ❑ five scored parameters of BPP (see Table 3)
- ❑ scores
  - 8-10: perinatal mortality rate 1:1,000; repeat BPP as clinically indicated
  - 6: perinatal mortality 31:1,000; repeat BPP in 24 hours
  - 0-4: perinatal mortality rate 200:1,000; deliver fetus if mature
- ❑ amniotic fluid volume (AFV) = a marker of chronic hypoxia, all other parameters indicate acute hypoxia
- ❑ false positive rate = < 30%, false negative rate = 0.1%

Table 3. Scoring of the Biophysical Profile

Parameter	Reassuring (2)	Non-Reassuring (0)
AFV	Fluid pocket of 2 cm in 2 axes	Oligohydramnios
NST	Reactive	Nonreactive
Breathing	At least one episode of breathing lasting at least 30 seconds	No breathing
Limb Movement	Three discrete movements	Two or less
Fetal Tone	At least one episode of limb extension followed by flexion	No movement

INTRA-PARTUM MONITORING

Vaginal Exam
- ❑ membrane status
- ❑ cervical effacement (thinning), dilatation, consistency, position, application
  - a - application
  - b - position
  - c - consistency
  - d - dilatation
  - e - effacement
- ❑ fetal presenting part, position, and station
- ❑ bony pelvis size and shape

Intrapartum Fetal Cardiotocography (CTG)
- ❑ intermittent auscultation or continuous electronic monitoring are acceptable methods of intrapartum surveillance in both low and high-risk pregnancies (longer checking intervals in uncomplicated pregnancies)
- ❑ no scientific evidence has identified the most effective method of fetal surveillance (including frequency and duration) that ensures optimum results
- ❑ continuous electronic monitoring: external (doppler) vs. internal (scalp electrode) monitoring
- ❑ internal monitoring recommended after abnormal fetal heart rate (FHR) found on external monitoring
- ❑ described in terms of baseline FHR, variability (short term, long term) and periodicity (accelerations, decelerations)
- ❑ baseline FHR
  - normal range is 120-160 bpm
  - a parameter of fetal well-being vs. distress

- ❑ variability: an indicator of fetal oxygenation
  - short term: beat to beat (requires scalp monitor)
  - long term: described with respect to frequency and amplitude of change in baseline
  - frequency: number of times in a 1 minute period of an increase or decrease of at least 5 bpm lasting 5 seconds (average frequency is 3)
  - amplitude: difference between highest and lowest FHR within a 1 minute period (11-25 bpm is average)
- ❑ periodicity
  - accelerations
    - increase of ≥ 15 bpm lasting ≥ 15 seconds, in response to fetal movement or uterine contraction
  - decelerations
    - 3 types, described in terms of shape, onset, depth, duration, recovery, occurrence, and impact on baseline FHR and variability
    - early decelerations (see Figure 1)
      - uniform shape with onset early in contraction; returns to baseline by end of contraction
      - slow gradual deceleration
      - often repetitive; no effect on baseline FHR or variability
      - due to vagal response to head compression
      - benign, usually seen with cervical dilatation of 4-7 cm

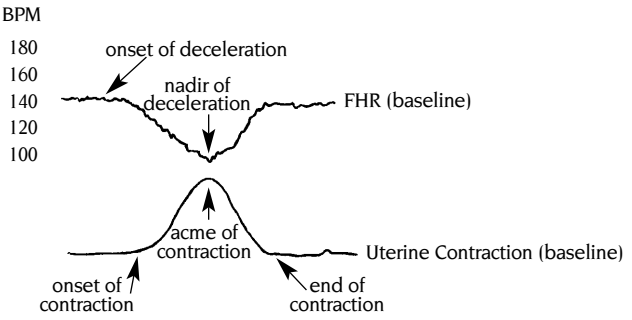

Figure 1. Early Decelerations

  - variable decelerations (see Figure 2)
    - variable in shape, onset, and duration
    - most common type of periodicity seen during labour
    - may or may not be repetitive
    - often with abrupt rapid drop in FHR; usually no effect on baseline FHR or variability
    - due to cord compression or, in second stage, forceful pushing with contractions
    - benign unless repetitive, with slow recovery, or when associated with other abnormalities of FHR
    - rule of 60s for severe variable decelerations: decels to < 60 bpm, or > 60 bpm below baseline and > 60 s in duration with slow return to baseline

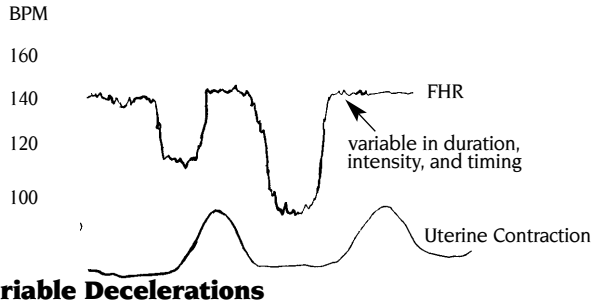

Figure 2. Variable Decelerations

  - late decelerations (see Figure 3)
    - uniform shape with onset late in contraction, lowest depth after peak of contraction, and return to baseline after end of contraction
    - may cause decreased variability and change in baseline FHR
    - must see 3 in a row, all with the same shape to define a late deceleration
    - due to fetal hypoxia and acidemia, maternal hypotension or uterine hypertonus
    - usually a sign of uteroplacental insufficiency (ominous)
    - manage with position change to left lateral decubitus, oxygen, stop oxytocin, correct maternal hypertension, fetal scalp pH, +/– C/S when necessary

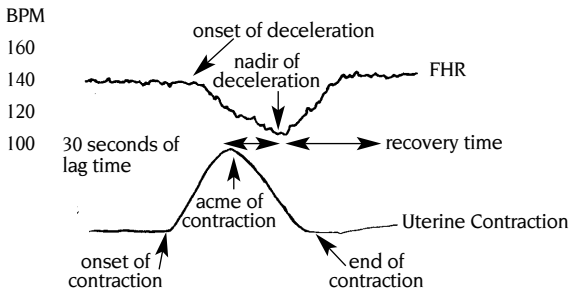

Figure 3. Late Decelerations

Table 4. Factors Affecting Fetal Heart Rate

	Fetal Tachycardia (FHR >160)	Fetal Bradycardia (FHR <120)	Decreased Variability
Maternal Factors	Fever Hyperthyroidism Anemia	Hypothermia Hypotension Hypoglycemia	Infection Dehydration
Fetal Factors	Arrhythmia Anemia	Rapid descent Dysrhythmia Heart block	CNS anomalies Dysrhythmia Inactivity/sleep cycle
Drugs	Sympathomimetics (ritodrine)	Beta blockers Anesthetics	Narcotics Magnesium sulphate
Uteroplacental	Early hypoxia (abruption, PIH) Chorioamnionitis	Late hypoxia (abruption, PIH) Acute cord prolapse Hypercontractility	Hypoxia

Approach to Abnormal FHR

- ❑ if external monitor, ensure fetal tracing and not maternal
- ❑ change position of mother
- ❑ give 100% oxygen by mask and discontinue oxytocin
- ❑ rule out cord prolapse
- ❑ consider fetal scalp electrode to assess beat-to-beat variability and fetal scalp blood sampling if abnormality persists
- ❑ immediate delivery if recurrent prolonged bradycardia

Fetal Scalp Blood Sampling

- ❑ indicated when fetal distress is suggested by clinical parameters including heavy meconium or moderate to severely abnormal FHR patterns
- ❑ > 7.25 is normal
- ❑ < 7.25 indicates that test should be repeated in 30 minutes
- ❑ < 7.20 indicates fetal acidosis severe enough to warrant immediate delivery

Meconium in the Amniotic Fluid

- ❑ usually not present early in labour
- ❑ classified as: early (prior to ROM) or late (after rupture of membranes (ROM) with clear fluid)
- ❑ can be thick or thin
    - thin meconium: light green or yellow, not usually associated with poor outcome
    - thick meconium: dark green or black, pea-soup consistency, associated with lower APGARs and increased risk of meconium aspiration
        - call pediatrics to delivery
        - may indicate undiagnosed breech
- ❑ increasing meconium during labour may be a sign of fetal distress
- ❑ 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

MULTIPLE GESTATION

BACKGROUND

- ❑ incidence of twins is 1/80 and triplets 1/6400 in North America
- ❑ 2/3 of twins are dizygotic (i.e. fraternal)
- ❑ hereditary factors (on maternal side only) and fertility drugs/procedures affect the dizygotic twins rate only
- ❑ monozygous twinning occurs at a constant rate worldwide (1/250)
- ❑ determination of zygosity by number of placentas, thickness of membranes, sex, blood type

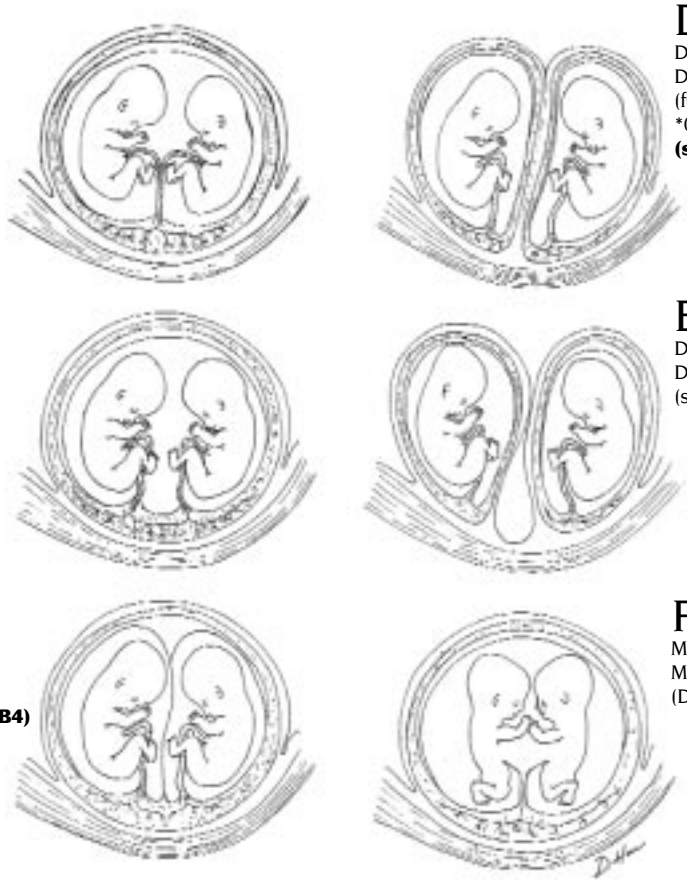

Figure 4. Classification of Twin Pregnancies *Indicates time of cleavage

Illustration by David Hou

Table 5. Complications Associated with Multiple Gestation

Maternal	Utero-placental	Fetal
Hyperemesis gravidarum GDM Preeclampsia/PIH Anemia (increased iron and folate needs) Increased physiological stress on all systems Increased compressive symptoms C/S	Increased PROM/PTL Polyhydramnios Placenta previa Placental abruption PPH (uterine atony) Umbilical cord prolapse Cord anomalies (velamentous insertion, 2 vessel cord)	Prematurity* IUGR Malpresentation Congenital anomalies Twin-twin transfusion (DA/MC) Increased perinatal morbidity and mortality Twin interlocking (twin A breech, twin B vertex) Single fetal demise (may lead to DIC)

*Most common cause of perinatal mortality in multiple gestation

PIH = pregnancy induced hypertension
PPH = postpartum hemorrhage
GDM = gestational diabetes mellitus
DIC = disseminated intravascular coagulation
DA = diamnionic
MC = monochorionic

MANAGEMENT
- ❑ rest in T3 (increases uterine blood flow)
- ❑ increased antenatal surveillance
  - nonstress test (NST) q weekly from 24 weeks GA
  - serial U/S q 2-3 weeks from 28 weeks GA to assess growth
  - doppler flow studies q weekly if discordant fetal growth
  - BPP as needed
- ❑ vaginal examinations in third trimester to check for cervical dilatation
- ❑ 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

TWIN-TWIN TRANSFUSION SYNDROME
- ❑ 10% of monochorionic twins
- ❑ arterial blood from donor twin passes through placenta into vein of recipient twin
- ❑ donor twin: IUGR, hypovolemia, hypotension, anemia, oligohydramnios
- ❑ recipient twin: hypervolemia, hypertension, CHF, polycythemia, edema, polyhydramnios, kernicterus in neonatal period
- ❑ management: therapeutic serial amniocentesis to decompress polyhydramnios of recipient twin and decrease pressure in cavity and on placenta; laser occlusion of placental vessels

MEDICAL CONDITIONS IN PREGANCY

URINARY TRACT INFECTION (UTI)
- ❑ most common medical complication of pregnancy
- ❑ asymptomatic bacteriuria in 2-7% of pregnant women depending on parity and socioeconomic factors

Pathophysiology
- ❑ due to increased urinary stasis from mechanical and hormonal (progesterone) factors
- ❑ organisms are the same as non-pregnant woman, and also GBS

Signs and Symptoms
- ❑ range from minimal to severe
- ❑ if urethra infected there will be some degree of dysuria, urgency, and frequency

Investigations
- ❑ urinalysis, urine culture
- ❑ VCUG, cystoscopy, and renal function tests in recurrent infections

Management
- ❑ uncomplicated UTI
  - first line: amoxicillin
  - alternatives: TMP-SMX (Septra) or nitrofurantoin (avoid sulpha drugs during last 6 weeks of pregnancy due to displacement of bilirubin from albumin and increased kernicterus in the newborn)
  - follow with monthly urine cultures
  - recurrence common
- ❑ pyelonephritis
  - hospitalization and IV antibiotics

Complications
- ❑ acute cystitis, pyelonephritis, and possible PPROM

RED FLAGS
- ❑ may be asymptomatic; treat ALL pregnant women with bacteriuria to prevent preterm labour

IRON DEFICIENCY ANEMIA
- ❑ iron requirements increase during pregnancy due to
  - fetal/placental growth (500 mg)
  - increased maternal RBC mass (500 mg)
  - losses (200 mg)
- ❑ mother needs 1,000 mg of elemental iron per fetus; this amount exceeds normal stores + dietary intake
- ❑ responsible for 80% of causes of nonphysiologic anemia during pregnancy

Etiology
- ❑ inadequate iron intake
- ❑ decreased iron absorption (malabsorption syndrome, antacid use)
- ❑ increased losses (bleeding from vaginal or other source)
- ❑ increased requirement (fetal growth, multiple gestation)

Signs and Symptoms
- ❑ non-specific symptoms: pallor, fatigability, palpitations, tachycardia, and dyspnea
- ❑ severe anemia: angular stomatitis, glossitis, and koilonychia

Investigations
- ❑ serum iron, serum ferritin, blood smear
  (total iron binding capacity (TIBC) not reliable because of increase during pregnancy)

Management
- ❑ prevention: 150 mg ferrous sulfate OD, 300 mg ferrous gluconate OD,
  or 30 mg of ferrous iron OD for all pregnant women in 2nd and 3rd trimester
- ❑ if anemic: 1 g ferrous sulfate OD (180 mg elemental Fe)

Complications
- ❑ maternal: angina, CHF, infection, slower recuperation, preterm labour
- ❑ fetal: decreased oxygen carrying capacity leading to fetal distress,
  IUGR, low birth weight and hydrops

FOLATE DEFICIENCY ANEMIA
- ❑ most often associated with iron deficiency anemia
- ❑ incidence varies from 0.5-25% depending on region, population, diet
- ❑ necessary for closure of neural tube during early fetal development
- ❑ minimum daily requirement is 0.4 mg
- ❑ takes approximately 18 weeks of folate deficient diet to produce anemia
- ❑ non-nutritional factors include: multiple gestation, drugs (phenytoin, methotrexate),
  chronic hemolytic anemia, malabsorption entities (sprue)

Signs and Symptoms
- ❑ non-specific symptoms: anorexia, nausea, vomiting, diarrhea, depression,
  pallor, UTI, sore mouth or tongue

Investigations
- ❑ serum folate, blood smear

Prevention
- ❑ 0.4 to 1 mg folic acid PO daily for 1-3 months preconceptually and throughout T1
- ❑ 4 mg folic acid per day with past history of NTD

Complications
- ❑ maternal: smaller blood volume, nausea, vomiting, anorexia
- ❑ fetal: NTD in T1, low birth weight, prematurity

DIABETES MELLITUS (DM)

Incidence
- ❑ 2-3% of pregnancies are complicated by diabetes mellitus

Normal Physiology in Pregnancy
- ❑ in early pregnancy (T1) insulin secretion is increased and its anabolic
  actions are potentiated, decreasing fasting maternal glucose levels and
  promoting maternal energy storage
- ❑ in later pregnancy (T2, T3) insulin resistance develops
- ❑ anti-insulin factors: human placental lactogen (increased secretion with growth of the placenta)
  and cortisol
- ❑ result: higher fasting glucose and enhanced lipolysis (increased FFA, TG, lipids, ketones)
  to supply energy for fetal growth

Classification of Diabetes Mellitus
- ❑ Insulin Dependent DM (Type 1)
- ❑ Non-Insulin Dependent DM (Type 2)
- ❑ Gestational Diabetes (GDM): DM occurring in pregnancy

Complications of Pregnancy in the Diabetic

❑ maternal
- hypertension/PET, polyhydramnios, pyelonephritis/UTI
- ketoacidosis, diabetic coma, worsening retinopathy and nephropathy in Type 1 or Type 2, NOT in GDM

❑ fetal
- macrosomia: maternal hyperglycemia leads to fetal hyperinsulinism resulting in accelerated anabolism and macrosomia
- congenital anomalies: cardiac (VSD), NTD, GU (cystic kidneys), GI (anal atresia), MSK (sacral agenesis)
- IUGR
- delayed fetal lung maturity (hyperglycemia interferes with surfactant synthesis)
- preterm labour/prematurity
- increased incidence of spontaneous abortion
- increased incidence of stillbirth
- note: pregnancies complicated by GDM do not manifest an increased risk of congenital anomalies because it develops later (i.e. after T1)

❑ neonatal
- macrosomia and associated birth trauma, hypoglycemia, hyperbilirubinemia and jaundice, hypocalcemia, polycythemia, and RDS

Management of Type 1 and Type 2 DM in Pregnancy

❑ before pregnancy
- optimize glycemic control
- counsel patient re: potential risks and complications
- advise preconception folic acid
- evaluate for diabetic retinopathy, neuropathy, coronary artery disease
- refer to diabetes clinic

❑ during pregnancy
- measure HbA1C (indicates glycemic control during embryogenesis and can be used to estimate risk of birth defects)
- switch type 2 diabetics to insulin (oral hypoglycemics contraindicated)
- monitor as for normal pregnancy plus
  - initial 24-hr urine protein and creatinine clearance, retinal exam, HbA1C
  - encourage increased blood sugar self-monitoring
  - periodic HbA1C and urine C&S
  - increased fetal surveillance (BPP, NST)
    - < 36 weeks: q 1-2 weeks
    - > 36 weeks: q weekly or biweekly
  - consider fetal echo for congenital heart disease
  - admit for blood sugar control as needed
  - note: in T2 the demand for insulin is increased; adjust dosages
  - MSAFP may be decreased in diabetic pregnancies, altering interpretation of this test

❑ during labour
- timing of delivery depends on fetal and maternal risk factors
- can wait for spontaneous labour if glucose well-controlled and BPP normal
- induce by 40 weeks
- increased risk of CPD and shoulder dystocia with babies > 4,000 g (8.8 lbs)
- elective C/S for predicted birthweights > 4,500 g (9.9 lbs) (controversial)
- during labour monitor sugars q1h with patient on insulin and dextrose drip; aim for blood sugar of 3.5 to 6.5 to reduce the risk of neonatal hypoglycemia

❑ postpartum
- insulin requirements dramatically drop with expulsion of placenta which produced insulin antagonists
- no insulin is required for 48-72 hours post-partum in most type 1 diabetes
- monitor glucose q6h, restart insulin at two-thirds pre-pregnancy dosage when glucose > 8 mmol/L

GESTATIONAL DIABETES MELLITUS (GDM)

❑ glucose intolerance that is present only during pregnancy
❑ 50% risk of developing Type 2 DM in next 20 years
❑ varies from 12% in racially heterogenous urban regions to 1% in rural areas with a predominantly white population

Risk Factors
- ❑ maternal: age > 25, obesity, excess weight gain in pregnancy, early PIH or polyhydramnios, repeated vaginal candidiasis, high risk ethnic groups
- ❑ past history: previous GDM, congenital anomaly, unexplained stillbirth, macrosomic infant (> 4.5 kg)
- ❑ family history: GDM, Type 2 DM, macrosomic infant
- ❑ fetal: wt > 4,500 g or large gestational age (LGA), multiple gestation

Diagnosis
- ❑ screen all women between 24-28 weeks with 50 g oral glucose challenge test (OGCT)
- ❑ > 7.8 mmol/L (140 mg/dL) at 1 hour is abnormal
- ❑ ≥ 10.3 mmol/L (200 mg/dL) at 1 hour is diagnostic of GDM
- ❑ if OGCT result is 7.8-10.3, follow-up with 2 hour 75 g oral glucose tolerance test (OGTT)
    - GDM diagnosis with 2 or more of: blood glucose before test ≥ 5.3, blood glucose at 1 hour ≥ 10.6 or 2 hour blood glucose ≥ 8.9

Management
- ❑ aim to achieve normal blood sugars post-prandially
- ❑ start with diabetic diet (note: weight-reducing diets not recommended due to risk of ketonemia)
- ❑ if blood sugars not well-controlled with diet alone, may need to add insulin
- ❑ oral hypoglycemic agents contraindicated in pregnancy
- ❑ fetal monitoring and timing of delivery same as for DM above
- ❑ insulin and diabetic diet should be stopped post-partum but encourage regular exercise and attainment of healthy body weight as there is increased risk of overt DM
- ❑ follow-up testing recommended (i.e. OGTT at 6 weeks and 6 months postpartum)

Complications
- ❑ maternal: polyhydramnios, pre-eclampsia, diabetic emergencies
- ❑ fetal: macrosomia with traumatic delivery, delayed organ maturity, congenital anomalies, IUGR

HYPERTENSIVE DISORDERS OF PREGNANCY

Classification
- ❑ A: preeclampsia/eclampsia/pregnancy-induced hypertension (incidence = 6%)
- ❑ B: chronic hypertension (incidence = 2.5%)
- ❑ C: chronic hypertension with superimposed preeclampsia/eclampsia
- ❑ D: transient hypertension

A. PREGNANCY-INDUCED HYPERTENSION/ PREECLAMPSIA/TOXEMIA/ECLAMPSIA (PIH)
- ❑ hypertension accompanied by proteinuria and/or non-dependent edema with onset > 20 weeks
    - systolic BP > 140 mmHg or at least 30 mmHg above non-pregnant/T1 sBP
    - diastolic BP > 90 mmHg or at least 15 mmHg above non-pregnant/T1 dBP
    - proteinuria is defined as > 1+ protein on random dipstick analysis or > 300 mg in a 24 hour urine collection
    - non-dependent edema (e.g. face, hands) that is generalized and usually associated with excessive weight gain (> 2 kg/week); this criterion is clinically useful but no longer strictly part of the PIH definition
- ❑ 50% of all hypertension in pregnancy
- ❑ due to an imbalance of thromboxane (vasoconstrictor) and prostaglandin (vasodilator), causing generalized arteriolar constriction —> capillary damage —> protein extravasation and hemorrhage
- ❑ caution: patients with GTN (mole or choriocarcinoma) may present with classic features of PIH in T1 or T2

Classification
- ❑ mild PIH: uncomplicated by neurologic symptoms or criteria for a diagnosis of severe PIH

Severe PIH

❑ severe PIH: complicated by at least two of the following
- BP > 160/110
- respiratory: pulmonary edema or cyanosis
- cardiac: congestive heart failure
- renal: proteinuria > 5 g/24 hours or > 2+ on dipstick, elevated serum creatinine, oliguria (< 400 mL/24 hours)
- hepatic: elevated liver enzymes, right upper quadrant (RUQ) or epigastric pain (subcapsular hemorrhage), ascites, hyperbilirubinemia, HELLP
- neurologic: visual disturvances (i.e. scotomas, loss or peripheral vision), hyperreflexia, clonus, headache (cerebral artery vasospasm), convulsions (eclampsia)
- gastrointestinal: severe nausea/vomiting
- hematologic: thrombocytopenia, microangiopathic hemolysis
- fetal: IUGR

❑ eclampsia: grand mal seizures in a woman with preeclampsia

Risk Factors for Development of PIH

❑ maternal factors
- 80-90% of cases in primagravidas
- past history or family history of PIH
- diabetes, chronic hypertension, or renal disease
- antiphospholipid antibody syndrome
- extremes of maternal age

❑ fetal factors
- IUGR
- hydatidiform mole
- > 1 fetus
- fetal hydrops

Prophylaxis (For Those with Risk Factors)

❑ calcium supplementation, aspirin, and fish oil supplementation have all been investigated as prophylactic treatments of PIH; results vary and are uncertain

Management of Mild PIH

❑ maternal evaluation
- history and physical examination (see above criteria)
- laboratory
  - CBC and electrolytes
  - renal function tests: BUN, creatinine, uric acid
  - liver enzymes and coagulation studies: PT, PTT, FDP, d-dimers
  - urinalysis for protein and casts
  - 24 hour urine for protein and creatinine clearance

❑ fetal evaluation of FHR, NST, BPP, doppler flow

❑ management with bed rest in left lateral decubitus position (reduces abdominal vessel compression)

❑ normal dietary salt and protein intake

❑ no use of diuretics/antihypertensives

Management of Severe PIH

❑ stabilize and deliver; the only "cure" is delivery

❑ vaginal induction of delivery is preferred unless patient or fetus is seriously ill, then C/S

❑ admit and complete maternal evaluation (same as for mild)
- keep NPO
- start IV, cross and type
- foley catheter to monitor urine output

❑ maternal monitoring
- hourly input and output, check urine q12 hours for protein
- vitals and DTR q1 hour

❑ fetal evaluation
- NST followed by continuous electronic fetal monitoring until delivery; doppler flow

❑ anticonvulsant therapy
- given to increase seizure threshold
- baseline magnesium blood level
- magnesium sulfate (4 g IV bolus over 20 min) followed by maintenance of 2-4 g/hour
- excretion of magnesium sulfate is via kidney therefore patients with oliguria require a lower infusion rate
- signs of magnesium toxicity: depressed deep tendon reflexes (DTR), RR < 10/minute, urine output < 25 cc/hr, decreased muscle tone, CNS or cardiac depression
- antagonist to magnesium sulfate is calcium gluconate (10%) 10 mL (1 g) IV over 2 minutes; may require mechanical ventilation if respiratory arrest occurs

❑ antihypertensive therapy
- decreasing the BP decreases the risk of stroke (indicated only if BP > 140-170/90-110)
- first line: hydralazine 5-10 mg IV bolus over 5 minutes q15-30 minutes until desired effect (an arteriolar vasodilator with minimal venous effect; short-term only)
- second line: labetalol 20-50 mg IV q10 minutes
- third line: nifedipine (Adalat) 10-20 mg po q20-60 minutes
- ACE-inhibitors should be avoided

❑ postpartum management
- all antepartum therapy and monitoring continued until stable
- risk of seizure highest in first 24 hours postpartum
- continue magnesium sulfate for 12-24 hours after delivery
- the patient who continues to remain in serious condition may have HELLP
- most women return to a normotensive BP within 2 weeks but BP may worsen transiently in that time

Management of Eclampsia
❑ airway, breathing, circulation
❑ seizure control and prevention (see Neurology Chapter)
- do not attempt to shorten or abolish the initial convulsion
- prevent maternal injury and maintain adequate oxygenation
- minimize risk of aspiration, auscultate lungs after every seizure
- give adequate magnesium sulphate as soon as convulsion has ended
- correct maternal acidemia (obtain post-ictal blood gases)
- some use valium for seizure control

Maternal Complications of PIH
❑ cerebral hemorrhage (50% of deaths)
❑ left ventricular failure/pulmonary edema
❑ liver and renal dysfunction
❑ abruption
❑ seizures
❑ DIC: release of placental thromboplastin, leading to a consumptive coagulopathy
❑ HELLP
- hemolysis, elevated liver enzymes, low platelets
- may only respond to fresh frozen plasma with plasma exchange

Fetal Complications of PIH
❑ mainly due to placental insufficiency
- fetal loss
- IUGR
- prematurity
- abruptio placentae

B. CHRONIC HYPERTENSION
❑ definition
- history of hypertension (> 140/90) before gestation or prior to 20 weeks gestation (unless there is a GTN)
- persistence of hypertension postpartum
- strong family history of hypertension
- most gravidas have essential hypertension, associated with an increased risk of preeclampsia or eclampsia, abruptio placenta, IUGR and IUD

❑ management
- methyldopa and/or labetalol and/or atenolol
- no ACE inhibitors, diuretics, propranolol (risk of teratogenic effects)
- monitor fetus with serial U/S

C. CHRONIC HYPERTENSION WITH SUPERIMPOSED PIH
❑ 2-7 fold increased likelihood of developing preeclampsia/eclampsia if pre-existing maternal hypertension
❑ tends to recur
❑ occurs early in pregnancy, tends to be severe, often with IUGR

D. TRANSIENT HYPERTENSION
❑ occurs in the second half of pregnancy, during labour, or 48 hours after delivery
❑ no significant proteinuria (< 300 mg/24 hours)
❑ extremely difficult to differentiate from preeclampsia (retrospective diagnosis)
❑ often predictive of later development of essential hypertension

HYPEREMESIS GRAVIDARUM

Definition
- ❑ intractable nausea and vomiting, causing weight loss, dehydration, ketonuria, electrolyte imbalance, acid-base disturbance and if severe, hepatic and renal damage
- ❑ usually present in T1 then diminishes; persists throughout pregnancy in a minority

Etiology
- ❑ presently thought to be multifactorial with hormonal, immunologic and psychologic components
- ❑ high or rapidly rising βhCG or estrogen levels are implicated

Differential Diagnosis of Nausea and Vomiting
- ❑ hyperemesis is a diagnosis of exclusion
- ❑ GI inflammation/infection
  - appendicitis
  - cholecystitis
  - hepatitis
  - gastroenteritis
  - pancreatitis
  - peptic ulcer disease (PUD)
  - fatty liver of pregnancy
- ❑ pyelonephritis
- ❑ thyrotoxicosis
- ❑ multiple gestation
- ❑ gestational trophoblastic neoplasia (GTN) (see Gynecology Chapter)
- ❑ HELLP syndrome

Investigations
- ❑ labs (CBC, lytes, BUN and creatinine, urinalysis, LFTs)
- ❑ ultrasound (to R/O molar pregnancy, multiple pregnancy and to assess liver, pancreas, gallbladder, etc.)

Management
- ❑ general
  - early recognition is important
  - if severe, admit to hospital
  - NPO initially, then small frequent meals of appealing foods
  - correct hypovolemia, electrolyte imbalance and ketosis
  - thiamine, if indicated
  - TPN if severe to reverse catabolic state
  - consider emotional support, dietary and psychologic counselling
- ❑ pharmacological options
  - Diclectin (vitamin B6 and doxylamine succinate)
- ❑ non-pharmacological options
  - accupressure at inner aspect of the wrists, just proximal to the flexor crease has been shown to significantly reduce symptoms of nausea and vomiting
  - avoid triggers (i.e. certain smells)
  - rest

Maternal Complications
- ❑ Mallory Weiss tears
- ❑ Wernicke's encephalopathy, if protracted course
- ❑ death

Fetal Complications
- ❑ usually none
- ❑ IUGR is 15x more common in women losing > 5% of prepregnant weight

ISOIMMUNIZATION

Etiology
- ❑ antibodies produced against a specific RBC antigen as a result of antigenic stimulation with RBC of another individual
- ❑ most common is anti-Rh Ab produced by a sensitized Rh-negative mother (more than 90% of cases of Rh isoimmunization are due to D antigens)
- ❑ other antibodies can lead to fetal red blood cell hemolysis
  - much less common and no prophylaxis is available
- ❑ overall risk of isoimmunization of an Rh-negative mother with a Rh-positive ABO-compatible infant is 16% (2% of reactions will occur antepartum, 7% within 6 months of delivery, and the remainder 7% in the second pregnancy)

Pathophysiology
- ❑ maternal-fetal circulation normally separated by placental barrier
- ❑ upon first exposure, initially IgM and then IgG antibodies are produced
- ❑ sensitization routes
    - incompatible blood transfusion
    - previous fetal-maternal transplacental hemorrhage
    - invasive procedure while pregnant
    - therapeutic abortion, D&C, amniocentesis
    - during labour and delivery

Diagnosis
- ❑ routine screening at first visit for blood group, Rh status, antibodies
- ❑ Ab titres < 1:16 considered benign
- ❑ Ab titres > 1:16 necessitates amniocentesis (correlation exists between amount of biliary pigment in amniotic fluid and severity of fetal anemia) from 27 weeks onwards
- ❑ Liley curve is used to determine bilirubin level and appropriate management
- ❑ Kleihauer-Betke test can be used to determine extent of fetomaternal hemorrhage
    - fetal red blood cells are identified on a slide treated with citrate phosphate buffer
    - adult hemoglobin is more readily eluted through cell membrane in presence of acid
- ❑ detailed U/S for fetal hydrops

Prophylaxis
- ❑ Rhogam binds to Rh Ag of fetus and prevents it from contacting maternal immune system
- ❑ Rhogam (300 µg) must be given to all Rh negative women
    - at 28 weeks
    - within 72 hours of the birth of an Rh positive fetus
    - with a positive Kleihauer-Betke test
    - with any invasive procedure in pregnancy
    - in ectopic pregnancy
    - with miscarriage, therapeutic abortion (50 ug)
    - with an antepartum hemorrhage
- ❑ if Rh neg and Ab screen positive, follow mother with serial monthly Ab titres throughout pregnancy +/– serial amniocentesis as needed (Rhogam of no benefit)

Management
- ❑ falling biliary pigment warrants no intervention (usually indicative of fetus which is unaffected or mildly affected)
- ❑ rising or stable biliary pigment on serial amniocentesis must be compared to a standard table which is divided into 3 zones based on severity of hemolysis (Liley Curve)
- ❑ cordocentesis for fetal Hb; should be used cautiously, not first line
- ❑ intrauterine transfusion of O-negative packed red blood cells 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, and ascites
- ❑ severe cases can lead to fetal hydrops (total body edema), or erythroblastosis fetalis

INFECTIONS DURING PREGNANCY
- ❑ see table 6

Table 6. Infections During Pregnancy * indicates **TORCH** infection

Infection	Agent	Source of Transmission	Greatest Transmission Risk	Effects on Fetus	Effects on Mother	Diagnosis	Treatment
***Toxoplasmosis**	Protozoa (Toxoplasma gondii)	Raw meat, unpasteurized goat's milk, cat feces/urine	T3 (but most severe if infected in T1); only concern if primary infection during pregnancy	Congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial cacification, MR, microcephaly) NB: 75% initially asymptomatic at birth	Majority subclinical; may have flu-like symptoms	IgM and IgG serology confirm with IgM in cord blood	Self-limiting in mother; spiramycin decreases fetal morbidity
***Rubella**	ssRNA togavirus	Respiratory droplets (highly contagious)	T1	SA or congenital rubella syndrome (hearing loss, cataracts, CV lesions, MR, IUGR, hepatitis, CNS defects, osseous changes)	Rash (50%), fever, posterior auricular or suboccipital 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; should offer vaccine following pregnancy (but avoid during pregnancy as it is live attenuated vaccine)
***CMV**	DNA virus (herpes family)	Blood/organ transfusion, sexual contact, breast milk, transplacental, during delivery	T1-T3	5-10% develop CNS involvement (MR, 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
***Herpes**	DNA virus	Intimate mucocutaneous contact	C/S delivery (if genital lesions present); less commonly in utero	Disseminated herpes (20%); CNS sequelae (35%); self-limited infection	Painful vesicular lesions	Clinical diagnosis	C/S if active genital lesions, even if remote from vulva
Syphilis	Treponema pallidum	Transplacental	T1-T3	Risk of preterm labour, multisystem involvement, fetal death	See Infectious Disease Chapter	VDRL screening for all pregnancies; confirm with TPHA or FTA-ABS	Pen G 2.4 M units IM; monitor VDRL monthly
Hepatitis B	DNA virus	Blood, saliva, semen, vaginal secretions, breast milk, transplacental	T3 10% vertical transmission if asymptomatic HBsAg +ve; 85-90% if HBsAg and HBcAg +ve	Prematurity, low birth weight, neonatal death	Fever, N/V, fatigue, jaundice, increased LFTs	Serologic screening for all pregnancies	Rx neonate with HBIG and vaccine (at birth, 1, 6 mo); 90% effective
Erythema Infectiosum (Fifth Disease)	Parvovirus B19	Respiratory, infected blood products, transplacental	10-20 weeks GA	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
HIV	Retrovirus	Intimate contact with body secretions, blood products, vertical (12-28%)	Greatest risk during L&D; also transplacental	Vertical transmission, IUGR, PTL, PROM	See Infectious Disease Chapter	All pregnancies offered screening	AZT decreases vertical transmission (25 -> 8%)
Chicken Pox	Varicella zoster virus (herpes family)	Direct, respiratory	13-30 weeks GA, and 5d pre to 2d post delivery	Congenital varicella syndrome (limb aplasia, chorioretinitis, cataracts, cutaneous scars, cortical atrophy, IUGR, hydrops), PTL	Fever, malaise, vesicular pruritic lesions	Clinical, +/- vesicle fluid culture, +/– serology	Note: do not administer vaccine during pregnancy (live attenuated)

ANTENATAL HEMORRHAGE

FIRST AND SECOND TRIMESTER BLEEDING

Etiology

❑ abortion (threatened, inevitable, incomplete, complete)
- < 5% of threatened abortions go on to abort (see Table 7)

❑ abnormal pregnancy (ectopic, molar) (see Gynecology Chapter)
❑ trauma (post-coital)
❑ physiologic bleeding (due to placental development)
❑ genital lesion (e.g. cervical polyp, neoplasms)

Table 7. Types of Abortions

Type	History	Cervix	Management*
Threatened	Vaginal bleeding +/– cramps	Closed - intact membranes	U/S shows viable fetus
Inevitable	Bleeding + cramps +/– ruptured membranes	Open > 2 cm	D&C +/– oxytocin
Incomplete	Heaviest bleeding + cramps; soft abdomen; may have passage of tissue	Open	D&C +/– oxytocin
Complete	Bleeding + complete sac and placenta passed	Open	No D&C
Missed	Fetal death and retention of products; presents as pregnancy not progressing	Closed	D&C +/– oxytocin
Habitual	3 or more consecutive spontaneous abortions		Evaluate environmental factors (smoking, alcohol, heavy caffeine), uterine anatomy, karyotype of both parents, TSH, antiphospholipid antibodies (including lupus anticoagulant and anticardiolipin antibodies)
Therapeutic	For genetic, medical, and psychological reasons		See below
Septic	Contents of uterus infected before, during or after abortion		D&C IV wide spectrum antibiotics Oxygen

* Rhogam to all Rh negative mothers

THERAPEUTIC ABORTIONS

❑ medical management
- < 9 weeks use methotrexate plus misoprostol
- > 12 weeks use prostaglandins intra- or extra-amniotically, or IM

❑ surgical management
- < 12-16 weeks use dilatation and curettage
- > 16 weeks use dilatation and evacuation

❑ complications
- pain, bleeding, low-grade fever
- perforation of uterus
- hemorrhage
- laceration of cervix
- risk of sterility
- infection/ endometritis - usually due to retained products
- Asherman syndrome (fibrosis of the uterus)

THIRD TRIMESTER BLEEDING

Etiology
- ❑ placenta previa
- ❑ abruptio placentae
- ❑ vasa previa
- ❑ bloody show (shedding of cervical mucous plug)
- ❑ marginal sinus bleeding
- ❑ cervical lesion (cervicitis, polyp, ectropion, cervical cancer)
- ❑ uterine rupture
- ❑ other: bleeding from bowel or bladder, placenta accreta, abnormal coagulation

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

PLACENTA PREVIA (see Colour Atlas OB2)

Definition
- ❑ abnormal location of the placenta at or near the internal cervical os
- ❑ many are low lying in early pregnancy but due to development of lower uterine segment appear to "move upward" as pregnancy nears term
- ❑ total: placenta completely covers the internal os
- ❑ partial: placenta partially covers the internal os
- ❑ marginal: within 2 cm of os but does not cover any part of os
- ❑ low lying (NOT a previa): placenta in lower segment but clear of os
    - can also bleed, usually later (i.e. in labour)

Etiology
- ❑ incidence = 1/200 at time of delivery
- ❑ 90-95% of previas diagnosed in T2 resolve by T3 (repeat U/S at 30-32 weeks to follow migration)
- ❑ associated conditions and risk factors
    - history of placenta previa (4-8% recurrence risk)
    - multiparity
    - increased maternal age
    - multiple pregnancy
    - uterine tumour (e.g. fibroids) or other uterine anomalies
    - uterine scar due to previous abortion, C/S, D&C, myomectomy

Presentation
- ❑ recurrent, PAINLESS bright red vaginal bleeding
    - onset of bleeding depends on degree of previa (i.e. complete bleed earlier)
    - mean GA is 30 weeks; one third present earlier
    - initially, bleeding may be minimal and cease spontaneously but can be catastrophic later
    - bleeding at onset of labour can occur with marginal placenta previa
- ❑ uterus soft and non-tender
- ❑ presenting part high or displaced

Investigations
- ❑ ultrasound (transabdominal ultrasound has 95% accuracy)

Management
- ❑ stabilize and monitor
    - maternal stabilization; large bore IV with hydration
    - electronic fetal monitoring
    - maternal monitoring: vitals, urine output, blood loss, bloodwork (hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match)
- ❑ U/S assessment
    - when fetal and maternal condition permit, perform careful U/S examination to determine fetal viability, gestational age and placental status/position
- ❑ manage
    - management decision depends on
        - previa characteristics (amount of bleeding, degree of previa)
        - fetal condition (GA, level of distress, presentation)
        - uterine activity
    - Rhogam if mother is Rh negative
    - expectant management and observation of mother and fetus if the initial bleeding episode is slight and GA < 37 weeks
        - admit to hospital
        - limited physical activity
        - no douches, enemas, or sexual intercourse
        - consider corticosteriods for fetal lung maturity
        - delivery when fetus is mature or hemorrhage dictates
    - C/S delivery if bleeding is profuse, GA > 36 weeks, or L/S ratio is 2:1 or greater (incision site dictated by location of previa)

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)
- post partum hemorrhage (because lower uterine segment is atonic)
- hysterectomy
- placenta accreta

ABRUPTIO PLACENTAE

Definition
❑ premature separation of a normally implanted placenta after 20 weeks gestation
❑ 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

❑ incidence: 1/100 deliveries

Associated Conditions
❑ previous abruption (recurrence rate 10%)
❑ maternal hypertension (chronic or PIH) in 50% of abruptions
❑ maternal vascular disease
❑ cigarette smoking
❑ excessive alcohol consumption
❑ cocaine
❑ multiparity
❑ maternal age > 35 (felt to reflect parity)
❑ PPROM
❑ uterine distension (polyhydramnios, multiple gestation)
❑ sudden decompression of the uterus (twins)
❑ uterine anomaly, fibroids
❑ trauma (e.g. motor vehicle collision, maternal battering)

Presentation
❑ classically: PAINFUL vaginal bleeding, uterine tenderness, uterine contractions
❑ pain: sudden onset, constant, localized to lower back and uterus
❑ +/– fetal distress, fetal demise (15% present with demise), bloody amniotic fluid

Diagnosis
❑ clinical diagnosis
❑ ultrasound not sensitive for abruption (sensitivity = 15%)

Table 8. Grades of Abruptio Placentae

Grade	Uterine Irritability	Maternal Hemodynamics	Maternal Fibrinogen	FHR
Mild	Mild	Normal	Normal	Normal
Moderate	Moderate-severe +/- tetany	BP normal but with postural drop Increased HR	↓	Distress: loss of variability, late decels
Severe	Tetany	Decreased BP Increased HR	↓ ↓	Fetal death

Management
- ❑ stabilize and monitor
  - maternal stabilization, IV hydration
  - fetal monitoring
  - monitor maternal vitals, urine output
  - blood for hemoglobin, platelets, PT/PTT, fibrinogen, FDP, cross and type
  - blood products on hand (red cells, platelets, cryoprecipitate) because of DIC risk
  - Rhogam if Rh negative
- ❑ mild abruption and GA < 36 weeks
  - close observation of fetal well-being and amount of bleeding
  - limited physical activity
  - serial Hct to assess concealed bleeding
  - delivery when fetus is mature or when hemorrhage dictates
- ❑ mild abruption and GA > 36 weeks
  - stabilization and delivery
- ❑ moderate to severe abruption
  - hydrate and restore blood loss and correct coagulation defect if present
  - vaginal delivery if no evidence of fetal or maternal distress and if cephalic presentation OR with dead fetus
  - labour must progress actively
- ❑ severe abruption and live fetus
  - C/S if fetal or maternal distress develops with fluid/blood replacement, labour fails to progress or non-cephalic fetal presentation

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

VASA PREVIA

Definition
- ❑ velamentous insertion of cord into membranes of placenta; unprotected fetal vessels pass over the cervical os
- ❑ 1 in 5,000 deliveries

Presentation
- ❑ painless vaginal bleeding and fetal distress (tachy- to bradyarrhythmia)

Investigations
- ❑ Apt test (NaOH mixed with the blood) can be done immediately to determine if the source of the 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
- ❑ emergent C/S

Complications
- ❑ 50% perinatal mortality, increasing to 75% if membranes rupture (most infants die of exsanguination)
- ❑ since bleeding is from fetus, a small amount of blood loss can have catastrophic consequences

GROWTH DISCREPANCIES

INTRA-UTERINE GROWTH RESTRICTION (IUGR)

Definition
- ❑ infants whose weight is < 10th percentile for a particular GA
- ❑ weight not associated with any constitutional or familial cause

Etiology
- ❑ maternal causes
  - poor nutrition, cigarette smoking, drug abuse, alcoholism, cyanotic heart disease, severe DM, SLE, pulmonary insufficiency
- ❑ maternal-fetal
  - any disease which causes placental insufficiency leading to inadequate transfer of substrate across the placenta
  - includes PIH, chronic HTN, chronic renal disease, gross placental morphological abnormalities (infarction, hemangiomas)
- ❑ fetal causes
  - TORCH infections, multiple gestation, congenital anomalies

Presentation
- ❑ symmetric/Type I (20%)
  - occurs early in pregnancy
  - inadequate growth of head and body although head:abdomen ratio may be normal
  - usually associated with congenital anomalies or TORCH
- ❑ asymmetric/Type II (80%)
  - occurs late in pregnancy
  - brain is spared, therefore the head:abdomen ratio is increased
  - usually associated with placental insufficiency
  - more favorable prognosis than Type I

Investigations
- ❑ SFH measurements at every antepartum visit
- ❑ more thorough assessment if mother is in high risk category or if SFH lags > 2 cm behind GA
- ❑ U/S exam should include assessment of BPD, head and abdomen circumference, head:body ratio, femur length and fetal weight
- ❑ doppler analysis of umbilical cord blood flow

Management
- ❑ most important consideration is accurate menstrual history and GA
- ❑ prevention via risk modification prior to pregnancy ideal
- ❑ modify controllable factors: smoking, alcohol, nutrition
- ❑ bed rest (in LLD position)
- ❑ serial BPP (monitor fetal growth)
- ❑ delivery when extrauterine existence is less dangerous than continued intrauterine existence or if GA > 34 weeks with significant oligohydramnios
- ❑ liberal use of C/S since IUGR fetus withstands labour poorly

Complications
- ❑ prone to meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation
- ❑ greater risk of perinatal morbidity and mortality

MACROSOMIA

Definition
- ❑ fetal weight > 90th percentile for GA, or > 4,000 grams

Etiology
- ❑ risk factors
  - obesity
  - diabetes mellitus
  - past history of macrosomic infant
  - prolonged gestation
  - multiparity

Investigations
- ❑ serial examination (SFH)
- ❑ investigations (U/S)
- ❑ U/S predictors: polyhydramnios, T3 AC growth > 1.5 cm/week, HC/AC ratio < 10th percentile, FL/AC ratio < 20th percentile

Management
- ❑ C/S often safer than vaginal delivery

Complications
- ❑ increased risk of perinatal mortality
- ❑ fetopelvic disproportion and birth injuries (shoulder dystocia, fetal bone fracture) more common
- ❑ complications of DM in labour (see Medical Conditions in Pregnancy section)

POLYHYDRAMNIOS

Definition
- ❑ amniotic fluid volume > 2,000 cc at any stage in pregnancy
- ❑ > 8 cm x 8 cm (3.1 x 3.1 in) pocket on U/S

Etiology
- ❑ incidence: 1/250 deliveries
- ❑ idiopathic: most common (40%)
- ❑ maternal
  - Type 1 diabetes: causes abnormalities of transchorionic flow
- ❑ maternal-fetal
  - chorioangiomas
  - multiple gestation
  - erythroblastosis
- ❑ fetal
  - chromosomal anomaly (up to 2/3 of fetuses with severe polyhydramnios)
  - respiratory - cystic adenomatoid malformed lung
  - CNS (anencephaly, hydrocephalus, meningocele)
  - GI (tracheoesophageal fistula, duodenal atresia)
  - facial clefts, neck masses (interfere with swallowing)

Presentation
- ❑ pressure symptoms from overdistended uterus (dyspnea, edema, hydronephrosis)
- ❑ uterus large for dates, difficulty palpating fetal parts and hearing fetal heart tones

Management
- ❑ determine cause
  - complete fetal U/S evaluation
  - screen for maternal disease/infection as indicated
- ❑ management depends on severity
  - mild to moderate cases require no treatment
  - if severe, hospitalize and consider therapeutic amniocentesis

Complications
- ❑ cord prolapse
- ❑ placental abruption
- ❑ malpresentation
- ❑ preterm labour
- ❑ uterine dysfunction and postpartum hemorrhage (PPH)
- ❑ increased perinatal mortality rate

OLIGOHYDRAMNIOS

Definition
- ❑ amniotic fluid index of 5 cm (2 in) or less

Etiology
- ❑ early onset oligohydramnios
  - decreased production
    - renal agenesis or dysplasia, urinary obstruction, posterior urethral valves (male)
    - poor placental perfusion
  - increased loss
    - prolonged amniotic fluid leak (although most often labour ensues)
    - 15-25% of cases have fetal anomalies
- ❑ late pregnancy onset oligohydramnios
  - amniotic fluid normally decreases after 35 weeks
  - common in post-term pregnancies
  - may be a marker for infants who may not tolerate labour well

Management
- ❑ oligohydramnios is an important sign of chronic placental insufficiency and always warrants admission and investigation
  - rule out ROM
  - fetal monitoring (NST, CTG, BPP)
  - consider delivery if at term

Complications
- ❑ cord compression
- ❑ T1 onset
  - Potter's facies
  - limb deformities
  - abdominal wall defects
- ❑ onset at > 20 weeks
  - pulmonary hypoplasia

ANTENATAL COMPLICATIONS

PRETERM LABOUR

Definition
- ❑ labour occurring between 20 and 37 weeks gestation
- ❑ preterm labour complicates about 10% of pregnancies

Etiology
- ❑ idiopathic (most common)
- ❑ maternal
  - prior history of premature delivery (recurrence risk of 17-40%)
  - history of abortions or stillbirths
  - maternal age < 18 or > 40 years
  - infection
    - recurrent pyelonephritis and untreated bacteriuria
    - maternal genital tract infection
    - chorioamnionitis
  - medical illness
    - preeclampsia/hypertension
    - uncontrolled diabetes
    - other medical illness (heart disease, renal disease, severe anemia, systemic infection, chronic vascular disease)
  - mechanical
    - fibroids or other uterine anomalies
    - incompetent cervix
  - previous incision into uterus or cervix (C/S, conization)
  - surgical
    - intra-abdominal surgery, cholecystitis, peritonitis
  - social
    - low socioeconomic status
    - lack of prenatal care
    - poor nutrition
    - low prepregnancy weight
    - smoking
    - drug addiction (alcohol, cocaine)
    - stress/anxiety/fatigue
- ❑ maternal-fetal
  - PPROM (a common cause)
  - polyhydramnios
  - placenta previa or abruption
- ❑ fetal
  - multiple gestation
  - congenital abnormalities of fetus

Presentation
- ❑ regular contractions (2 in 10 minutes)
- ❑ cervix > 2 cm dilated or 80% effaced OR documented change in cervix

Management
- ❑ initial
  - transfer to appropriate facility
  - hydration (NS @ 150 mL/hour)
  - bed rest in left lateral decubitus position
  - sedation (morphine)
  - avoid repeated pelvic exams (increased infection risk)
  - U/S examination of fetus (for GA, BPP, position)
  - prophylactic antibiotics; controversial but may help delay delivery, also important to consider PPROM
- ❑ aggressiveness depends on the GA
- ❑ tocolytic agents - if no contraindications present; agent used depends on clinical situation
  - do not inhibit preterm labour completely, but may buy time to allow celestone use or to transfer to appropriate centre
  - beta-mimetics: ritodrine, terbutaline
  - magnesium sulfate (if diabetes or cardiovascular disease present)
  - calcium channel blockers: nifedipine
  - prostaglandin (PG) synthesis inhibitors (2nd line agent): indomethacin
- ❑ ensure availability of necessary personnel and equipment to assess mother and fetus during labour and care for baby of the predicted GA if therapy fails

Requirements for Consideration of Labour Suppression (Tocolysis)
- ❑ live fetus
- ❑ fetal immaturity
- ❑ intact membranes
- ❑ cervical dilatation of 4 cm or less
- ❑ absence of maternal or fetal contraindications (see below)

Maternal Contraindications to Tocolysis
- ❑ bleeding (placenta previa or abruption)
- ❑ maternal disease (hypertension, diabetes, heart disease)
- ❑ preeclampsia or eclampsia
- ❑ chorioamnionitis

Fetal Contraindications to Tocolysis
- ❑ erythroblastosis fetalis
- ❑ severe congenital anomalies
- ❑ fetal distress/demise
- ❑ IUGR, multiple gestation (relative)

Enhancement of Pulmonary Maturity
- ❑ betamethasone valerate (Celestone) 12 mg IM q24h x 2
- ❑ most effective between 28 and 34 weeks gestation
- ❑ specific maternal contraindications: active TB, viral keratosis, maternal DM

Complications of Premature Labour
- ❑ prematurity is the leading cause of perinatal morbidity and mortality
  - at 30 weeks or 1,500 g (3.3 lbs) = 90% survival
  - at 33 weeks or 2,000 g (4.4 lbs) = 99% survival
- ❑ major causes of morbidity = asphyxia, sepsis, respiratory distress syndrome (RDS)
- ❑ intrapartum asphyxia may lead to cerebral hemorrhage

Prevention of Preterm Labour
- ❑ good prenatal care
- ❑ identify pregnancies at risk
- ❑ treat silent vaginal infection or UTI
- ❑ patient education
- ❑ the following may help but evidence for their effectiveness is lacking
  - rest, time off work, stress reduction
  - improved nutrition
  - U/S measurement of cervical length or frequent vaginal exams to assess cervix; this would catch PTL earlier so tocolysis would be more effective

RUPTURE OF MEMBRANES

Premature ROM (PROM)
- ❑ rupture of membranes prior to the onset of labour at any GA

Prolonged ROM
- ❑ if 24 hours elapse between rupture of membranes and onset of labour

Preterm ROM
- ❑ ROM occurring before 37 weeks gestation (associated with PTL)

Preterm Premature ROM (PPROM)
- ❑ rupture of membranes before 37 weeks AND prior to onset of labour

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

Presentation
❑ 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")

❑ amniotic fluid turns nitrazine paper blue (low specificity as can be blood, urine or semen)
❑ ferning (high salt content of amniotic fluid evaporates and looks like ferns under microscope)
❑ U/S to R/O fetal anomalies, assess GA and amniotic fluid volume

Management
❑ avoid introducing infection with examinations (do not do a digital pelvic exam)
❑ cultures (cervix for GC, lower vagina for GBS)
❑ dependent upon gestational age; must weigh degree of prematurity vs risk of amnionitis and sepsis by remaining in utero
- < 24 weeks consider termination (poor outlook due to pulmonary hypoplasia)
- 26-34 weeks: expectant management as prematurity complications are significant
- 34-36 weeks: "grey zone" where risk of death from RDS and neonatal sepsis is the same
- > 36 weeks: induction of labour since the risk of death from sepsis is greater than RDS

❑ assess fetal lung maturity by L/S ratio of amniotic fluid
❑ consider administration of betamethasone valerate (Celestone) to accelerate maturity
❑ if not in labour or labour not indicated, consider antibiotics (controversial)
❑ admit and monitor vitals q4h, daily BPP and WBC count

Complications
❑ cord prolapse
❑ intrauterine infection (chorioamnionitis)
❑ premature delivery

CHORIOAMNIONITIS

Definition
❑ infection of the chorion, amnion and amniotic fluid

Etiology/Risk Factors
❑ prolonged ROM
❑ long labour
❑ multiple vaginal exams during labour
❑ internal monitoring
❑ bacterial vaginosis and other vaginal infections

Presentation
❑ maternal fever, maternal or fetal tachycardia, uterine tenderness, foul cervical discharge leukocytosis, presence of leukocytes or bacteria in amniotic fluid

Management
❑ blood and amniotic fluid cultures
❑ IV antibiotics (ampicillin and gentamycin)
❑ expedient delivery regardless of gestational age

POST-DATE PREGNANCY

Definition
❑ pregnancy beyond 42 weeks
❑ accurate dating essential
❑ 10% of pregnancies

Management
❑ fetal movement count by the mother
❑ BPP twice weekly from 40 weeks (if BPP < 10/10 at any time, deliver)
❑ deliver after 41 weeks (induction or C/S) if not already in labour since perinatal mortality is higher secondary to progressive uteroplacental insufficiency

Complications
- ❑ perinatal mortality 2-3x higher
- ❑ oligohydramnios
- ❑ meconium passage: risk of meconium aspiration
- ❑ asphyxia
- ❑ macrosomia
- ❑ placental insufficiency; infarction of aging placenta
- ❑ postmaturity syndrome:10-20% of post-term pregnancies (fetal weight loss, reduction in subcutaneous fat, scaling, dry skin from placental insufficiency)
- ❑ morbidity increased with hypertension/PET, DM, abruption, IUGR and multiple gestation

INTRAUTERINE FETAL DEATH
- ❑ 1% of pregnancies

Etiology
- ❑ unknown in 50%
- ❑ hypertension, DM
- ❑ erythroblastosis fetalis
- ❑ congenital anomalies
- ❑ umbilical cord or placental complications
- ❑ intrauterine infection
- ❑ antiphospholipid Abx

Presentation
- ❑ decreased perception of fetal movement by mother
- ❑ SFH and maternal weight not increasing
- ❑ absent fetal heart tones (not diagnostic)
- ❑ absent cardiac activity and fetal movement on U/S required for diagnosis
- ❑ high MSAFP

Investigations (To Determine Cause)
- ❑ maternal: HbA1c, Kleihauer-Betke, VDRL, ANA, anti-cardiolipin antibodies, PTT, serum/urine toxicology screens
- ❑ fetal: chromosomes, cord blood, skin biopsy, genetics evaluation (dysmorphology), autopsy
- ❑ placenta: pathology

Management
- ❑ labour induction
- ❑ monitor for maternal coagulopathy (10% risk of DIC)
- ❑ psychologic aspects of fetal loss

NORMAL LABOUR AND DELIVERY

THE FETUS

Fetal Lie
- ❑ refers to the orientation of the long axis of the fetus with respect to the long axis of the uterus (longitudinal, transverse, oblique)
- ❑ transverse/oblique often due to uterine anomalies (C/S if they don't convert)

Fetal Presentation
- ❑ refers to the fetal part presenting at pelvic outlet
  - breech (complete, frank, footling) (Figure 7)
  - cephalic
    - vertex (area between fontanelles and laterally by parietal eminences)
    - brow/sinciput
    - face
  - shoulder
  - compound (fetal extremity prolapses along with presenting part)
- ❑ all except vertex are considered malpresentations (see Abnormal Labour section)

Fetal Position
- ❑ refers to position of fetal occiput in relation to maternal pelvis
  - occiput anterior (OA): commonest presentation ("normal")
  - occiput posterior (OP): most rotate spontaneously to OA; may cause prolonged second stage of labour
  - occiput transverse (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)

Attitude
❑ refers to 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
❑ refers to position of presenting part relative to ischial spines
- at ischial spines = station 0 = engaged
- 2 cm below ischial spines = station +2

THE CERVIX
❑ dilatation: latent phase: 0-3 cm; active phase: 4-10 cm
❑ effacement: thinning of the cervix (25%-50%-100%)
❑ consistency: soft vs. hard
❑ position: posterior vs. anterior
❑ application: contact between the cervix and presenting part
❑ Note: For Bishop Score, see Induction of Labour section

DEFINITION OF LABOUR
❑ progressive DILATATION and EFFACEMENT of cervix, normally associated with DESCENT of presenting part
- preterm (> 20 but < 37 weeks GA)
- term (37-42 weeks)
- post-term (> 42 weeks)

❑ Braxton-Hick contractions
- irregular, occur throughout pregnancy and not associated with any dilatation, effacement or descent

FOUR STAGES OF LABOUR

First Stage of Labour
❑ latent phase
- uterine contractions typically infrequent and irregular
- slow cervical dilatation (usually to 3-4 cm) and effacement

❑ active phase
- rapid cervical dilatation to full dilatation (nulliparous ~1.2 cm/h and ~1.5 cm/h in multiparous)
- phase of maximum slope on Friedman curve (see Figure 6)
- painful, regular contractions ~q2 min, lasting 45-60 seconds
- contractions strongest at fundus, weakest at lower segment

Second Stage of Labour
❑ from full dilatation to delivery of the baby
❑ mother feels a desire to bear down and push with each contraction
❑ progress measured by descent

Third Stage of Labour
❑ separation and expulsion of the placenta
❑ can last up to 30 minutes before intervention indicated
❑ signs of placenta separation: gush of blood, lengthening of cord, uterus becomes globular and fundus rises

Fourth Stage of Labour
❑ first postpartum hour
❑ monitor vital signs and bleeding +/– oxytocin
❑ repair lacerations
❑ ensure uterus is contracted (palpate uterus and monitor uterine bleeding)
❑ 3rd and 4th stages of labour most dangerous to the mother (i.e. hemorrhage)

Table 9. Course of Normal Labour

Stage	Nulliparous	Multiparous
First	6-18 hours	2-10 hours
Second	30 minutes-3 hours	5-30 minutes
Third	5-30 minutes	5-30 minutes

CARDINAL MOVEMENTS OF THE FETUS DURING DELIVERY

- ❑ Engagement
- ❑ Descent
- ❑ Flexion
- ❑ Internal Rotation (to OA position ideally)
- ❑ Extension (delivery of head)
- ❑ External Rotation (restitution); head rotates in line with the shoulders
- ❑ Expulsion (delivery of shoulders and body)

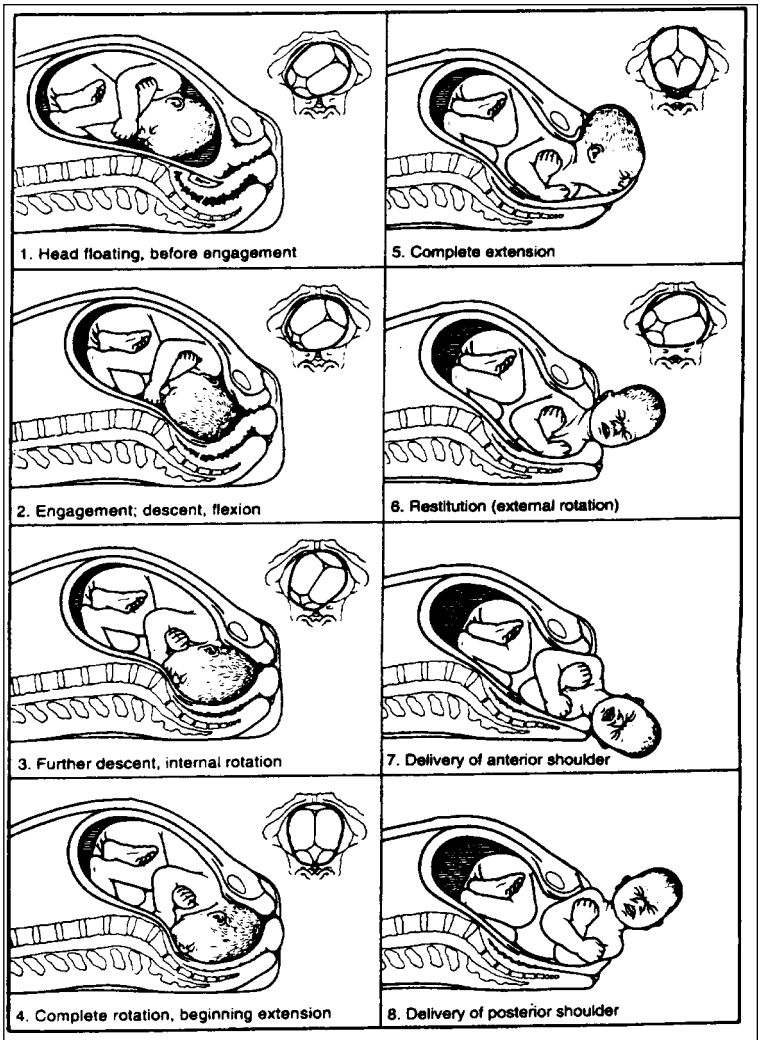

Figure 5. Movements of the Fetus During Labour and Delivery, Left Occiput Anterior Position

(*Reproduced with permission from Cunningham* FG, *MacDonald* PC, *Leveno* KJ *et al* (*eds*): *Williams Obstetrics. 19Th ed. Stanford, Appleton and Lange,* 1993)

ABNORMAL LABOUR

INDUCTION OF LABOUR

Definition
❑ the artificial initiation of labour to maintain maternal health or to remove the fetus from a potentially harmful environment

Prerequisites for Labour Induction
❑ maternal
- short anterior cervix with open os ("inducible" or "ripe")
- if cervix is not ripe, use prostaglandin (PG) gel (see below)

❑ fetal
- adequate fetal monitoring available
- cephalic presentation
- good fetal health

❑ likelihood of success determined by Bishop Score (see Table 10)
- score of 9-13 associated with high likelihood of vaginal delivery

Table 10. 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	+1

Indications
❑ maternal factors
- pregnancy-induced hypertension
- maternal medical problems, e.g. diabetes, renal or lung disease

❑ maternal-fetal factors
- Rh isoimmunization
- PROM
- chorioamnionitis
- post-term pregnancy

❑ fetal factors
- suspected fetal jeopardy as evidenced by biochemical or biophysical indications
- fetal demise

Contraindications
❑ maternal
- prior classical incision or complete transection of the uterus
- unstable maternal condition
- gross cephalopelvic diameter (CPD)
- active maternal genital herpes

❑ maternal-fetal
- placenta or vasa previa

❑ fetal
- distress
- malpresentation
- preterm fetus without lung maturity

INDUCTION METHODS

Cervical Ripening
❑ PG synthesized by cervical cells and in amniotic fluid to facilitate labour onset and progression

❑ PG gel used to augment slow or arrested cervical dilatation or effacement
- intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM
- vaginal (Cervidil) when cervix favorable, may use with ROM
- use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction
- risks include hyperstimulation and fetal heart rate abnormalities
- obtain reactive NST prior to administration

❑ other methods include placement of Foley catheter to mechanically dilate the cervix, or the use of osmotic dilators (laminaria)

Medical
- ❑ oxytocin/pitocin: start at 0.5-2 mU/minute IV, increasing by 1-2 mU/minute q20-60 minutes to a maximum of 36-48 mU/minute
- ❑ 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
- ❑ PGF-2 alpha used for intrauterine fetal demise (IUFD)

Surgical
- ❑ artificial rupture of membranes (amniotomy) to stimulate PG synthesis and secretion; may try this as initial measure

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 2 mU/minute IV, increased by 1-2 mU/minute q20-60 minutes to a maximum of 36-48 mU/minute
- ❑ half-life of oxytocin is 3-5 minutes (thus need continuous drip because effects wear off fast)

ABNORMAL PROGRESSION OF LABOUR
- ❑ expected patterns of descent of the presenting part and cervical dilatation fail to occur in the appropriate time frame (see Figure 6)
- ❑ can occur in all stages of labour
- ❑ traditionally three causes of abnormal labour have been recognized
  - Power: poor, inadequate or uncoordinated uterine contractions
  - Passenger: fetus too large in size or unusual presentation
  - Passage: cephalopelvic disproportion (CPD) = pelvis of inadequate size, shape or consistency, or maternal soft tissue resistance relative to fetus
- ❑ initial diagnosis of CPD requires progression into the active phase and the presence of adequate uterine contractions

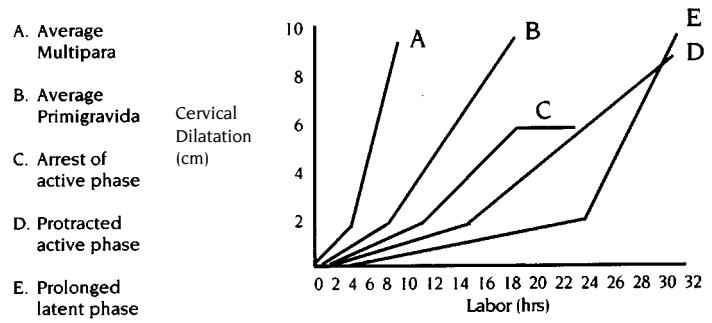

Figure 6. Normal and Abnormal Courses of the First Stage of Labour (Friedman Curve)

Arrest Disorder (Curve C)
- ❑ of dilatation: progress in dilatation does not occur for a period of 2 hours or more in a patient who has entered the active phase
  - arrest usually occurs at a cervical dilatation of 5 to 8 cm
- ❑ of descent: no progress in station for > 1 hour during second stage
  - should search for factors causing CPD (nearly 50%; requires C/S)
  - CPD diagnosed if adequate contractions measured by intrauterine pressure catheter (IUPC) with no descent/dilatation for > 2 hours
- ❑ if CPD ruled out, IV oxytocin and amniotomy can be attempted

Protraction Disorders (Curve D)
- ❑ of dilatation: when slope of cervical dilatation is less than 1.2 cm/hour in the primigravida or 1.5 cm/hour in the multigravida
- ❑ of descent: a rate of descent of less than 1.0 cm/hour in the primigravida or 2.0 cm/hour in the multigravida
- ❑ in about 1/3 of protraction disorders CPD will be present so that secondary arrest of dilatation usually develops
- ❑ 2/3 of protraction disorders will progress steadily through labour with ultimate uneventful vaginal delivery
- ❑ treatment: oxytocin augmentation if contractions are inadequate and/or amniotomy

Prolonged Latent Phase (Curve E)
- ❑ a period of 20 hours or more in the primigravida or 14 hours or more in the multigravida during which labour has not progressed to the active phase
- ❑ most often patient not really in labour (avoid amniotomy for fear of false labour and increased risk of intrauterine infection)
- ❑ premature or excessive use of sedation or analgesia commonly seen in these patients
- ❑ careful search for factors of CPD should be made
- ❑ treatment: oxytocin augmentation if diagnosis of labour is certain, otherwise rest +/– sedation

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 malpresentations, low-lying placenta, polyhydramnios, multiple gestation, CPD

Presentation
- ❑ visible or palpable cord
- ❑ FHR changes (variable decelerations, bradycardia or both)

Management
- ❑ emergent C/S
- ❑ adjunctive measures
    - alleviate pressure of the presenting part on the cord
    - keep cord warm and moist by replacing it into the vagina and/or applying warm saline soaks

SHOULDER DYSTOCIA

Definition
- ❑ impaction of anterior shoulder of fetus against symphysis pubis after fetal head has been delivered (life threatening emergency)

Etiology/Epidemiology
- ❑ incidence is 0.15-1.4% of deliveries
- ❑ occurs when breadth of shoulders is greater than biparietal diameter of the head
- ❑ risk factors
    - maternal
        - maternal obesity
        - diabetes
        - multiparity
    - fetal
        - prolonged gestation
        - macrosomia
    - labour
        - prolonged 1st and 2nd stages
        - prolonged deceleration phase (8-10 cm)
        - instrumental midpelvic delivery

Presentation
- ❑ watch for “turtle sign” (head advances during contraction but returns to previous position at end of contraction)

Complications
- ❑ chest compression by vagina or cord compression by pelvis can lead to hypoxia
- ❑ danger of brachial plexus injury (Erb palsy)
- ❑ fetal fracture (clavicle, humerus, cervical spine)
- ❑ maternal perineal injury, may result in PPH

Management
- ❑ goal: to displace anterior shoulder from behind symphysis pubis; follow a stepwise approach of maneuvers until goal achieved
  - **A**: apply suprapubic pressure
  - **A**: ask for help
  - **L**: legs in full flexion (McRobert's maneuver)
  - **A**: anterior shoulder disimpaction
  - **R**: release posterior shoulder
  - **M**: maneuver of Wood's corkscrew
  - **E**: episiotomy
- ❑ Other (last resort)
  - cleidotomy: deliberate fracture of the clavicle
  - Zavanelli maneuver: replacement of fetus into uterine cavity and emergent C/S

BREECH PRESENTATION

Definition
- ❑ fetal buttocks or lower extremity is the presenting part (Figure 7)
- ❑ complete: flexion at hips and knees
- ❑ frank: flexion at hips, extension at knees
  - most common type of breech presentation
  - most common breech presentation to be delivered vaginally
- ❑ footling: may be single or double with extension at hip(s) and knee(s) so that foot is the presenting part

Etiology/Epidemiology
- ❑ occurs in 3-4% of pregnancies at term (25% before 28 weeks)
- ❑ risk factors
  - maternal risk factors
    - pelvis (contracted)
    - uterus (shape abnormalities, intrauterine tumours, fibroids extrauterine tumours causing compression)
    - grand multiparity
  - maternal-fetal
    - placenta (previa)
    - amniotic fluid (poly/oligohydramnios)
  - fetal
    - prematurity
    - multiple gestation
    - congenital malformations (found in 6% of breeches; 2-3x the incidence in vertex presentations)

Presentation
- ❑ noted by Leopold maneuvers and U/S

Management
- ❑ extenal breech version
  - criteria: > 37 weeks, singleton, unengaged presenting part, reactive NST
  - contraindications: previous T3 bleed, prior classical C/S, previous myomectomy, oligohydramnios, PROM, placenta previa, abnormal U/S, suspected IUGR, hypertension, uteroplacental insufficiency
  - risks: abruption, cord compression
  - method: tocometry, followed by ultrasound guided transabdominal manipulation of fetus
  - if patient Rh negative, give Rhogam prior to procedure
  - good prognostic factors (for a successful version)
    - multiparous
    - good fluid volume
    - small baby
    - skilled obstetrician
- ❑ criteria for vaginal delivery
  - frank or complete breech, GA > 36 weeks
  - estimated birth weight (EBW) 2,500-3,800 g based on clinical and U/S assessment (5.5-8.5 lb)
  - fetal head flexed
  - continuous fetal monitoring
  - maternal pelvis adequately large (clinically, or "proven" by previous delivery)
  - no other indication for C/S
  - experienced obstetrician, assistant, and anesthetist present

- ❑ C/S for all other presentations (except mentoanterior face presentation)
- ❑ C/S is recommended if the breech has not descended to the perineum in the second stage of labour after two hours, in the absence of active pushing, or if vaginal delivery is not imminent after one hour of active pushing
- ❑ a recent randomized multicentre trial (Toronto, CA) has demonstrated that for women with frank or complete breech presentations, perinatal mortality, neonatal mortality, and serious neonatal morbidity is significantly lower for those with planned C/S over those with planned vaginal birth, with no significant difference in maternal complications

A. Complete Breech B. Frank Breech C. Footling Breech

Figure 7. Types of Breech Presentation

Illustration by Jennifer Bosy

VAGINAL BIRTH AFTER CESAREAN (VBAC)

- ❑ 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 (i.e. need to do a repeat C-section before onset of labour)

- ❑ previous classical, inverted T, or unknown uterine incision, or complete transection of uterus (6% risk of rupture)
- ❑ history of hysterotomy or previous uterine rupture
- ❑ multiple gestation
- ❑ estimated fetal weight > 4,000 g (9 lbs)
- ❑ non-vertex presentation or placenta previa
- ❑ inadequate facilities or personnel for emergency C/S

UTERINE RUPTURE

- ❑ 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

Management

- ❑ immediate delivery for fetal survival
- ❑ maternal stabilization (may require hysterectomy)

Complications

- ❑ maternal mortality 1-10%
- ❑ maternal hemorrhage and shock
- ❑ DIC
- ❑ amniotic fluid embolus
- ❑ hysterectomy
- ❑ fetal distress —> 50% mortality

AMNIOTIC FLUID EMBOLUS

Definition

- ❑ amniotic fluid debris in maternal circulation

Etiology/Epidemiology

- ❑ rare intrapartum or immediate postpartum complication
- ❑ 80% mortality
- ❑ risk factors
  - placental abruption
  - rapid labour
  - multiparity
  - uterine rupture

Presentation

- ❑ sudden onset of respiratory distress, cardiovascular collapse and coagulopathy

Management

- ❑ supportive measures, coagulopathy correction

OPERATIVE OBSTETRICS

❑ operative vaginal delivery = forceps or vacuum extraction

INDICATIONS FOR OPERATIVE VAGINAL DELIVERY

❑ fetal
- non-reassuring fetal status
- 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 (cardiac/cerebrovascular disease)
- exhaustion, lack of cooperation and excessive analgesia may impair pushing effort

FORCEPS

Outlet Forceps

❑ head visible between labia in between contractions
❑ sagittal suture in or close to A-P diameter
❑ rotation cannot exceed 45 degrees

Low Forceps

❑ presenting part at station +2 or greater
❑ similar to outlet forceps
❑ subdivided based on whether rotation less than or greater than 45 degrees

Mid Forceps

❑ presenting part below spines but above station +2

Types of Forceps

❑ Simpson forceps for OA presentations
❑ rotational forceps (Kjelland) when must rotate head to OA
❑ Piper forceps for breech

Prerequisites

A: anesthesia
B: bladder empty
C: cervix fully dilated
D: determine position of fetal head
E: equipment ready (including facilities for emergent C/S)
F: fontanelle ("Position For Safety" is posterior fontanelle midway between shanks, fenestration barely palpable)
G: gentle traction
H: handle elevated
I: incision (episiotomy)
J: once jaw visible, remove forceps

(modified from J. B*achman*, 1989)

Complications

❑ maternal: anesthesia risk, lacerations, injury to bladder, uterus, bone, pelvic nerve damage, PPH, infections
❑ fetal: fractures, facial nerve palsy, trauma to face/scalp, intracerebralhemorrhage (ICH), cephalohematoma, cord compression

VACUUM EXTRACTION

❑ traction instrument used as alternative to forceps delivery; aids maternal pushing
❑ same indications as forceps
❑ advantages
- easier to apply
- less force on fetal head
- less anesthesia required
- less maternal and fetal injury
- will lose suction and dislodge if unrecognized CPD present

❑ disadvantages
- suitable only for vertex presentations
- maternal pushing required
- contraindicated in preterm delivery

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

EPISIOTOMY

Definition

- ❑ incision in the perineal body at the time of delivery
- ❑ midline: incision through central tendinous portion of perineal body and though bulbocavernosus muscle; better healing but increased risk of deep tear
- ❑ mediolateral: incision through bulbocavernosus, transverse perineal muscle, and levator ani; reduced risk of extensive tear but poorer healing and more pain

Indications

- ❑ to prevent a tear (episiotomy easier to repair)
- ❑ to relieve obstruction of the unyielding perineum
- ❑ instrumental delivery
- ❑ controversy over whether it is preferable to make a cut, or let the perineum tear as needed; current evidence suggests we should generally let patients tear

CESAREAN DELIVERY

Indications

- ❑ maternal
  - obstruction, active herpetic lesion on vulva, invasive cervical cancer, previous uterine surgery, underlying maternal illness (eclampsia, HELLP syndrome, heart disease)
- ❑ maternal-fetal
  - failure to progress, placental abruption or previa
- ❑ fetal
  - fetal distress, malpresentation, cord prolapse, certain congenital anomalies

Risks

- ❑ anesthesia
- ❑ hemorrhage
- ❑ infection (UTI, wound, endometritis)
- ❑ injury to surrounding structures
- ❑ thromboembolic phenomena
- ❑ increased recovery time/hospital stay

OBSTETRICAL ANESTHESIA

PAIN PATHWAYS DURING LABOUR

- ❑ early first stage: pain via visceral afferents enter the spinal cord at T10-L1
  - dilatation of the cervix
  - lower uterine distension
  - contraction of the uterus
- ❑ late first stage and second stage pain via visceral and somatic afferents (pudendal nerve) enter the spinal cord at S2-S4
  - contraction of the uterus
  - distension and stretching of pelvic structures (pelvic peritoneum, fascia, ligaments, and muscles)
  - pressure on lumbar nerves
- ❑ third stage of labour is usually well tolerated with spontaneous placental delivery
  - analgesia may be necessary for manual extraction of placenta

ANALGESIA

Psychoprophylaxis and Physical Analgesia

- ❑ "natural childbirth" (e.g. Lamaze prenatal classes) whereby an informed mother utilizes relaxation techniques to stimulate the descending inhibitory pathways
- ❑ whirlpool baths, transcutaneous nerve stimulation (TNS), and acupuncture inhibit nociceptive impulses and reduce pain propagating muscle tension
- ❑ especially effective in early stages of labour

Intravenous Analgesia

- ❑ meperidine (Demerol)
  - best used in early stages of labour, less effective once labour is well established
  - rapidly cleared by fetus if IV (prolonged if IM)
  - peak fetal level 2-4 hours after maternal injection IM
  - can suppress respiration in the newbom (treat with naloxone)
  - side effects: orthostatic hypotension, nausea, and vomiting

Inhalational Analgesia

- ❑ nitrous oxide
  - 50% nitrous oxide in O_2
  - self-administered during contractions
  - does not prolong labour or interfere with uterine contractions but administration > 20 minutes may result in neonatal depression
  - provides partial pain relief during labour as well as at delivery

ANESTHESIA

Local Perineal Anesthesia

- ❑ local blocks
- ❑ lidocaine for episiotomy
- ❑ pudendal blocks

Regional Anesthesia

- ❑ epidural
  - most commonly used technique for both labour and delivery
  - does not prolong first stage, but may reduce maternal expulsive efforts, therefore usually used in earlier stages of labour (< 4 cm)
  - 0.25% bupivacaine (Marcaine) usually used for labour (longer acting compared to lidocaine and less motor block)
  - 2% lidocaine (Xylocaine) usually used for vaginal deliveries and C/S in varying doses
  - 19 gauge indwelling catheter inserted into lumbar epidural space
  - preload mother with 500-1000 mL IV fluid to prevent maternal hypotension associated with epidural (fetal depression rare if maternal hypotension avoided)
  - test dose given first to check for spinal block followed by another dose to rule out intravascular injection; if no dizziness or tinnitus, rest of dose is given
  - complications: inadvertent total spinal with cardiovascular collapse and respiratory arrest, inadvertent intravascular injection with seizures, post-ictal depression and possible cardiac arrest
- ❑ walking epidural
  - goal is effective analgesia with no motor blockade
  - 0.125% bupivacaine plus low dose fentanyl
- ❑ spinal block
  - for C/S need anesthesia of T4-T8
  - injection of local anesthetic into subarachnoid space
  - fastest onset
  - least drug exposure for fetus because small dose required
  - not appropriate for labour due to intense motor blockade
  - beware of rapid hypotension and preload mother with 1,000 mL fluid

General Anesthesia

- ❑ not used for vaginal deliveries, but may be used for C/S in certain circumstances
- ❑ rapid sequence induction to prevent aspiration
- ❑ pre-oxygenate mother with 100% O_2 as she is prone to hypoxia during intubation secondary to decreased FRC and increased O_2 consumption

NORMAL PUERPERIUM

DEFINITION
- ❑ period of adjustment after pregnancy when pregnancy-induced anatomic and physiologic changes are reversed
- ❑ traditionally, puerperium lasts 6 weeks

POST-DELIVERY EXAMINATION
- ❑ The **8 Bs**: **B**lues (post-partum), **B**reathing (DVT/PE), **B**reast, **B**elly, **B**owels, **B**ladder, **B**leeding, **B**aby

BREAST
- ❑ 2 events stimulate lactation
  - sudden drop in placental hormones (especially estrogen)
  - suckling stimulates release of prolactin and oxytocin
- ❑ colostrum secreted for ~ 2 days after delivery (contains protein, fat, minerals, IgA)
  - replaced by milk after ~ 3-6 days (contains protein, lactose, water, fat)
- ❑ breast-feeding encouraged (see Pediatrics Chapter)

UTERUS
- ❑ through process of catabolism, uterus weight rapidly diminishes
- ❑ cervix loses its elasticity and regains firmness
- ❑ start oxytocin drip or give oxytocin 10 U IM after 3rd stage (i.e. after delivery of placenta; some give IM dose after delivery of head)
- ❑ generally should involute ~ 1 cm (1 finger breadth) below umbilicus per day in first 4-5 days
- ❑ involution then slows down; reaches non-pregnant state in 4-6 weeks postpartum

LOCHIA (Normal Vaginal Discharge Postpartum)
- ❑ monitored for signs of infection or bleeding
- ❑ normally decreases and changes colour from red (lochia rubra; due to presence of erythrocytes) to yellow (lochia serosa) to white (lochia alba; residual leukorrhea) over 3-6 weeks
- ❑ foul smelling lochia suggests endometritis

POSTPARTUM CARE
- ❑ bowel- encourage plenty of fluids and high-fibre foods, bulk laxatives may be helpful
- ❑ bladder: maintain high fluid intake
- ❑ sex: wait until 4-6 weeks post-delivery
- ❑ hemorrhoids/perineal tenderness: pain meds, doughnut cushion, Sitz baths, ice compresses
- ❑ exercise: encourage gradual increases in walking, Kegel exercises
- ❑ do not use douches or tampons for 4-6 weeks post-delivery

PUERPERAL COMPLICATIONS

RETAINED PLACENTA

Definition
- ❑ placenta undelivered after 30 minutes

Etiology/Epidemiology
- ❑ placenta separated but not delivered, or abnormal placental implantation
  - placenta accreta: placenta adherent to myometrium
  - placenta increta: invasion of myometrium
  - placenta percreta: invasion of myometrium beyond serosa
- ❑ risk factors: placenta previa, prior C/S, post-pregnancy curettage, prior manual placental removal, uterine infection

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

Complications
- ❑ increased risk of infection or bleeding

UTERINE INVERSION

Definition
❑ uterus prolapses through the cervix and passes out of the vaginal introitus

Etiology
❑ often iatrogenic (excess cord traction)
❑ more common in grand multiparous (lax uterine ligaments)
❑ can cause profound vasovagal response with vasodilation and hypovolemic shock

Management
❑ urgent management essential (may require general anesthetic if unsuccessful)
❑ call anesthesia
❑ initiate IV crystalloids
❑ replace uterus without removing placenta
❑ remove placenta manually and withdraw slowly
❑ can use tocolytic drug (e.g. terbutaline) to relax uterus
❑ IV oxytocin infusion
❑ re-explore uterus
❑ may require GA +/– surgery

POSTPARTUM PYREXIA

Definition
❑ fever > 38.0ºC on any 2 of the first 10 days postpartum, except the first day

Causes
❑ wind (atelectasis, pneumonia)
❑ water (UTI)
❑ wound (gram +/–, aerobes, and anaerobes)
- C/S incision site
- episiotomy site
- empiric treatment: clindamycin + gentamicin
- prophylaxis against post-C/S endometritis
  - begin antibiotic immediately after cord clamping and administer only 1-3 doses
  - cefazolin is most common

❑ walking
- pelvic thrombophlebitis (diagnosis of exclusion)
- DVT

❑ breast
- engorgement may cause slight physiologic temperature rise on first day
- mastitis (*Staphylococcus aureus* most common)

❑ endometritis
- blood and genital cultures

POSTPARTUM HEMORRHAGE (PPH)

Definition
❑ loss of > 500 mL of blood at the time of vaginal delivery, or > 1,000 mL with C/S

Etiology (4 Ts)
❑ Tone: uterine atony (most common cause of PPH; try to avoid by giving IV oxytocin with delivery of the anterior shoulder)
- occurs within first 24 hours
- labour (prolonged, precipitous)
- uterus (infection, over-distension)
- placenta (abruption, previa)
- maternal factors (grand multiparity, GA)
- halothane anesthesia

❑ Tissue: retained placenta (see above)
❑ Trauma: laceration (vagina, cervix, uterus), episiotomy, hematoma, uterine rupture, uterine inversion (see above)
❑ Thrombin: coagulopathy
- most identified prior to delivery (low platelets increases risk)
- includes hemophilia, DIC, aspirin use, ITP, TTP, VWD (most common)
- monitor fibrinogen, platelets

Investigations
❑ pelvic U/S if indicated to look for cause if unknown

PUERPERAL COMPLICATIONS . . . CONT.

Management
- ❑ determine cause, call for help
- ❑ supportive
  - ABCs, fluid, cross and type 4 units PRBC
- ❑ examination
  - reexamine patient, ensure complete delivery of placenta
  - check for uterine atony and drain bladder
  - check for cervical and vaginal lacerations
  - elevate the uterus and massage through patient's abdomen
- ❑ medical
  - oxytocin (5 U IV push then 40 U/L NS drip)
  - methylergonavine maleate (ergotamine; 0.2 mg PO or 0.25 mg IM) (normotensive patients only; must explore uterus before giving ergotamine)
  - prostaglandins (PGF-2 alpha intrauterine or IM, misoprostol)
  - hemabate (prostaglandin; 0.25-1.00 mg intramyometrium every 15 minutes)
  - uterine packing (3-4 five yard Kerlex rolls tied together and soaked in betadine and removed in 12-24 hours; controversial)
- ❑ surgical
  - seek and suture lower genital tract lacerations
  - D&C (beware of vigorous scraping which may cause Asherman syndrome)
  - hypogastric, ovarian artery or uterine artery ligation
  - arterial embolization
  - hysterectomy (last option)

Complications
- ❑ Sheehan syndrome (pituitary necrosis)

POSTPARTUM MOOD ALTERATIONS

Postpartum blues
- ❑ very common, 85% of new mothers
- ❑ onset day 3-10
- ❑ considered an extension of the "normal" hormonal changes and adjustment to a new baby
- ❑ self-limited, does not last more than 2 weeks

Postpartum Depression
- ❑ signs and symptoms of major depression occurring in a woman within 6 months of childbirth (see Psychiatry Chapter)
- ❑ incidence:10-20%
- ❑ suspect if the "blues" last beyond 2 weeks, or if the symptoms in the first two weeks are severe (e.g. extreme disinterest in the baby, suicidal or homicidal ideation)
- ❑ treatment with antidepressants is often necessary
- ❑ interferes with bonding and attachment between mother and baby so it can have long term effects

Postpartum Psychosis
- ❑ rare (0.2%)
- ❑ presents as an acute psychotic episode, or in the context of depression

DRUGS CONTRAINDICATED IN PREGNANCY

- ❑ most drugs cross the placenta to some extent
- ❑ use any drug with caution and only if necessary
- ❑ Motherisk at the Hospital for Sick Children in Toronto is a valuable resource **(416-813-6780)**

ANTIBIOTICS
- ❑ safest = ampicillin, cephalosporins
- ❑ erythromycin
  - maternal liver damage (acute fatty liver)
  - used only if contraindication to penicillin use
- ❑ tetracyclines
  - staining of child's teeth
- ❑ sulpha drugs
  - antifolates, therefore theoretical risk in first trimester
  - risk of kernicterus in third trimester
- ❑ metronidazole
  - antimetabolite, therefore theoretical risk in first trimester
- ❑ chloramphenicol
  - grey baby syndrome (fetal circulatory collapse secondary to accumulation since fetus cannot metabolize this drug)
- ❑ fluoroquinolones
  - risk of cartilage damage (in dog and rat studies)

OTHER DRUGS

- ❑ alcohol
  - increased incidence of abortion and stillbirth, congenital anomalies, fetal alcohol syndrome (growth retardation, CNS involvement and facial anomalies)
- ❑ cigarettes
  - decreased birth weight, placenta previa/abruption, increased spontaneous abortion, preterm labour and stillbirth
- ❑ anticoagulants
  - fetal warfarin syndrome: nasal hypoplasia, epiphyseal stippling, optic atrophy, mental retardation, intracranial hemorrhage
  - also spontaneous abortion, stillbirth, prematurity, IUGR
- ❑ ACE inhibitors
- ❑ anticonvulsants
  - phenytoin associated with fetal hydantoin syndrome in 5-10%: IUGR, mental retardation, facial dysmorphogenesis, congenital anomalies
  - valproate associated with NTD in 1%
  - carbamazepine associated wiht NTD in 1-2%
  - generally recommended that pregnant women remain on the lowest dose anticonvulsant appropriate for their seizure type
- ❑ lithium
  - Ebstein's cardiac anomaly, goitre, hyponatremia
- ❑ cocaine
  - microcephaly, growth retardation, prematurity, MR
- ❑ DES (and other estrogenic or androgenic compounds)
  - vaginal adenosis, adenocarcinoma, uterine malformation in daughters exposed to DES in utero
- ❑ retinoids (e.g. Accutane)
  - CNS, craniofacial, cardiovascular, and thymic anomalies

IMMUNIZATIONS

- ❑ administration is dependent on the risk of infection vs. risk of immunization complications
- ❑ safe
  - tetanus toxoid, typhoid fever (killed bacterial), diphtheria, influenza, hepatitis B
- ❑ avoid live vaccines (risk of placental and fetal infection)
  - polio and mumps, varicella
- ❑ contraindicated
  - rubella

BREASTFEEDING AND DRUGS

- ❑ safe
  - penicillins, aminoglycosides, cephalosporins
  - oral contraceptive use (low dose) is now believed to be safe
  - depoprovera
- ❑ avoid
  - chloramphenicol (bone marrow suppression)
  - metronidazole (mutagenic in vitro)
  - sulphonamides (hemolysis with G6PD deficiency)
  - nitrofurantoin (hemolysis with G6PD deficiency)
  - tetracycline (stains teeth and bones)
  - lithium
  - antineoplastics and immunosuppressants
  - psychotropic drugs (relative)

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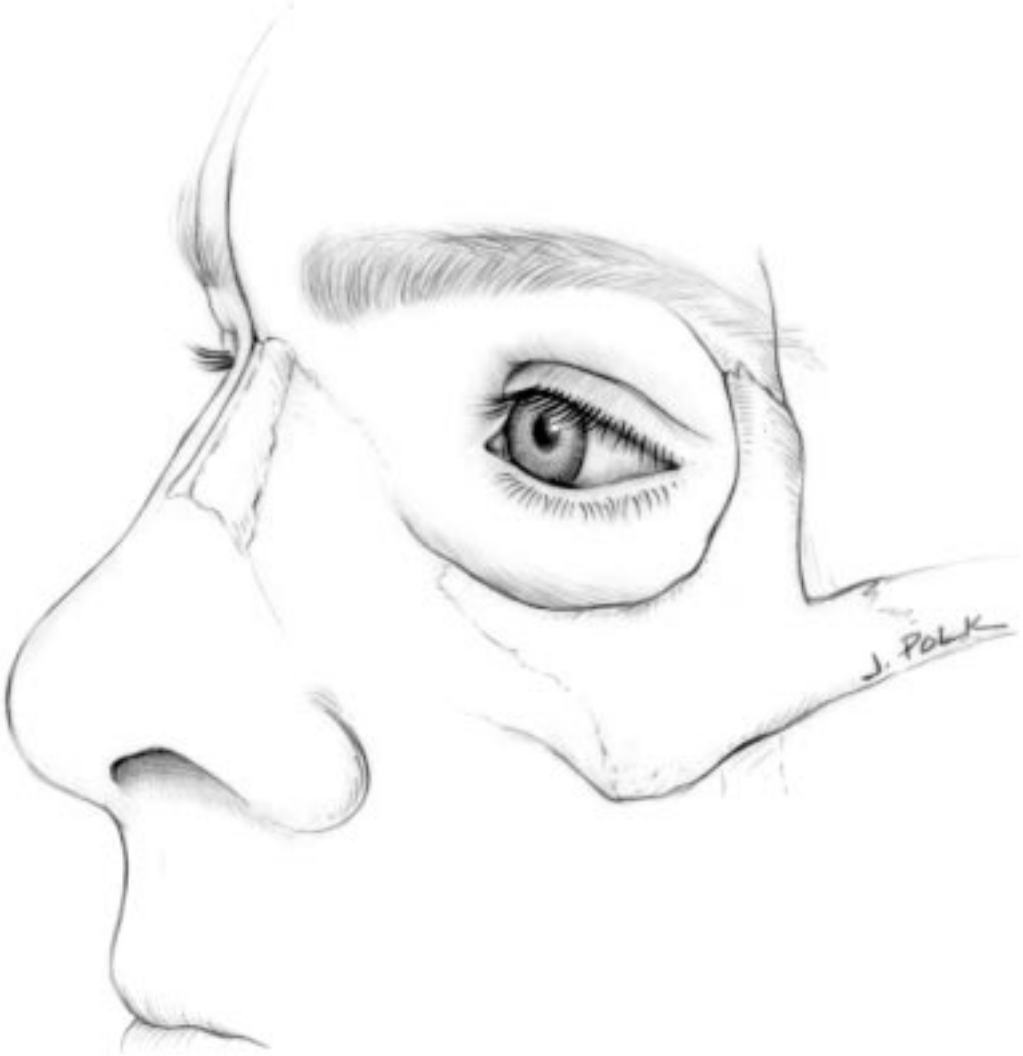

Illustrated by Jen Polk