OBSTETRICS

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DEFINITIONS

Gravidity
- ❑ the total number of pregnancies of any gestation
  - includes 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 digit number (T P A L)
  - 1st digit: number of term infants delivered (> 37 weeks)
  - 2nd digit: number of premature infants delivered (20 to 37 weeks)
  - 3rd digit: number of abortions (< 20 weeks)
  - 4th digit: number of living children

Trimesters
- ❑ T1: 0 to 12 weeks
- ❑ T2: 12 to 28 weeks
- ❑ T3: 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 1000 total births

Perinatal Mortality Rate
- ❑ the annual number of stillbirths and early neonatal deaths (in the first seven days of life) per 1000 total births
- ❑ causes
  - prematurity
  - congenital anomalies

Neonatal Mortality Rate
- ❑ the annual number of deaths of liveborn infants within 28 days per 1000 live births

Infant Mortality Rate
- ❑ the annual number of deaths of liveborn infants in the first year of life per 1000 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, PIH, PPH, infection, PE
  - indirect: from pre-existing illness or by accident

Birth Rate
- ❑ the annual number of live births per 1000 population

Fertility Rate
- ❑ the annual number of live births per 1000 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 sign): 4-6 weeks
- ❑ bluish discoloration of the cervix and vagina due to engorgement of pelvic vasculature (Chadwick sign): 6 weeks
- ❑ uterine enlargement
- ❑ softening of the isthmus (Hegar sign): 6-8 weeks

Investigations
- ❑ ßhCG
  - positive in the serum at 9 days post-conception
  - positive in the urine 28 days after LMP
- ❑ transvaginal ultrasound
  - 5 weeks: gestational sac visible (ßhCG = 1200 -1500 mIU/mL)
  - 6 weeks: fetal pole seen
  - 7-8 weeks: fetal heart tones visible
- ❑ transabdominal U/S
  - intrauterine pregnancy visible at ßhCG = 5000 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
- ❑ increased cardiac output, heart rate, and blood volume (hyperdynamic circulation)
- ❑ decreased blood pressure (especially diastolic, maximal in T2) due to decreased peripheral vascular resistance
- ❑ blood flow to the uterus, kidneys, breasts, and skin increases with gestational age
- ❑ enlarging uterus compresses 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 DVT and 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
  - 5000 to 12 000/uL in pregnancy
  - up to 25 000/uL in labour/postpartum
  - often have improvement in autoimmune conditions

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, FRC and residual volume
- ❑ vital capacity unchanged
- ❑ increased tidal volume by 35-50%
- ❑ increased alveolar ventilation by 65%

Gastrointestinal 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 in pregnancy
- ❑ hemorrhoids caused by constipation and elevated venous pressure

Genitourinary System
- ❑ increased GFR 50% (therefore decreased BUN and serum creatinine) but no change in urine output because of increased reabsorption in tubules
- ❑ 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 due to stasis
  - asymptomatic bacteriuria more likely to become a clinically significant infection (i.e. pyelonephritis) in pregnancy and therefore should be treated

Endocrine System
- ❑ estrogen
  - main estrogen is estradiol (E3)
  - 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 actions: maintains the corpus luteum
  - serum ßhCG positive 8-9 days after ovulation
  - plasma levels double every 1-2 days, peak (8-10 weeks) and then fall to a plateau until delivery
  - rule of 10's
    - 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

- ❑ Ca^{++} metabolism
  - total maternal Ca^{++} decreased due to decreased albumin
  - free ionized (i.e. active) proportion remains the same due to increased PTH which results in increased bone resorption and gut absorption
  - bone turnover increased but no loss of bone density because estrogen counteracts the PTH effect by inhibiting resorption

Neurologic System
- ❑ carpal tunnel syndrome and Bell's palsy more common

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

PRENATAL CARE

PRECONCEPTION COUNSELLING
- ❑ folic acid to prevent NTD's (0.4 to 1 mg daily in all women, 4 mg if past NTD)
- ❑ genetic history and risk factors
- ❑ modify medications, alcohol, smoking
- ❑ rubella immunity
- ❑ proper nutrition
- ❑ use of prenatal vitamin and iron supplementation
- ❑ impact on family and occupation (maternity/paternity leave)
- ❑ domestic violence (50% of domestic violence begins in pregnancy)
- ❑ depression / mental health

INITIAL VISIT
- ❑ generally after 12 weeks

History
- ❑ determine GA by dates from the first day of the LMP (if regular periods and sure dates)
- ❑ if LMP unsure, get a dating ultrasound
- ❑ determine EDC using the Naegele Rule
  - first day of LMP + 7 days – 3 months
  - e.g. LMP = 1 Apr. 1999, EDC = 8 Jan. 2000
  - modify appropriately for longer or shorter cycles
- ❑ obtain obstetric history of all previous pregnancies (GTPAL)
- ❑ obtain relevant medical, social, and family history
- ❑ counselling (see Preconception Counselling Section)
  - drug use, alcohol consumption, smoking
  - breastfeeding

Physical
- ❑ complete physical exam
- ❑ baseline BP (very important for relating 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
- ❑ 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

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
❑ 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 with only 40% of weight gain products of conception
❑ blood pressure
❑ symphyseal-fundal height 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 @ pubic symphysis
  • 20 weeks @ umbilicus
  • 37 weeks @ sternum
❑ differential diagnosis of uterus incorrect size for dates (accurate dates essential)
  • maternal—> DM
  • maternal-fetal—> poly/oligo-hydramnios, multiple gestation
  • fetal—> abnormal karyotype, IUGR, fetal anomaly
❑ examination of abdomen for lie, position and presentation (Leopold maneuvers) in T3
❑ fetal heart tones starting at ~ 12 weeks if using doppler U/S

Table 1. Gestation-Dependent Management

Gestational Age (weeks)	Management Issues
10-12	CVS
15-16 or up to term	Amniocentesis
16	MSS
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 • depression/mental health

Maternal Serum Screen (MSS or Triple Screen)

❑ offers a risk estimate of whether the fetus may be affected with Down's syndrome, trisomy 18, or a NTD
❑ to make accurate diagnosis, positive MSS should be followed up with U/S and/or amniocentesis
❑ three markers (MSAFP, ßhCG, uE3)
  • Trisomy 21: low MSAFP, high ßhCG, low uE3
  • Trisomy 18: low MSAFP, low ßhCG, low uE3
❑ differential diagnosis of high MSAFP
  • wrong gestational age
  • > 1 fetus (e.g. twins)
  • fetal demise
  • NTD
  • abdominal wall defects (e.g. omphalocele)

- ❑ differential diagnosis of low MSAFP
  - 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
- ❑ MSS has a 6-7% false positive rate
- ❑ detection rate of Trisomy 21 with the 3 markers is 2-3 times higher than with MSAFP alone, however will still miss 20-30% of Trisomy 21 pregnancies in older women and will not reliably detect other chromosomal anomalies that occur more frequently in older women so amniocentesis should still be offered to high risk women

Group B Streptococcus
- ❑ danger of vertical transmission (neonatal sepsis, meningitis or pneumonia)
- ❑ indications for antibiotic prophylaxis (intrapartum ampicillin or clindamycin in pen-allergic - guidelines controversial)
  - positive GBS screen based on vaginal cultures taken at 36-38 weeks or
  - GBS status unknown and one of the following risk factors
    - previous GBS bacteriuria even if treated
    - previous infant with GBS infection
    - preterm labour
    - PROM > 12 hours
    - maternal intrapartum temperature > 37.7ºC
    - fetal tachycardia

PRENATAL DIAGNOSIS

Indications
- ❑ maternal age > 35 (increased risk of some chromosomal anomalies)
- ❑ abnormal MSS or ultrasound
- ❑ past history of pregnancy with chromosomal anomaly or genetic disease
- ❑ either parent a known carrier of a genetic disorder or balanced translocation
- ❑ three or more miscarriages
- ❑ family history of chromosomal anomaly, genetic disorder, birth defect, or undiagnosed mental retardation
- ❑ consanguinity

Amniocentesis
- ❑ ultrasound-guided transabdominal extraction of amniotic fluid
- ❑ at 15-16 weeks gestation to identify genetic problems such as trisomies
- ❑ during 3rd trimester for assessment of fetal lung maturity
  - L/S ratio: if > 2:1, fetal lungs are mature enough that RDS less likely to occur
- ❑ used to quantitate amniotic fluid bilirubin concentration in Rh-isoimmunized pregnancies
- ❑ advantages
  - screen for NTD (acetyl cholinesterase 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
- ❑ 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)
- ❑ disadvantages
  - 1-2% risk of spontaneous abortion
  - does not screen for neural tube defects (NTD)
  - risk of limb injury
  - poor test because of genetic mosaicism

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)
  - 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
  - confirm intrauterine pregnancy
  - identify multiple pregnancy
  - past history of early fetal losses
  - bleeding or other complications
  - measure fetal growth and identify IUGR
  - placental localization
  - determine gestational age (most accurately determined through measurement of crown-rump length prior to 11-12 weeks gestational age)

Non-Stress Test (NST)

- ❑ constant fetal heart rate (FHR) tracing using an external doppler to assess fetal heart rate and its relationship to fetal movement (see Intrapartum Fetal Cardiotocography)
- ❑ indicated when there is any suggestion of uteroplacental insufficiency or suspected fetal distress
- ❑ reactive NST (normal)
  - observation of two accelerations of FHR > 15 bpm from the baseline lasting ≥ 15 seconds in 20 minutes
- ❑ nonreactive NST (abnormal)
  - one or no FHR acceleration of at least 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 BPP

Biophysical Profile (BPP)

- ❑ consists of NST and 30 minute ultrasound assessment of the fetus
- ❑ five scored parameters of BPP (see Table 2)
- ❑ scores
  - 8-10 perinatal mortality rate 1: 1000 repeat BPP as clinically indicated
  - 6 perinatal mortality 31:1000 repeat BPP in 24 hours
  - 0-4 perinatal mortality rate 200:1000 deliver fetus if mature
- ❑ AFV a marker of chronic hypoxia, all other parameters indicative of acute hypoxia

Table 2. Scoring of the Biophysical Profile

Parameter	Normal (2)	Abnormal (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
- ❑ fetal presenting part, position, and station
- ❑ bony pelvis size and shape

Intrapartum Fetal Cardiotocography (CTG)
- ❑ external (doppler) vs. internal (scalp electrode) monitoring
- ❑ describe in terms of baseline FHR, variability (short term, long term) and periodicity (accelerations, decelerations) (see Table 3)
- ❑ baseline FHR
  - normal range is 120-160 bpm
  - a parameter of fetal well-being vs. distress
- ❑ variability
  - short term - beat to beat (requires scalp monitor)
  - long term - described with respect to frequency and amplitude of change in baseline
  - frequency is defined as number of times in a 1 minute period with an increase or decrease of at least 5 bpm lasting 5 seconds (average frequency is 3)
  - amplitude is based on difference between highest and lowest FHR within a 1 minute period (11-25 bpm is average)
- ❑ periodicity
  - accelerations
    - excursion of 15 bpm or more lasting for at least 15 seconds, in response to fetal movement or uterine contraction
  - decelerations
    - describe 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 4-7cm
  - variable decelerations (see Figure 2)
    - most common type of periodicity seen during labour
    - variable in shape, onset and duration
    - 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
  - late decelerations (see Figure 3)
    - uniform (symmetric) in shape, with onset late in contraction, lowest depth after peak of contraction, and returns 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 as 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, stopping oxytocin, C/S

Table 3. Factors Affecting Fetal Heart Rate

Fetal Tachycardia (FHR > 160)	Fetal Bradycardia (FHR < 120)	Decreased Variability
fetal or maternal anemia	uterine hypercontractility	hypoxia
fetal arrhythmia	congenital heart block	narcotics
early hypoxia (abruption, PIH)	late hypoxia (abruption, PIH)	magnesium sulphate
chorioamnionitis	maternal hypotension	CNS anomalies of fetus
maternal fever	maternal use of beta blockers	fetal inactivity / sleep (< 20 min)
sympathomimetics (i.e. ritodrine)	rapid descent during labour	maternal dehydration
hyperthyroidism / thyrotoxicosis	acute cord prolapse	infection

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

- ❑ indicator of fetal distress
- ❑ > 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
- ❑ may occur prior to ROM or after rupture has occurred with passage of clear fluid
- ❑ classified as thick or thin
- ❑ thin meconium appears as a lightly stained yellowish or greenish fluid
- ❑ thick meconium appears dark green or black and may have pea-soup consistency
  - associated with lower APGARS and increased risk of meconium aspiration
  - call pediatrics to delivery
  - may indicate undiagnosed breech
- ❑ increasing amount during labour may be a sign of fetal distress

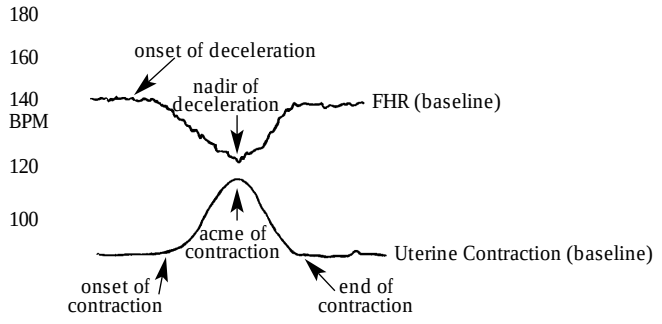


Figure 1. Early Decelerations

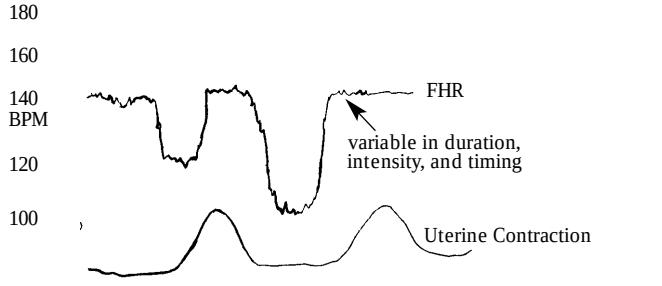


Figure 2. Variable Decelerations

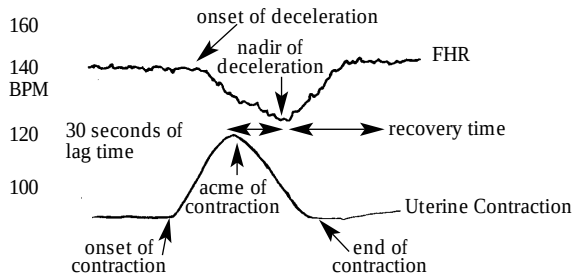


Figure 3. Late Decelerations

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

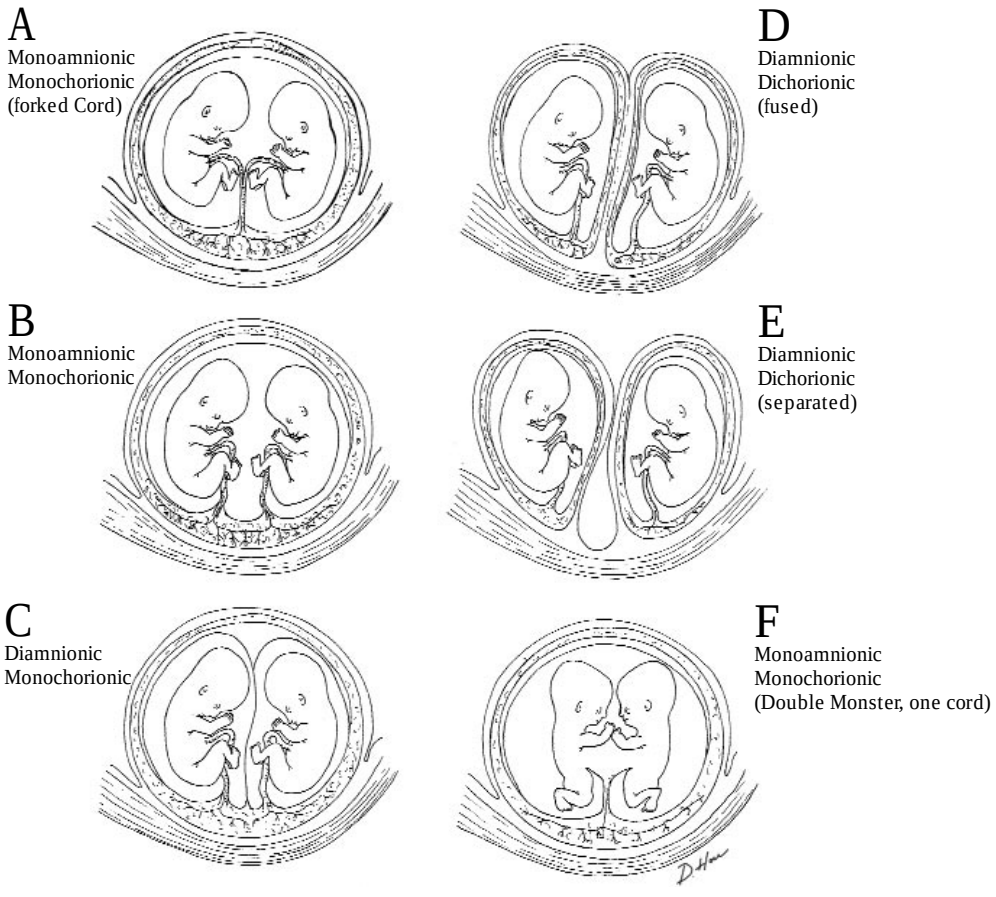


Figure 4. Classification of Twin Pregnancies

Drawing by David Hou

Table 4. Complications Associated with Multiple Gestation		
Maternal	Maternal-Fetal	Fetal
hyperemesis gravidarum DM preeclampsia / PIH PPH (uterine atony) placental abruption anemia (increased iron and folate needs) increased physiological stress on all systems increased compressive symptoms C-section	increased PROM / PTL polyhydramnios umbilical cord prolapse placenta previa	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
* most common cause of perinatal mortality in multiple gestation		

Management
- ❑ rest in T3
- ❑ increased antenatal surveillance
- ❑ close monitoring for growth (serial ultrasounds)
- ❑ vaginal examinations in third trimester to check for cervical dilatation
- ❑ may attempt vaginal delivery if twin A presents as vertex, otherwise C-section
- ❑ twin B should be delivered within 15-20 minutes after twin A (may be longer if FHR tracing adequate)

MEDICAL CONDITIONS IN PREGANCY

URINARY TRACT INFECTION

- ❑ occurs more frequently in pregnancy and puerperium
- ❑ most common medical complication of pregnancy
- ❑ due to increased urinary stasis from mechanical and hormonal (progesterone) factors
- ❑ organisms
  - same as non-pregnant woman, also GBS
- ❑ may be symptomatic or asymptomatic; treat all
- ❑ risk of acute cystitis, pyelonephritis, and possible PPROM
- ❑ treatment of asymptomatic bacteruria or acute cystitis
  - amoxicillin first line
  - alternatives are TMP-SMX (Septra) or nitrofurantoin (avoid during last 6 weeks of pregnancy)
  - follow with monthly urine cultures
  - recurrence common
- ❑ treatment of pyelonephritis during pregnancy requires hospitalization and IV antibiotics

IRON DEFICIENCY ANEMIA

- ❑ iron requirements increase during pregnancy (mother needs 1000 mg of elemental iron per fetus; this amount exceeds normal stores)
  - fetus (500 mg)
  - RBC mass (500 mg)
  - losses (200 mg)

Etiology
- ❑ inadequate iron intake
- ❑ iron malabsorption
- ❑ bleeding, vaginal or other source
- ❑ multiple gestation
- ❑ concurrent antacid use (may prevent iron absorption)

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

Diagnosis
- ❑ CBC, blood film, serum ferritin (changes in ferritin stores first sign of anemia)
- ❑ microcytic, hypochromic anemia with decreased ferritin
- ❑ morphology not good indicator because of RBC half life
- ❑ TIBC not reliable because increased during pregnancy

Treatment
- ❑ prevention
  - dietary iron and iron mobilized from stores insufficient to meet demands
  - adequate iron intake (30 mg elemental iron/day) for all women
- ❑ oral supplement of 200 mg/day of elemental iron if anemic
- ❑ monitor

FOLATE DEFICIENCY ANEMIA
- ❑ most often associated with iron deficiency anemia
- ❑ necessary for closure of neural tube during early fetal development
- ❑ minimum daily requirement is 0.5 mg
- ❑ takes approximately 18 weeks of folate deficient diet to produce anemia
- ❑ leafy green vegetables good source of dietary folate

Etiology
- ❑ malnutrition
- ❑ malabsorption (e.g. sprue)
- ❑ chronic hemolytic anemia (e.g. SCD)
- ❑ multiple gestation
- ❑ medications (i.e. phenytoin, trimethoprim-sulfamethoxazole, oral contraceptives)

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

Diagnosis
- ❑ suspect if iron deficiency anemia fails to respond to treatment
- ❑ CBC, blood film, red blood cell folate levels
- ❑ megaloblastic anemia and hypersegmented neutrophils on smear
- ❑ glossitis and skin roughness
- ❑ NO neurologic symptoms (unlike B12 deficiency)
- ❑ elevated serum iron and transferrin saturation

Treatment
- ❑ 1 mg folic acid PO daily
- ❑ 4 mg folic acid per day with past history of neural tube defect

DIABETES MELLITUS

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 (DM)
- ❑ Insulin Dependent DM (Type I)
- ❑ Non-Insulin Dependent DM (Type II)
- ❑ Gestational Diabetes: DM diagnosed during pregnancy

Complications of Pregnancy in the Diabetic
- ❑ maternal
  - hypertension/PET, polyhydramnios, pyelonephritis/UTI
  - ketoacidosis, diabetic coma, worsening retinopathy in Type I or Type II, NOT in GDM
- ❑ fetal
  - maternal hyperglycemia leads to fetal hyperinsulinism; accelerated anabolism and macrosomia result
  - increased congenital anomalies and miscarriage from preconception or T1 hyperglycemia
  - cardiac (VSD), neural tube, genitourinary, gastrointestinal and MSK (sacral agenesis) defects
  - IUGR if mother has end-organ damage
  - delayed fetal lung maturity
  - preterm labour/prematurity
  - increased incidence of stillbirth
- ❑ pregnancies complicated by GESTATIONAL diabetes 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

Treatment of DM in Pregnancy
- ❑ T1
  - see prior to pregnancy to optimize glycemic control (will reduce risk of congenital anomalies)
  - since oral hypoglycemics are contraindicated, Type II's must be switched to insulin
  - counsel re: potential complications and risks
  - advise preconception folic acid
  - see early and date pregnancy
  - consult internist and dietitian to manage insulin and diet
  - measure hemoglobin A_1C early in T1 or preconception if possible; this gives an indication of glycemic control during embryogenesis and can be used to estimate risk of birth defects
  - initial evaluations: 24 hour urine (protein and creatinine clearance), retinal exam, ECG, urine C&S, hemoglobin A_{1C}
  - throughout pregnancy monitor BP, urine dip (protein/glucose/ketones), weight gain, blood glucose (self-monitor) every visit and occasional urine C&S and hemoglobin A1C
  - in early pregnancy transfer of glucose and amino acids to the fetus results in a tendency toward maternal hypoglycemia
  - nausea and vomiting may reduce food intake, therefore may need to decrease insulin dose
- ❑ T2
  - office visits q 2 weeks
  - MSAFP (at 16 weeks) and 3 detailed U/S examinations
  - consider fetal echocardiography to exclude congenital heart defect
  - admit for blood sugar control if needed
  - in the second half of pregnancy, the diabetogenic action of placental hormones outweigh the continuous siphoning of glucose by the fetus
  - demand for insulin is increased, hence insulin dosages need to be increased
- ❑ T3
  - office visits q 1 week
  - fetal surveillance (BPP, NST); frequency depends on risk
    - < 36 weeks = q weekly
    - > 36 weeks = q weekly or biweekly

- timing of delivery dependent upon fetal and maternal risk factors
- can wait for spontaneous labour if glucose well-controlled and BPP normal
- induce by 40 weeks

❑ labour
- increased risk of CPD, shoulder dystocia with babies weighing over 4000 g
- elective C-section for predicted birthweights of greater than 4500 g (controversial)
- during labour monitor sugars q1h with patient on insulin and dextrose drip and aim for blood sugar of 3.5 to 6.5 to reduce the risk of neonatal hypoglycemia

❑ postpartum
- increased risk of hypoglycemia
- once eating a regular diet, resume insulin at two-thirds of prepregnancy dose and monitor q6h

GESTATIONAL DIABETES

❑ glucose intolerance that is present only during pregnancy
❑ genetic predisposition to the development of glucose intolerance exists in this population of women
❑ 50% risk of developing Type II DM in next 20 years

Risk Factors

❑ age > 30
❑ previous history of high blood glucose, GDM, or macrosomic infant (> 4.5 kg)
❑ positive family history (GDM, Type II DM, macrosomic infant)
❑ excessive weight gain in pregnancy, prepregnancy obesity
❑ baby > 4.5 kg or large for GA
❑ previous unexplained stillbirth
❑ previous congenital anomaly
❑ early preeclampsia or polyhydramnios
❑ repeated vaginal candidiasis
❑ member of high risk ethnic group
❑ multiple gestation

Diagnosis

❑ screen at 26 weeks (or earlier) with 50 g oral glucose challenge test if risk factors or glycosuria are present
❑ > 7.8 mmol/L at 1 hour is abnormal
❑ confirm with 3 hour 100 g oral glucose tolerance test (OGTT)
- need 2 out of 4 values to be abnormal to diagnose GDM
  - fasting: > 5.8 mmol/L
  - 1 hour: > 10.6 mmol/L
  - 2 hour: > 9.2 mmol/L
  - 3 hour: > 8.1 mmol/L

Management of Gestational Diabetes

❑ controversial
❑ aim to achieve normal blood sugars post-prandial (i.e. < 6.7 mmol/L)
❑ start with diabetic diet
❑ if blood sugars 2 hours post-prandial are > 6.7, add insulin (Humulin)
❑ 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
❑ follow-up testing recommended postpartum because of increased risk of overt diabetes (i.e. OGTT at 6 weeks postpartum)

HYPERTENSIVE DISORDERS OF PREGNANCY

Classification

❑ preeclampsia/eclampsia
❑ chronic hypertension
❑ chronic hypertension with superimposed preeclampsia/eclampsia
❑ transient or gestational hypertension

Preeclampsia/Preeclamptic Toxemia/Eclampsia (PET)
- ❑ hypertension accompanied by proteinuria and/or non-dependent edema with onset > 20 weeks
  - BP > 140/90 mmHg or an increment of 30 mmHg systolic and 15 mmHg diastolic over a nonpregnant or T1 BP
  - non-dependent edema (e.g. face, hands) is generalized and usually associated with excessive weight gain (> 2 kg/week)
  - proteinuria is defined as > 1+ protein on random dipstick analysis or > 300 mg in a 24 hour urine collection
- ❑ 50% of all hypertension in pregnancy
- ❑ due to an imbalance of thromboxane (vasoconstrictor) and prostaglandin (vasodilator), causing generalized arteriolar constriction
- ❑ resultant vasospasm damages capillaries, leading to protein extravasation and hemorrhage

Conditions Associated with Preeclampsia/Eclampsia
- ❑ maternal factors
  - 80-90% of cases in primagravidas
  - past history or family history of preeclampsia/eclampsia
  - diabetes, chronic hypertension, or renal disease
  - extremes of maternal age
- ❑ fetal factors
  - IUGR
  - hydatidiform mole
  - > 1 fetus
  - fetal hydrops

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

Maternal Complications
- ❑ 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

Mild Preeclampsia
- ❑ PET uncomplicated by neurologic symptoms or criteria for a diagnosis of severe PET

Severe Preeclampsia
- ❑ PET complicated by at least two of the following
  - BP > 160/110
  - congestive heart failure
  - pulmonary edema or cyanosis
  - proteinuria > 5 g/24 hours or > 2+ on dipstick
  - elevated serum creatinine
  - oliguria (< 400 mL/24 hours)
  - thrombocytopenia (< 100 000 - 150 000/mL)
  - ascites
  - RUQ or epigastric pain (subcapsular hemorrhage)
  - elevated liver enzymes
  - hyperbilirubinemia
  - headache (cerebral artery vasospasm)
  - visual disturbances (i.e. scotomas, loss of peripheral vision)
  - hyperreflexia, clonus
  - IUGR

Eclampsia
- ❑ grand mal seizures in a woman with preeclampsia

Management of Mild Preeclampsia
- ❑ 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
    - urinalysis for protein and casts
    - 24 hour urine for protein and creatinine clearance
- ❑ fetal evaluation of FHR, NST, BPP
- ❑ 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 Preeclampsia
- ❑ stabilize and deliver; the only "cure" is delivery
- ❑ 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 q 12 hours for protein
  - vitals and DTR q 1 hour
- ❑ fetal evaluation
  - NST followed by continuous electronic fetal monitoring until delivery
- ❑ anticonvulsant therapy
  - given to increase seizure threshold
  - baseline magnesium blood level
  - magnesium sulphate (4g IV push) 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 (> 13 mg % serum level)
  - depression of DTR
  - depression of RR < 10/minute
  - decreased muscle tonicity
  - CNS or cardiac depression
  - antagonist to magnesium sulphate is calcium gluconate (10%) 10 mL IV 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 push over 5 minutes q 15 - 30 minutes until desired effect (an arteriolar vasodilator with minimal venous effect)
  - controls BP for hours not days (deliver as soon as possible)
  - next dose is given ~6 hours later with BP readings q 15 minutes
  - also used in postpartum state if BP remains elevated and urinary output < 25 mL/hour
  - second line: labetalol 20 - 50 mg IV q 10 minutes
  - third line: nifedipine (Adalat) 10 -20 mg po q 20 - 60 minutes (puncture capsule and swallow liquid)
- ❑ 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 Notes)
  - 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

Chronic Hypertension
- ❑ features
  - history of hypertension (> 140/90) before gestation
  - detection of hypertension 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
  - no ACE inhibitors, diuretics, propranolol

Chronic Hypertension with Superimposed Preeclampsia/ Eclampsia
- ❑ 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

Transient or Gestational Hypertension
- ❑ hypertension alone that develops during the latter half of pregnancy or during the first 24 hours after delivery and disappears within 10 days following parturition
- ❑ monitor for signs of preeclampsia/eclampsia

HYPEREMESIS GRAVIDARUM

Definition
- ❑ intractable nausea and vomiting to extent of weight loss, dehydration and 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 ßhCG or estrogen levels are implicated

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

Differential Diagnosis of Nausea and Vomiting
- ❑ hyperemesis is a diagnosis of exclusion
- ❑ GI inflammation/infection
  - appendicitis
  - cholecystitis
  - hepatitis

- gastroenteritis
- pancreatitis
- PUD
- fatty liver of pregnancy

❑ pyelonephritis
❑ thyrotoxicosis
❑ multiple gestation
❑ GTN (see Gynecology Notes)
❑ 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...)

Treatment
❑ 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
- dimenhydrinate (Gravol)
- vitamin B6 and doxylamine succinate (Diclectin)

❑ 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)

ISOIMMUNIZATION

❑ 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
❑ other antibodies can lead to fetal red blood cell hemolysis
- much less common and no prophylaxis is available

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

❑ complications
- anti-Rh Ab can cross the placenta and cause fetal hemolysis resulting in fetal anemia, CHF, edema, and ascites
- severe cases can lead to fetal hydrops (total body edema), or erythroblastosis fetalis

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 24 weeks onwards
❑ Liley curve is used to determine bilirubin level and appropriate management (see below)
❑ 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

Prophylaxis
- ❑ Rhogam binds to Rh Ag of fetus and prevents it from contacting maternal immune system
- ❑ Rhogam must be given to all Rh negative women
  - at 28 weeks
  - within 48 hours of the birth of an Rh positive fetus
  - positive Kleihauer-Betke test
  - with any invasive procedure in pregnancy
  - in the case of ectopic pregnancy
  - with miscarriage, therapeutic abortion
  - 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)

Treatment
- ❑ 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)
- ❑ 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

INFECTIONS DURING PREGNANCY

Toxoplasmosis
- ❑ protozoal infection (*Toxoplasma gondii*)
- ❑ incidence: 1/1000 pregnancies
- ❑ source: raw meat, unpasteurized goat's milk, cat urine/feces
- ❑ greatest risk of transmission in T3
- ❑ severity of fetal infection greatest in T1
- ❑ 75% asymptomatic at birth, but may later develop sequelae
- ❑ risk of congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial calcifications, MR, microcephaly) if primary maternal infection during pregnancy
- ❑ diagnosis based on serologic testing for both IgM and IgG
- ❑ confirmation of diagnosis based on presence of IgM antibodies in cord blood
- ❑ self-limiting infection, spiramycin (macrolide) decreases fetal morbidity

Rubella
- ❑ RNA togavirus with transmission by respiratory droplets (highly contagious)
- ❑ maternal infection during pregnancy (greatest in T1) may cause spontaneous abortion or Congenital Rubella Syndrome: hearing loss, cataracts, cardiovascular lesions, MR, symmetric IUGR, hepatitis, CNS defects and osseous changes
- ❑ diagnosis best made by serologic testing
- ❑ all pregnant women screened for rubella immunity (rubella titer > 1:16 = immune)
- ❑ non-immune
  - should be offered vaccination following pregnancy (not a contraindication for breast feeding)
  - rubella vaccine should be avoided before (3 months) or during pregnancy since it is an attenuated live vaccine

Cytomegalovirus
- ❑ DNA virus (herpes family)
- ❑ transmission: blood transfusion, organ transplant, sexual contact, breast milk, transplacental, or direct contact during delivery
- ❑ congenital infection can occur from primary or re-infection of the mother
- ❑ increased fetal morbidity with primary infection
- ❑ risk of transmission constant across trimesters
- ❑ 5-10% of fetuses infected in utero will develop neurologic involvement (MR, cerebral calcification, hydrocephalus or microcephaly, deafness, chorioretinitis)
- ❑ diagnosis: isolation of virus in urine culture (or culture of other secretions), serologic screening for antibodies

Herpes
- ❑ DNA herpes virus
- ❑ transmission: intimate mucocutaneous contact
- ❑ primary infection during pregnancy increases risk of neonatal complications
- ❑ 50% transmission if primary infection, 4% transmission if secondary recurrence
- ❑ infection to fetus may occur in utero but more commonly occurs during delivery
- ❑ C-section if active genital lesions present within 4-6 hours of ROM, even if lesions remote from vulvar area

Syphilis
- ❑ *Treponema pallidum*
- ❑ may have transplacental transmission
- ❑ serological tests
  - VDRL screening done at first prenatal visit (non-specific)
- ❑ to confirm a positive VDRL
  - TPHA (Treponema Pallidum Hemagglutinating Ab)
  - FTA-ABS (Fluorescent Treponema Antibody Absorption) Test
- ❑ risk of preterm labour, fetal death
- ❑ treatment: Penicillin G 2.4 million units IM, monthly VDRL during pregnancy to ensure treatment is adequate

Hepatitis B
- ❑ transmitted via blood, saliva, vaginal secretions, semen, breast milk, transplacental
- ❑ fetal infection most likely with T3 maternal infection
- ❑ risk of vertical transmission 10% if asymptomatic HBsAg +ve
- ❑ risk of vertical transmission 85-90% if HBsAg +ve and HBcAg +ve
- ❑ chronic active hepatitis increases risk of prematurity, low birth weight, neonatal death
- ❑ treatment of neonate with Hep B immune globulin (HBIG) and vaccine (at birth, one and six months) is 90% effective
- ❑ vaccine safe during pregnancy

Erythema Infectiosum (Fifth Disease)
- ❑ parvovirus B19
- ❑ febrile illness with bilateral erythema of cheeks (“slapped cheek” rash) followed by maculopapular rash of trunk and extremities
- ❑ fetus of infected woman may develop hydrops in utero
  - follow fetus with weekly U/S (if hydrops occurs, consider fetal transfusion)
- ❑ risk of intrauterine death 1-12 weeks after infection

HIV
- ❑ offer screening to all women
- ❑ risk of vertical transmission 12 to 28%; more likely if maternal CD4 count < 300
- ❑ risks to infected mom include decreased CD4 count, cancer, increased opportunistic infection (PCP, TB, CMV, toxoplasmosis, mycoplasma)
- ❑ care of HIV positive patient
  - PCP prophylaxis with Bactrim if CD4 < 200
  - AZT shown to decrease transmission to fetus from 25% to 8% risk
  - exclude cervical dysplasia
  - toxoplasmosis and CMV antibodies

Group B Streptococccus (see Prenatal Care Section)

Table 5. Surgical Conditions in Pregnancy	
Acute	Nonemergent
acute appendicitis acute cholecystitis acute pancreatitis abdominal trauma torsion of the uterine adnexa pelvic abcess peptic ulcer disease bowel obstruction intracranial hemorrhage thromboembolic disease	adnexal tumours cervical cancer breast cancer gastrointestinal trauma melanoma, osteosarcoma

ACUTE SURGICAL CONDITIONS

- ❑ incidence is approximately 1 in 500 pregnancies
- ❑ generally manage acute surgical condition regardless of pregnancy
- ❑ pregnancy substantially increases complications associated with surgery

NON-EMERGENCY SURGICAL CONDITIONS

- ❑ surgery in first trimester has highest risk of teratogenicity and spontaneous abortion
- ❑ surgery for nonemergent conditions usually delayed until the more stable second trimester

ANTENATAL HEMORRHAGE

FIRST AND SECOND TRIMESTER BLEEDING

Differential Diagnosis

- ❑ abortion (threatened, inevitable, incomplete, complete)
  - • < 5% of threatened abortions go on to abort (see Table 6)
- ❑ abnormal pregnancy (ectopic, molar)
  - • ectopic, molar (see Gynecology Notes)
- ❑ trauma (post-coital)
- ❑ physiologic bleeding (due to placental development)
- ❑ genital lesion (e.g. cervical polyp, neoplasms)

Table 6. 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 (experimental)
  - > 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
  - perforation of uterus
  - hemorrhage
  - laceration of cervix
  - risk of sterility
  - infection - usually due to retained products, occasionally endometritis
  - Asherman's syndrome (fibrosis of the uterus)

THIRD TRIMESTER BLEEDING

Differential Diagnosis

- ❑ 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
- ❑ NB - do NOT perform a vaginal exam until placenta previa has been ruled out by U/S

PLACENTA PREVIA

- ❑ abnormal location of the placenta at or near the internal cervical os
- ❑ 1/200 at time of delivery
- ❑ many are low lying in early pregnancy but due to development of lower uterine segment appear to "move upward" as pregnancy nears term
- ❑ 95% of previas diagnosed in T2 resolve by T3; repeat U/S at 30-32 weeks GA

Classification

- ❑ total
  - placenta completely covers the internal os
- ❑ partial
  - placenta partially covers the internal os
- ❑ marginal
  - placenta reaches margin but does not cover any part of the intemal os
- ❑ low lying (NOT a previa)
  - placenta in lower segment but clear of os
  - can also bleed, usually later (i.e. in labour)

Etiology

- ❑ unknown but many associated conditions and risk factors
  - multiparity
  - history of placenta previa (4-8% recurrence risk)
  - multiple pregnancy
  - increased maternal age
  - uterine scar due to previous abortion, C-section, D&C, myomectomy
  - uterine tumour (e.g. fibroids) or other uterine anomalies

Fetal Complications

- ❑ 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 Complications

- ❑ < 1% maternal mortality
- ❑ hemorrhage and hypovolemic shock
- ❑ anemia
- ❑ acute renal failure
- ❑ pituitary necrosis (Sheehan syndrome)
- ❑ PPH (because lower uterine segment is atonic)
- ❑ hysterectomy
- ❑ placenta accreta

Clinical Features

- ❑ 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 before
  - 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
- ❑ diagnosed by U/S (95% accuracy with transabdominal)

Management

- ❑ maternal stabilization; large bore IV with hydration
- ❑ electronic fetal monitoring

- ❑ maternal monitoring
  - vitals, urine output, blood loss
  - bloodwork including hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match
- ❑ when fetal and maternal condition permit, perform careful U/S examination to determine fetal viability, gestational age and placental status/position
- ❑ Rhogam given if mother is Rh negative
- ❑ management decision depends on
  - previa characteristics (amount of bleeding, degree of previa)
  - fetal condition (GA, level of distress, presentation)
  - uterine activity
- ❑ 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
- ❑ delivery if bleeding is profuse, GA > 36 weeks, or L/S ratio is 2:1 or greater
  - usually C-section (incision site dictated by location of previa)

ABRUPTIO PLACENTAE

- ❑ premature separation of a normally implanted placenta after 20 weeks gestation
- ❑ incidence = 0.5-1.5%

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

Etiology

- ❑ unknown, but associated with
  - maternal hypertension (chronic or PIH) in 50% of abruptions
  - multiparity
  - previous abruption (recurrence rate 10%)
  - PROM
  - maternal age > 35 (felt to reflect parity)
  - maternal vascular disease
  - cigarette smoking
  - alcohol consumption
  - uterine distension (polyhydramnios, multiple gestation)
  - short cord
  - trauma
  - sudden decompression of the uterus (twins)
  - uterine anomaly, fibroids

Fetal Complications

- ❑ perinatal mortality 25-60%
- ❑ prematurity
- ❑ intrauterine hypoxia

Maternal Complications

- ❑ < 1% maternal mortality
- ❑ DIC (in 20% of abruptions)
- ❑ acute renal failure
- ❑ anemia
- ❑ hemorrhagic shock
- ❑ pituitary necrosis (Sheehan syndrome)
- ❑ amniotic fluid embolus

Clinical Features

- ❑ PAINFUL vaginal bleeding; blood may be bright red or dark or clotted
- ❑ uterine tenderness and increased tone

- ❑ degree of anemia may not correlate with degree of observed blood loss
- ❑ fetal distress; loss of variability, late decelerations (see Fetal Monitoring Section)
- ❑ 15% present with fetal demise

Diagnosis
- ❑ clinical
- ❑ U/S NOT helpful except to rule out placenta previa

Management
- ❑ initial management
  - 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-section if fetal or maternal distress develops with fluid/blood replacement, labour fails to progress or non-cephalic fetal presentation

VASA PREVIA

- ❑ incidence 1 in 5000
- ❑ occurs with velamentous insertion of cord into membranes of placenta; unprotected fetal vessels pass over the cervical os
- ❑ since bleeding is from fetus a small amount of blood loss can have catastrophic consequences
  - presents with painless vaginal bleeding and fetal distress (tachy- to bradyarrhythmia)
- ❑ 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 is STAT C-section
- ❑ 50% perinatal mortality, increasing to 75% if membranes rupture (most infants die of exsanguination)

INTRA-UTERINE GROWTH RESTRICTION

Definition
- ❑ infants whose weight is < 10th %ile for a particular GA
- ❑ weight not associated with any constitutional or familial cause
- ❑ prone to problems such as meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation
- ❑ greater risk of perinatal morbidity and mortality

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

Clinical Features
- ❑ symmetric/Type I (20%)
  - occurs early in pregnancy
  - inadequate growth of head and body although the 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

Diagnosis
- ❑ clinical suspicion
- ❑ 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
- ❑ prevention via risk modification prior to pregnancy ideal
- ❑ most important consideration is accurate menstrual history and GA in which to assess the above data
- ❑ 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-section since IUGR fetus withstands labour poorly

MACROSOMIA

Definition
- ❑ fetal weight > 90th %ile for GA, > 4000 g at term

Clinical Features
- ❑ maternal associations
  - obesity
  - DM
  - past history of macrosomic infant
  - prolonged gestation
  - multiparity

Diagnosis
- ❑ maternal history for associated conditions
- ❑ 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

Complications
- ❑ increased risk of perinatal mortality
- ❑ fetopelvic disproportion and shoulder dystocia more common
- ❑ complications of DM in labour (see Medical lllnesses in Pregnancy Section)

POLYHYDRAMNIOS

Definition
- ❑ amniotic fluid volume > 2 L at any stage in pregnancy
- ❑ > 8 cm x 8 cm pocket on U/S
- ❑ 1/250 deliveries
- ❑ up to 2/3 of fetuses with severe polyhydramnios have chromosomal problems

Causes
- ❑ idiopathic
  - most common
- ❑ maternal
  - DM: causes abnormalities of transchorionic flow (IDDM)
- ❑ maternal-fetal
  - chorioangiomas, multiple gestation, erythroblastosis
- ❑ fetal
  - chromosomal anomaly
  - respiratory - cystic adenomatoid malformed lung
  - CNS (anencephaly, hydrocephalus, meningocele)
  - GI (tracheoesophageal fistula, duodenal atresia)
  - facial clefts, neck masses (interfere with swallowing)

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

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

Management
- ❑ find cause (40% idiopathic)
- ❑ complete fetal U/S evaluation
- ❑ mild to moderate cases require no treatment
- ❑ screen for maternal disease/infection as indicated
- ❑ if severely symptomatic, hospitalize and consider therapeutic amniocentesis
- ❑ admit if contracting or cervix dilating

OLIGOHYDRAMNIOS

Definition
- ❑ amniotic fluid index of 5 cm or less

Etiology of Early Onset Oligohydramnios
- ❑ decreased production
  - renal agenesis or dysplasia, urinary obstruction, posterior urethral valves (male)

- ❑ increased loss
  - prolonged amniotic fluid leak (although most often labour ensues)
- ❑ 15-25% of cases have fetal anomalies

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

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 (see below)
  - fetal monitoring (NST, CTG, BPP)
  - consider delivery if at term

ANTENATAL COMPLICATIONS

PRETERM LABOUR

Definition
- ❑ labour occurring between 20 and 37 weeks gestation
- ❑ complicates about 10% of pregnancies
- ❑ prematurity is the leading cause of perinatal morbidity and mortality
  - at 30 weeks or 1500 g = 90% survival
  - at 33 weeks or 2000 g = 99% survival
- ❑ major causes of morbidity = asphyxia, sepsis, RDS
- ❑ intrapartum asphyxia may lead to cerebral hemorrhage

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

- ❑ maternal-fetal
  - PPROM (a common cause)
  - polyhydramnios
- ❑ fetal
  - multiple gestation
  - congenital abnormalities of fetus

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)
- ❑ availability of necessary personnel and equipment to assess mother and fetus during labour and care for baby of the predicted GA if therapy fails

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)

Diagnosis

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

Prevention

- ❑ 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

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
- ❑ aggressiveness depends on the GA
- ❑ tocolytic agents - if no contraindications present
  - have no impact on neonatal morbidity or mortality but may buy time to allow celestone use or to transfer to appropriate centre
  - beta-mimetics: ritodrine, terbutaline
  - magnesium sulphate (if diabetes or cardiovascular disease present)
  - calcium channel blockers: nifedipine
  - PG synthesis inhibitors (2nd line agent): indomethacin

Enhancement of Pulmonary Maturity
- ❑ most effective between 28 and 34 weeks gestation
- ❑ treatment: betamethasone valerate (Celestone) 12 mg IM q12h times 2
- ❑ wait 24 hours for delivery
- ❑ specific maternal contraindications
    - active TB
    - viral keratosis
    - maternal DM

RUPTURE OF MEMBRANES

Premature ROM
- ❑ 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)

PPROM
- ❑ preterm premature rupture of membranes (not in labour)

Associated Conditions
- ❑ congenital anomaly
- ❑ infection

Causes
- ❑ idiopathic (most common)
- ❑ frequently associated with
    - multiparity
    - cervical incompetence
    - infection: cervicitis, vaginitis, STD, UTI
    - multiple gestation
    - family history of PROM
    - low socioeconomic class/poor nutrition
    - and other risk factors associated with PTL (see above)

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

Diagnosis
- ❑ history of fluid gush or continued leakage
- ❑ avoid introducing infection with examinations (do not do a digital pelvic exam)
- ❑ sterile speculum exam
    - pooling of fluid in the posterior fornix
    - may observe fluid leaking out of cervix on valsalva
- ❑ 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

Management
- ❑ 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 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

UMBILICAL CORD PROLAPSE

❑ descent of the cord to a level adjacent to or below the presenting part causing cord compression between presenting part and pelvis
❑ visible or palpable cord
❑ FHR changes (variable decelerations, bradycardia or both)
❑ increased incidence with prematurity/PROM, fetal malpresentations, low-lying placenta, polyhydramnios, multiple gestation, CPD

Management

❑ "STAT" C-section
❑ 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

CHORIOAMNIONITIS

❑ definition: infection of the chorion, amnion and amniotic fluid
❑ risk factors: prolonged ROM, long labour, multiple vaginal exams during labour, internal monitoring, bacterial vaginosis and other vaginal infections
❑ clinical features: 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 and Clinical Features

❑ pregnancy beyond 42 weeks (10% of pregnancies)
❑ accurate dating essential
❑ etiology unknown
❑ morbidity increased with hypertension/PET, DM, abruption, IUGR and multiple gestation

Complications

❑ perinatal mortality 2-3 x 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)

Management

❑ fetal movement count by the mother
❑ BPP twice weekly from 40 weeks
❑ delivery after 41 weeks if not already in labour since perinatal mortality is higher secondary to progressive uteroplacental insufficiency
❑ if BPP < 10/10 at any time, deliver
❑ decreased tolerance for asphyxia intrapartum

INTRAUTERINE FETAL DEATH

❑ incidence = 1% of pregnancies

Etiology

❑ unknown in 50%
❑ hypertension, DM
❑ erythroblastosis fetalis
❑ congenital anomalies
❑ umbilical cord or placental complications
❑ intrauterine infection
❑ antiphospholipid Ab's

Clinical Features

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

Management

❑ labour induction (see Abnormal Labour Section)
❑ must monitor for maternal coagulopathy (10% risk of DIC)
❑ psychologic aspects of fetal loss
❑ investigations to determine cause
❑ subsequent pregnancies high risk

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-section if they don't convert)

Fetal Presentation

❑ refers to the fetal part presenting at pelvic outlet
  • breech (complete, frank, footling)
  • cephalic
    • vertex
    • brow
    • face
    • sinciput
  • shoulder
  • compound (fetal extremity prolapses along with presenting part)
❑ all except vertex 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-section)
  - face presentation: head fully extended (mentum posterior always requires C-section, mentum anterior will deliver vaginally)

Station
- ❑ defined by position of presenting part relative to ischial spines
  - at ischial spines = station 0 = engaged

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

DEFINITION OF LABOUR
- ❑ regular, painful uterine contractions increasing in frequency, accompanied by progressive DILATATION and EFFACEMENT of cervix, and 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 and painless, occur throughout pregnancy and not associated with any dilatation, effacement or descent

FOUR STAGES OF LABOUR

First Stage of Labour (see Table 7)
- ❑ 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 multiparas)
  - phase of maximum slope on Friedman curve (see Figure 6)
  - painful, regular contractions ~q 2 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

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 7. 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

THE 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)

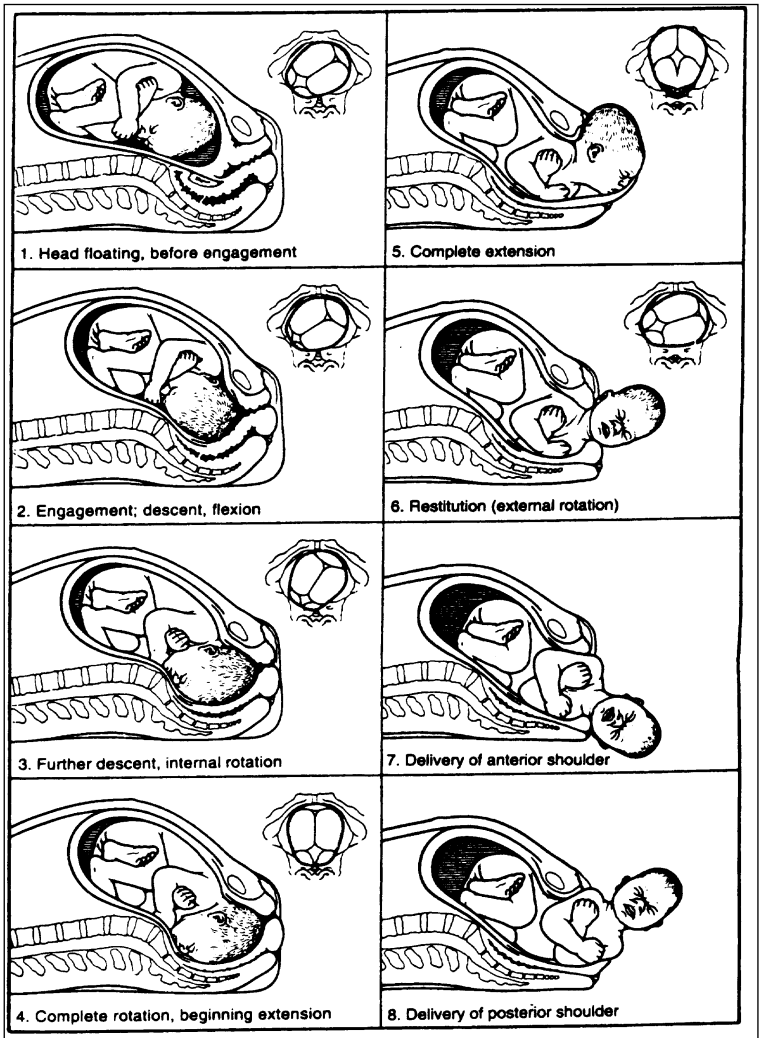


Figure 5. Movements of the Fetus During Labour and Delivery, Left Occiput Anterior Position

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

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

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 CPD
  - active maternal genital herpes
- ❑ maternal-fetal
  - placenta or vasa previa
- ❑ fetal
  - distress
  - malpresentation

Cervical Ripening Principles
- ❑ 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 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
- ❑ Foley catheter may be used to mechanically dilate the cervix

Medical
- ❑ oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 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) - 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 q 20-30 minutes to a maximum of 36-48 mU/minute
- half-life of oxytocin is ~2 minutes (thus need continuous drip because effects wear off fast)

ABNORMAL PROGRESS 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

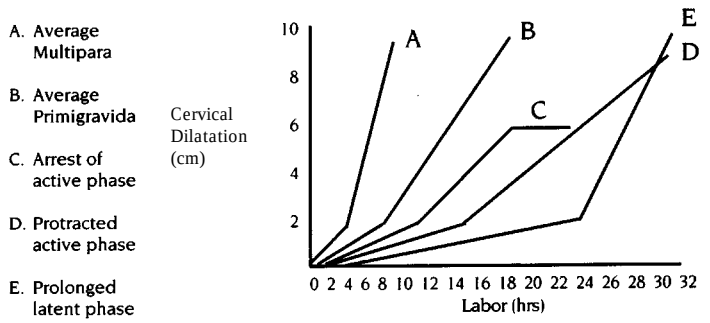


Figure 6. Normal and Abnormal Courses of the First Stage of Labour (Friedman Curve)

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)
- too early and too heavy sedation is present in 30-40% of these patients
- careful search for factors of CPD should be made
- treatment: oxytocin augmentation if diagnosis of labour is certain otherwise rest +/– sedation

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 CPD will be present so that secondary arrest of dilatation usually develops
- 2/3 will progress steadily through labour with ultimate uneventful vaginal delivery
- treatment: oxytocin augmentation if contractions are inadequate (see Augmentation of Labour Section) and/or amniotomy

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-section)
- ❑ CPD if adequate contractions measured by intrauterine pressure catheter (IUPC) and no descent/dilatation for > 2 hours
- ❑ if CPD ruled out, IV oxytocin and amniotomy can be attempted

SHOULDER DYSTOCIA

Definition, Incidence and Complications

- ❑ impaction of anterior shoulder of fetus against symphysis pubis after fetal head has been delivered (life threatening emergency)
- ❑ occurs when breadth of shoulders is greater than biparietal diameter of the head
- ❑ incidence is 0.15-1.4% of deliveries
- ❑ watch for "turtle sign" (head advances during contraction but returns to previous position at end of contraction)
- ❑ 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

Associated Conditions

- ❑ maternal
  - maternal obesity
  - diabetes
  - multiparity
- ❑ fetal
  - prolonged gestation
  - macrosomia
- ❑ labour
  - prolonged 1st and 2nd stages
  - prolonged deceleration phase (8-10 cm)
  - instrumental midpelvic delivery

Management

- ❑ goal: to displace anterior shoulder from behind symphysis pubis
- ❑ initial gentle traction with maternal pushing
- ❑ adequate analgesia
- ❑ apply suprapubic pressure (to dislodge shoulder) with downward traction
- ❑ ask for help
- ❑ legs into hyperflexion on maternal abdomen (McRobert maneuver)
- ❑ anterior shoulder disimpaction
- ❑ release posterior shoulder (deliver posterior arm and shoulder)
- ❑ maneuver of Wood's corkscrew (insert hand beyond occiput into vagina and push anterior shoulder forward to the oblique or push the posterior shoulder through a 180 degree arc to reduce the biacromial diameter presented to the pelvic inlet)
- ❑ episiotomy (midline)
- ❑ cleidotomy: deliberate fracture of the clavicle (last resort)
- ❑ Zavanelli maneuver (involves flexion of the fetal head, replacement of the fetus within the uterine cavity and emergent C-section; reported success in a small series)

BREECH PRESENTATION

Definition

- ❑ fetal buttocks is the presenting part

Incidence

- ❑ occurs in 3-4% of pregnancies at term (25% before 28 weeks)

Classification (see Figure 7)

- ❑ complete: flexion at hips and knees

- ❑ frank: flexion at hips, extension at knees
  - most common type of breech presentation
  - only breech presentation delivered vaginally
- ❑ footling: may be single or double with extension at hip(s) and knee(s) so that foot is the presenting part

Etiology

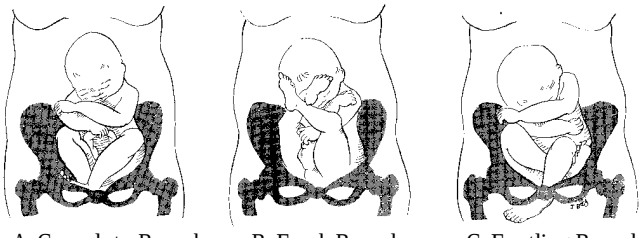
- ❑ maternal
  - pelvis (contracted)
  - uterus (shape abnormalities, intrauterine tumours, fibroids extrauterine tumours causing compression)
  - grand multiparity
- ❑ maternal-fetal
  - placenta (previa)
  - amniotic fluid (poly/oligohydramnios)
- ❑ fetal
  - prematurity
  - multiple gestation
  - congenital malformations (found in 6% of breeches; 2-3 x the incidence in vertex presentations)

Diagnosis

- ❑ 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-section, previous myomectomy, oligohydramnios, PROM, placenta previa, abnormal U/S, suspected IUGR
  - risks: abruption, cord compression
  - method: tocolysis, followed by transabdominal manipulation of fetus, guided by ultrasound
  - if patient Rh negative Rhogam given prior to procedure
- ❑ good prognostic factors (for a successful version)
  - multiparous
  - good fluid volume
  - small baby
  - skilled obstetrician
- ❑ criteria for vaginal delivery
  - frank breech, GA > 36 weeks
  - EBW 2200-3800 g based on clinical and U/S assessment
  - fetal head flexed
  - continuous fetal monitoring
  - maternal pelvis adequately large (clinically, or “proven” by previous delivery)
  - no other indication for C/S
  - experienced obstetrician
- ❑ C-section for all other presentations (except mentoanterior face presentation)



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-section (generally 60-80%)
- ❑ risk of uterine rupture (< 1% with low transverse incision)

Contraindications

(i.e. need to do a repeat C/S 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 > 4000 g
- ❑ non-vertex presentation or placenta previa
- ❑ inadequate facilities or personnel for emergency C-section

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 prior with a classical incision

Complications

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

Management

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

AMNIOTIC FLUID EMBOLUS

Definition

- ❑ amniotic fluid debris in maternal circulation
- ❑ rare intrapartum or immediate postpartum complication
- ❑ 80% mortality

Presentation

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

Risk Factors

- ❑ placental abruption
- ❑ rapid labour
- ❑ multiparity
- ❑ uterine rupture

Treatment

- ❑ supportive measures, coagulopathy correction

INDICATIONS FOR OPERATIVE VAGINAL DELIVERY

- ❑ operative vaginal delivery is with forceps or vacuum extraction
- ❑ 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

Low Forceps

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

Mid Forceps

- ❑ presenting part below spines but not yet visible at introitus
- ❑ not below 2+ spines

Types of Forceps

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

Absolute Prerequisites

- ❑ vertex, face or breech presentations
- ❑ fully dilated cervix
- ❑ empty bladder (risk of tear if full)
- ❑ adequate analgesia
- ❑ ruptured membranes
- ❑ position and station are known
- ❑ presenting part below ischial spines
- ❑ experienced obstetrician
- ❑ pelvis of adequate size and shape
- ❑ uterine contractions present
- ❑ facilities to perform emergency C-section if needed

VACUUM EXTRACTION

- ❑ traction instrument used as alternative to forcep 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 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 lumen

EPISIOTOMY

Definition
- ❑ making an incision in the perineal body at the time of delivery
- ❑ midline (better healing, increased risk of deep tear) vs. mediolateral (less risk of extensive tear, poorer healing/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

CESAREAN DELIVERY

Indications
- ❑ maternal
  - obstruction, active herpetic lesion on vulva, invasive cervical cancer, previous uterine surgery
- ❑ 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)
- ❑ 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 S1-S5
  - contraction of the uterus
  - distention 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 (treatment with naloxone)
- side effects of 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
- 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-section 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, 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-section 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 1000 mL IV fluid

General Anesthesia

❑ not used for vaginal deliveries
❑ 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

Notes

DEFINITION
- ❑ period of adjustment after pregnancy when anatomic and physiologic changes are reversed
- ❑ immediate - first 24 hours after delivery
- ❑ early - first week
- ❑ traditionally, puerperium lasts 6 weeks

POST-DELIVERY EXAMINATION
- ❑ The 8 B's: Blues (post-partum), Breathing (DVT/PE), Breast, Belly, Bowels, Bladder, Bleeding, Baby

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

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

PUERPERAL COMPLICATIONS

RETAINED PLACENTA
- ❑ placenta undelivered after 30 minutes
- ❑ risk factors: placenta previa, prior C/S, post-pregnancy curettage, prior manual placental removal, uterine infection
- ❑ 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
- ❑ increased risk of infection or bleeding
- ❑ 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

UTERINE INVERSION
- ❑ uterus prolapses through the cervix and passes out of the vaginal introitus
- ❑ often iatrogenic (excess cord traction)
- ❑ more common in grand multiparous (lax uterine ligaments)

- ❑ urgent management essential (may require general anesthetic if unsuccessful)
  - replace uterus without removing placenta
  - remove placenta manually and withdraw slowly
  - IV oxytocin infusion
  - re-explore uterus

POSTPARTUM PYREXIA

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

Causes
- ❑ Wind
  - atelectasis (especially after general anesthesia)
  - pneumonia
- ❑ Water (UTI)
- ❑ Wound (gram +/–, aerobes, and anaerobes)
  - C-section 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 after delivery (most women probably lose > 500 mL, often underestimated)

Etiology (4 T's)
- ❑ tone: uterine atony (most common cause of PPH)
  - 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)
  - maintain fibrinogen > 1000 mg/mL, platelets > 50 000

Management
- ❑ determine cause, call for help
- ❑ supportive
  - ABC's, fluid, +/– transfusions, +/– other blood products
- ❑ 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

- ❑ investigations
  - pelvic U/S if indicated to look for cause
- ❑ 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)
  - 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 gract lacerataions
  - D&C (beware of vigorous scraping which may cause Asherman)
  - 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 Notes)
  - incidence of 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 can occur in the context of a 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)

OTHER DRUGS

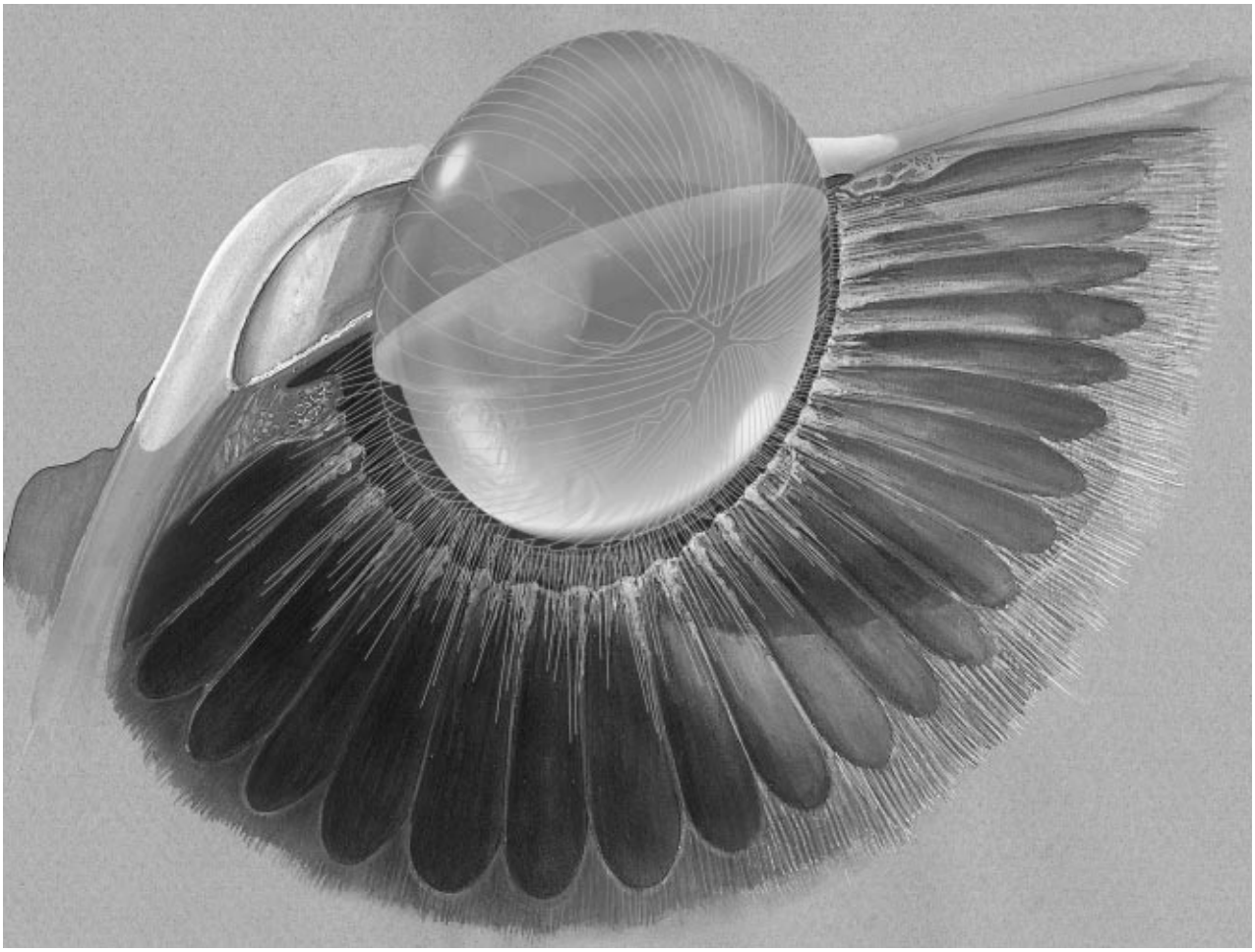
- ❑ alcohol
  - increased incidence of abortion and stillbirth, congenital anomalies, fetal alcohol syndrome (growth retardation, CNS involvement and facial anomalies)
- ❑ cigarettes
  - decreased birth weight, placenta previa/abruption, increased spontaneous abortion, preterm labour and stillbirth
- ❑ anticoagulants
  - warfarin crosses placenta, heparin does not
  - fetal warfarin syndrome: nasal hypoplasia, epiphyseal stippling, optic atrophy, MR, intracranial hemorrhage
  - also spontaneous abortion, stillbirth, prematurity, IUGR
- ❑ ACE inhibitors
- ❑ anticonvulsants
  - facial dysmorphogenesis, IUGR, mild MR, NTD's, congenital anomalies
- ❑ 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)

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
- ❑ contraindicated
  - rubella (see Antenatal Complications Section)

BREASTFEEDING AND DRUGS

- ❑ safe
  - penicillins, aminoglycosides, cephalosporins
  - oral contraceptive use (low dose) is now believed to be safe
- ❑ 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)



Drawing by M. Brierley