OB Obstetrics

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Basic Anatomy Review

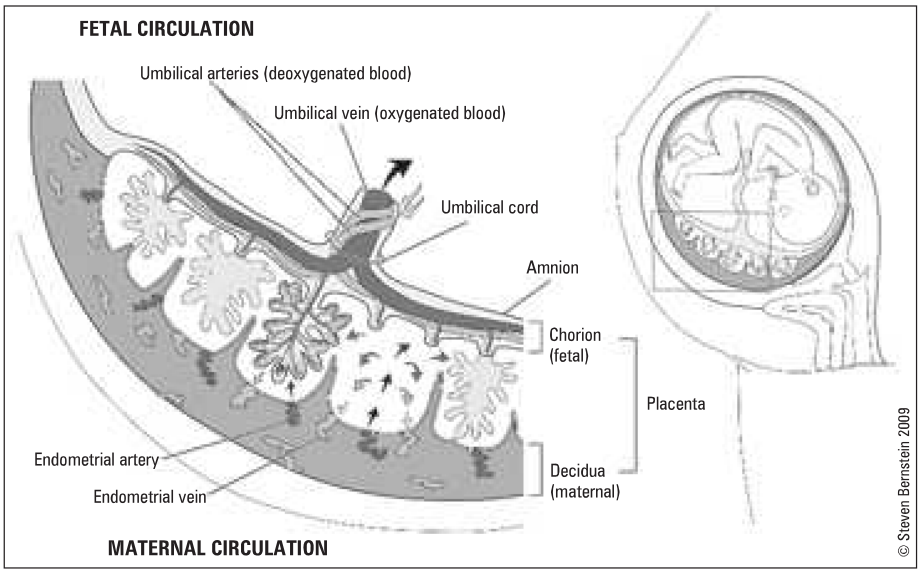

Figure 1. Placental Blood Flow

Umbilical Vessels
Always check the umbilical cord for 2 arteries and 1 vein: about 1/3 of babies with a single uterine artery will have another anomaly.

Placenta
- site of fetal nutritive, respiratory, and excretory function
- discoid mass composed of fetal (chorion frondosum) and maternal (decidua basalis) tissues divided by fissures into cotyledons (lobules) on the uterine side
- produces hormones such as progesterone, placental lactogen, estrogen, relaxin, beta-hCG and IGFs
- poor implantation can lead to spontaneous abortion
- abnormal location, implantation, or detachment can lead to antepartum hemorrhage (see *Antepartum Hemorrhage,* OB23)

Pregnancy

Diagnosis of Pregnancy

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

Trimesters
- T1 (first trimester): 0-12 wks
- T2 (second trimester): 12-28 wks
- T3 (third trimester): 28-40 wks
- Normal pregnancy term: 37-42 wks

Physical Signs of Pregnancy
CHUG
Chadwick's sign
Hegar's sign
Uterine enlargement
Goodell's sign

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

Investigations

- **beta-hCG:** peptide hormone composed of alpha and beta subunits produced by placental trophoblastic cells – maintains the corpus luteum during pregnancy
  - positive in serum 9 days post-conception, positive in urine 28 days after first day of last menstrual period (LMP)
  - plasma levels double every 1-2 days, peak at 8-10 weeks, then fall to a plateau until delivery
    - levels less than expected by dates suggest: ectopic pregnancy, abortion, or inaccurate dates
    - levels higher than expected suggest: multiple gestation, molar pregnancy, trisomy 21, or inaccurate dates
- **U/S**
  - transvaginal
    - 5 weeks: gestational sac visible (beta-hCG ≥1,200-1,500 mIU/mL)
    - 6 weeks: fetal pole seen
    - 7-8 weeks: fetal heart tones visible
  - transabdominal
    - 6-8 weeks: intrauterine pregnancy visible (beta-hCG ≥6,500 mIU/mL)

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

Maternal Physiology

Table 1. Physiologic Changes During Pregnancy

Skin	Increased pigmentation of perineum and areola, chloasma (pigmentation changes under eyes and on bridge of nose), linea nigra (midline abdominal pigmentation) Other: spider angiomas, palmar erythema due to increased estrogen, striae gravidarum due to connective tissue changes
Cardiovascular	Hyperdynamic circulation Increased CO, HR and blood volume Decreased BP due to decreased PVR Enlarging uterus compresses IVC and pelvic veins Decreased venous return leads to risk of hypotension Increased venous pressure leads to risk of varicose veins, hemorrhoids, leg edema
Hematologic	Hemodilution causes physiologic anemia and apparent decrease in hemoglobin and hematocrit Increased leukocyte count but impaired function leads to improvement in autoimmune diseases Gestational thrombocytopenia: mild (platelets >70,000/μL) and asymptomatic, normalizes within 2-12 weeks following delivery Hypercoagulable state: increased risk of DVT and PE but also decreased bleeding at delivery Influence of beta-hCG resets osmostat leading to non-pathological hyponatremia
Respiratory	Increased incidence of nasal congestion and epistaxis Increased O_2 consumption to meet increased metabolic requirements Elevated diaphragm i.e. patient appears more "barrel-chested" Increased minute ventilation leads to decreased CO_2 resulting in mild respiratory alkalosis that helps CO_2 diffuse across the placenta from fetal to maternal circulation No change in VC and FEV_1 Decreased TLC, FRC, and RV
Gastrointestinal	GERD due to increased intra-abdominal pressure and progesterone (causing decreased sphincter tone and delayed gastric emptying) Increased gallstones due to progesterone causing increased gallbladder stasis Constipation and hemorrhoids due to progesterone causing decreased GI motility Atypical appendicitis presentation due to upward displacement of appendix (e.g. RUQ pain)
Genitourinary	Increased urinary frequency due to increased total urinary output Increased incidence of UTI and pyelonephritis due to urinary stasis (see *Urinary Tract Infection,* OB18) Glycosuria that can be physiologic, must test for gestational diabetes mellitus (GDM) Ureters and renal pelvis dilation (R>L) due to progesterone-induced smooth muscle relaxation and uterine enlargement Increased CO and thus increased GFR leads to decreased creatinine (normal in pregnancy 35-44 mmol/L), uric acid, and BUN
Neurologic	Increased incidence of carpal tunnel syndrome and Bell's palsy
Endocrine	Thyroid: moderate enlargement and increased basal metabolic rate Increased total thyroxine and thyroxine binding globulin (TBG) Free thyroxine index and TSH levels are normal Adrenal: maternal cortisol rises throughout pregnancy (total and free) Calcium: decreased total maternal Ca due to decreased albumin Free ionized Ca (i.e. active) proportion remains the same due to parathyroid hormone (PTH), results in: increased bone resorption and gut absorption, increased bone turnover (but no loss of bone density due to estrogen inhibition)
Cervix	Goodell's sign, Chadwick's sign, Hegar's sign (see *Physical Signs of Pregnancy*, OB2)

Prenatal Care

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

Note: Guidelines vary by institution.

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

Preconception Counselling

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

- 3-8 weeks gestational age (GA) is a critical period of organogenesis, so early preparation is vital
- **past medical history:** optimize medical illnesses and necessary medications prior to pregnancy (see *Medical Conditions in Pregnancy*, OB11, and *Drugs in Pregnancy*, OB52)
- **supplementation**
  - folic acid: encourage diet rich in folic acid and supplement 8-12 wks preconception to prevent neural tube defects (NTDs)
    - 0.4-1 mg daily in all women, 5 mg if previous NTD, anti-epileptic medications, diabetes mellitus or BMI >35 kg/m^2) and continue for T1 of pregnancy
  - iron supplementation, prenatal vitamins
- **risk modification**
  - lifestyle: balanced nutrition and physical fitness
  - infection screening: rubella, HBsAg, VDRL, Pap smear, gonorrhea/chlamydia, HIV, toxoplasmosis, CMV, TB, varicella
  - genetic testing as appropriate for high risk groups (see *Prenatal Screening* section, Table 4, OB7); consider genetics referral in known carriers, recurrent pregnancy loss/stillbirth, family members with developmental delay or birth anomalies
  - social: alcohol, smoking, drug use, domestic violence
    - use Antenatal Psychosocial Health Assessment (ALPHA) form to screen for antenatal risk factors associated with poor postpartum family outcomes (woman abuse, child abuse, postpartum depression, marital dysfunction and increased physical illness)

Initial Prenatal Visit

- within 12 weeks of the first day of LMP or earlier if <20 or >35 years old
- fill out Antenatal Records

History

Ask every woman about abuse – not just those whose situations raise suspicion of abuse AND ask as early as possible in pregnancy.
Be careful not to congratulate women on pregnancy, as many are unplanned and may be unwanted.

- gestational age by dates from the first day of the LMP
- if LMP unreliable, get a dating ultrasound (see below)
- estimated date of confinement (EDC) using Naegle's Rule:
  - 1st day of LMP + 7 days – 3 months
  - e.g. LMP = 1 Apr 2011, EDC = 8 Jan 2012 (modify if cycle >28 days by adding number of days >28)
- history of present pregnancy (e.g. bleeding, nausea, vomiting)
- history of all previous pregnancies: GTPAL, year, sex, weight, gestational age, mode of delivery, length of labour, complications
- past medical history, past gynecological history
- prescription and non-prescription medications
- family history: genetic disease, birth defects, multiple gestation
- social history: smoking, alcohol, drug use, domestic violence (use ALPHA form)

Physical Examination

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

Investigations

Tests for HIV, prenatal and genetic screening are voluntary and require proper counseling and informed consent before proceeding.

- bloodwork
  - CBC, blood group and type, Rh antibodies, infection screening as per preconception counselling
- urine R&M, C&S
  - screen for bacteriuria and proteinuria
- pelvic exam
  - Pap smear (unless done within last 6-12 mo), cervical culture for *N. gonorrhoeae* (GC) and *C. trachomatis*, bacterial vaginosis (BV) vaginal swab

Counselling

- exercise
  - under physician guidance
  - absolute contraindications
    - ruptured membranes, preterm labour, hypertensive disorders of pregnancy, incompetent cervix, IUGR, multiple gestation (>3), placenta previa after 28th week, persistent 2nd or 3rd trimester bleeding, uncontrolled type I diabetes, thyroid disease, or other serious cardiovascular, respiratory or systemic disorder
  - relative contraindications
    - previous spontaneous abortion, previous preterm birth, mild/moderate cardiovascular or respiratory disorder, anemia (Hb ≤100 g/L), malnutrition or eating disorder, twin pregnancy after 28th week, other significant medical conditions
- nutrition
  - Canada's Food Guide to Healthy Eating suggests:
    - 3-4 servings of milk products daily (greater if multiple gestation)
    - a daily caloric increase of ~100 cal/d in the 1st trimester, ~300 cal/d in the second and third trimesters and ~450 cal/d during lactation
  - daily multivitamin should be continued in the 2nd trimester for women who do not consume an adequate diet; otherwise routine vitamin supplementation is not necessary (avoid excess vitamin A)
  - see *Drug and Food Safety in Pregnancy*, OB52
- nutrients important during pregnancy
  - folate: 0.4-5 mg per day
    - supports maternal increase in blood volume, growth of maternal and fetal tissue, decreases incidence of neural tube defects
    - foods rich in folic acid include: spinach, lentils, chick peas, asparagus, broccoli, peas, Brussels sprouts, corn and oranges
  - calcium: 1200-1500 mg per day
    - maintains integrity of maternal bones, skeletal development of fetus, breast milk production
  - vitamin D: 400 IU
    - promotes calcium absorption
  - iron: 0.8 mg/d in T1, 4-5 mg/d in T2 and >6 mg/d in T3
    - supports maternal increase in blood cell mass, supports fetal and placental tissue
    - required amounts exceed normal body stores and typical intake, and therefore need supplemental iron
    - iron is the only known nutrient for which requirements during pregnancy cannot be met by diet alone (see *Iron Deficiency Anemia,* OB11)
  - essential fatty acids (EFA) – supports fetal neural and visual development
    - contained in vegetable oils, margarines, peanuts, fatty fish
- weight gain: optimal gain depends on pre-pregnancy weight (varies from 6.8-18.2 kg)
- work: strenuous work, extended hours and shift work during pregnancy may be associated with greater risk of low birth weight, prematurity, and spontaneous abortion
- travel: not harmful, but stress related to travel may be associated with preterm labour
  - air travel is acceptable in second trimester but discouraged after 36 weeks
- sexual intercourse: may continue except in patients at risk for abortion, preterm labour, or placenta previa; breast stimulation may induce uterine activity and is discouraged in high-risk patients near term
- address social issues including physical or sexual abuse
- smoking: assist/encourage to reduce or quit smoking
- alcohol: encourage abstinence from alcohol during pregnancy
- genetic screening must be offered to all women (see *Prenatal Screening,* OB7 and *Chromosomal Screening,* OB9)

Risk Factors for Neural Tube Defects

GRIMM

- **G**enetics: family history of NTD (risk of having second child with NTD is increased to 2-5%), consanguinity, chromosomal (characteristic of trisomy 13, 18, and 21)
- **R**ace: European Caucasians > than African Americans, 3-fold higher in Hispanics
- **I**nsufficient vitamins: zinc and folate
- **M**aternal chronic disease (e.g. diabetes)
- **M**aternal use of anti-epileptic drugs

General population risk for NTD is 0.1%.

Expected Weight Gain

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

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

Subsequent Prenatal Visits

Timing

- for uncomplicated pregnancies, q4-6 weeks until 28 weeks, q2 weeks from 28 to 36 weeks and weekly from 36 weeks until delivery

Assess at Every Visit

- record estimated GA
- history of present pregnancy: fetal movements, uterine bleeding, leaking, cramping
- physical exam: BP, weight gain, symphysis fundal height (SFH), Leopold's maneuvers (T3) for lie, position and presentation of fetus
- investigations: urinalysis for glucosuria, ketones, proteinuria; fetal heart tones starting at 12 weeks using Doppler U/S

Symphysis Fundal Height (SFH)

12 weeks:	Uterine fundus at pubic symphysis
20 weeks:	Fundus at umbilicus SFH should be within 2 cm of GA between 20 and 36 weeks
37 weeks:	Fundus at sternum

Small for dates:
Date miscalculation
IUGR
Fetal Demise
Oligohydramnios

Large for dates:
Date miscalculation
Multiple gestation
Polyhydramnios

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

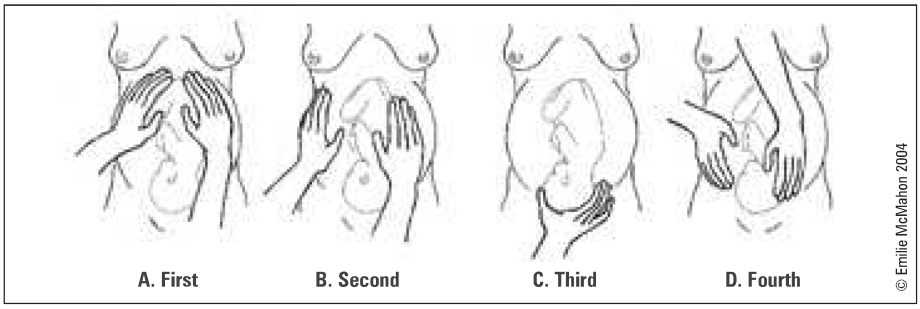

Figure 2. Leopold's Maneuvers (T3)
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Prenatal Fetal Monitoring

DDx of Decreased Fetal Movements

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

Fetal Movements
- generally first noticed ("quickening") at 18-20 wks in primigravidas; can occur 1-2 wks earlier in multigravidas
- if mother is concerned about decreased movement: mother chooses a time when fetus is normally active to count movements (usually recommended after 28 wks)
  - if <6 movements in 2 hours, try drinking juice, eating, changing position or moving to a quiet room and count for another 2 hours
  - if decreased movement persists, notify MD

NON-STRESS TEST (NST)

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

Definition
- fetal heart rate (FHR) tracing ≥20 minutes using an external Doppler to assess FHR and its relationship to fetal movement (see *Fetal Monitoring in Labour* section, OB33)

Indication
- any suggestion of uteroplacental insufficiency or suspected fetal distress

Table 2. Classification of Antepartum Non-Stress Test

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

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

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

Interpretation
- normal (reassuring NST): at least 2 accelerations of FHR >15 bpm from the baseline lasting >15 seconds, in 20 minutes
- abnormal (non-reassuring NST): <2 accelerations of FHR 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 abnormal, then perform biophysical profile (BPP)

BIOPHYSICAL PROFILE (BPP)

Definition
- U/S assessment of the fetus ± NST

Indications
- non-reassuring NST
- post-term pregnancy
- decreased fetal movement
- any other suggestion of fetal distress or uteroplacental insufficiency

Reassuring BPP (8/8)

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

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

Interpretation
- 8: 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 benefits of delivery outweigh risks

Table 3. Scoring of the Biophysical Profile

Parameter	Reassuring (2 points)	Non-Reassuring (0 points)
AFV*	Fluid pocket of 2 cm in 2 axes	Oligohydramnios
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

*Amniotic fluid volume (AFV) is a marker of chronic hypoxia, all other parameters indicate acute hypoxia

Prenatal Screening

- testing should only occur following counselling and with the informed consent of the patient

Table 4. High-Risk Population Screening Tests

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

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

Table 5. Gestation-Dependent Screening Investigations

Gestational Age (weeks)	Investigations
8-12	Dating U/S, initial Pap smear, chlamydia/gonorrhea cultures
10-12	Chorionic villus sampling (CVS)
11-14	First trimester screening (FTS) Integrated prenatal screening (IPS) Part 1
11-14	Nuchal translucency U/S
15-16 to term	Amniocentesis
15-18	Integrated prenatal screening (IPS) Part 2
16-18	Maternal serum screen (or MSAFP only for patients who did FTS earlier)
18-20 to term	Fetal movements (quickening)
18-20	U/S for dates, fetal growth and anatomy assessment
24-28	50 g oral glucose challenge test (OGCT)
28	Repeat CBC RhIG for all Rh negative women
36	Rh antibody screen if indicated Group B *Streptococcus* (GBS) Screen
6 weeks postpartum	Discuss contraception, menses, breastfeeding, depression, mental health, support Physical exam: breast exam, pelvic exam including Pap smear

Routine T2 U/S at 18-22 weeks, helps determine:
- Number of fetuses
- GA (if no prior U/S)
- Location of placenta
- Fetal anomalies

ULTRASOUND SCREENING

- dating ultrasound best done between 8-12 weeks GA
  - measurement of crown-rump length (margin of error ± 3 days)
  - change EDC to U/S date if >1 week discrepancy from EDC based on LMP
- nuchal translucency ultrasound (NTUS) at 11-14 weeks GA
  - measures the amount of fluid behind the neck of the fetus
  - early screen for serious congenital anomalies (Down syndrome)
  - NT measurement is necessary for the FTS and IPS Part 1
- fetal growth and anatomy ultrasound routinely done at 18-20 weeks GA (margin of error ± 7 days) (see Pediatrics, P43 for congenital anomalies)
- earlier or subsequent ultrasounds performed when medically indicated

Table 6. Comparison of FTS, MSS and IPS

First Trimester Screen (FTS)	Maternal Serum Screen (MSS)	Integrated Prenatal Screen (IPS)
11-14 wks	15-18 wks	Nuchal translucency on 12 wk U/S FTS at 11-14 wks MSS + inhibin A at 15-18 wks
Measures 1. Nuchal translucency on U/S 2. Beta-hCG 3. Pregnancy-associated plasma protein A (PAPP-A)	Measures 1. Maternal serum alpha-fetoprotein (MSAFP) 2. Beta-hCG 3. Unconjugated estrogen (estriol or uE3)	
Risk estimate for 1. Down syndrome (Trisomy 21): increased NT, increased beta-hCG, decreased PAPP-A	Risk estimate for 1. Open neural tube defect (oNTD): increased MSAFP (sensitivity 80-90%) 2. Trisomy 21: decreased MSAFP, increased beta-hCG, decreased uE3 (sensitivity 65%) 3. Trisomy 18: decreased MSAFP, decreased beta-hCG, decreased uE3 (sensitivity 80%)	Risk estimate for oNTD, Trisomy 21, Trisomy 18 Sensitivity ~85-90% 2% false positive rate Patients with positive screen should be offered U/S and/or amniocentesis
Note: does not measure risk of oNTD and should be combined with MSAFP at 16 weeks Useful where patient wants results within the first trimester More accurate estimate of Down syndrome risk than MSS, sensitivity ~85% (when combined with age) 5% false positive rate Patients with positive screen should be offered CVS or amniocentesis	Only offered alone if patient missed the time window for IPS or FTS 8% baseline false positive rate for t21, lower for oNTD and t18 Patients with positive screen should be offered U/S or amniocentesis	

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

CHROMOSOMAL SCREENING

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

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

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

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

Indications
- identification of genetic anomalies (15-16 weeks gestation) as per indications above
- assessment of fetal lung maturity (T3) via the L/S ratio (lecithin:sphingomyelin)
  - if >2:1, respiratory distress syndrome (RDS) is less likely to occur
- assessment of amniotic fluid bilirubin concentration in Rh-isoimmunized pregnancies

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

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

Disadvantages
- 0.5% risk of spontaneous abortion and risk of fetal limb injury
- results take 14-28 days

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

CHORIONIC VILLUS SAMPLING (CVS)
- biopsy of fetal-derived chorion using a trans-abdominal needle or trans-cervical catheter at 10-12 weeks

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

Disadvantages
- 1-2% risk of spontaneous abortion and risk of fetal limb injury
- does not screen for open neural tube defects
- 1-2% incidence of genetic mosaicism → false negative results

ISOIMMUNIZATION SCREENING

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

Etiology
- maternal-fetal circulation normally separated by placental barrier, but sensitization can occur and can affect the current pregnancy, or more commonly, future pregnancies
- in pregnancy, anti-Rh Ab produced by a sensitized Rh-negative mother can lead to fetal hemolytic anemia
- overall risk of isoimmunization of an Rh-negative mother with an Rh-positive ABO-compatible infant is 16% (2% antepartum, 7% within 6 months of delivery, and 7% in the second pregnancy)
- sensitization routes
  - incompatible blood transfusions
  - previous fetal-maternal transplacental hemorrhage (e.g. ectopic pregnancy)
  - invasive procedures in pregnancy (e.g. prenatal diagnosis, cerclage, D&C)
  - any type of abortion
  - labour and delivery

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

Investigations
- routine screening at first visit for blood group, Rh status, and antibodies are measured by the indirect Coombs test
- if Rh positive with antibodies present, the severity of fetal anemia is determined primarily by antibody concentration
  - Ab titres <1:16 considered benign
  - Ab titres ≥1:16 necessitates amniocentesis to determine severity of fetal anemia (which correlates with the amount of biliary pigment in amniotic fluid from 27 wks onward)
- Kleihauer-Betke test used to determine extent of fetomaternal hemorrhage
  - fetal red blood cells identified on a slide treated with citrate phosphate buffer because adult hemoglobin elutes through cell membrane in presence of acid more readily
- detailed U/S for hydrops fetalis

Prophylaxis
- exogenous Rh IgG (Rhogam® or WinRho®) binds to Rh antigens of fetal cells and prevents them from contacting maternal immune system
- Rhogam® (300 μg) given to all Rh negative women in the following scenarios:
  - routinely at 28 weeks GA (provides protection for ~12 wks)
  - within 72 hours of the birth of an Rh positive fetus
  - with a positive Kleihauer-Betke test
  - with any invasive procedure in pregnancy (CVS, amniocentesis)
  - in ectopic pregnancy
  - with miscarriage or therapeutic abortion (only 50 μg required)
  - with an antepartum hemorrhage
- if Rh negative and Ab screen positive, follow mother with serial monthly Ab titres throughout pregnancy ± serial amniocentesis as needed (Rhogam® has no benefit)

Investigations
- bilirubin is measured by serial amniocentesis to assess the severity of hemolysis
- cordocentesis for fetal Hb: should be used cautiously (not first line)

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

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

GROUP B STREPTOCOCCUS (GBS) SCREEN

Screening vs. Risk-based Approach for GBS Prevention in Newborns
NEJM 2002; 347:233-9
Study: Large retrospective cohort study comparing the effectiveness of screening and risk-based approaches in preventing early-onset GBS disease (within 7 days of birth).
Patients: Stratified random sample of 629,912 live births in areas where there was active surveillance for GBS infection, the records for 5144 live births (screened group: n=2628; risk-based group: n=2515) were randomly selected to be reviewed, including all births where newborns had early-onset disease (n=312).
Intervention: Screening approach (routine screening with cultures for GBS between 35-37 wks GA and offering intrapartum antiobiotic prophylaxis to carriers) vs. risk-based approach (offering intrapartum antiobiotic prophylaxis to women presenting at time of labour with clinical risk factors for GBS transmission – fever, prolonged ROM, preterm delivery, etc.).
Main outcome: Early-onset GBS disease
Results: Infants of women in the screened group had a significantly lower risk of early-onset disease compared to those in the risk-based group (RR=0.46; 95% CI=0.36 to 0.60). The greatest risk factors for early-onset disease were (a) intrapartum fever (RR=5.99; 95% CI=4.28-8.38) and (b) history of a previous child with GBS disease (RR=3.79, 95% CI=1.30-11.11).
Conclusion: Routine screening for GBS during pregnancy is more effective for preventing GBS disease in newborns than the risk-based approach.

Epidemiology
- 15-40% vaginal carrier rate

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

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

Investigations
- offer screening to all women at 35-37 weeks with vaginal and anorectal swabs for C&S

Treatment
- treatment of maternal GBS at delivery decreases neonatal morbidity and mortality
- indications for antibiotic prophylaxis: positive GBS screen or GBS status unknown and one of the risk factors (see above)
- antibiotics for GBS prophylaxis
  - penicillin G 5 million units IV then 2.5 million units IV q4h until delivery
  - penicillin allergic but not at risk for anaphylaxis – cefazolin 2 g IV then 1 g q8h
  - penicillin allergic and at risk for anaphylaxis – clindamycin 900 mg IV q8h or erythromycin 500 mg IV q6h
- if fever, broad spectrum antibiotic coverage is advised

Termination of Pregnancy

Definition
- active termination of a pregnancy before fetal viability (usually <500 g or 20 weeks GA)

Indications
- inability to carry a pregnancy to term due to medical or social reasons (including patient preference)

Management
- **medical**
  - <9 weeks: methotrexate + misoprostol
  - >12 weeks: prostaglandins (intra- or extra-amniotically or IM) or misoprostol
- **surgical**
  - <12-16 weeks: dilatation + vacuum aspiration ± curettage
  - >16 weeks: dilatation and evacuation, early induction of labour
  - common complications: pain or discomfort
  - less common complications: hemorrhage, perforation of uterus, laceration of cervix, risk of infertility, infection/endometritis, Asherman's syndrome (adhesions within the endometrial cavity causing amenorrhea/infertility), retained products of conception
- **counselling**
  - supportive services
  - future contraception plans
  - ensure follow-up

CMA policy (1988)
"Induced abortion should be uniformly available to all women in Canada" and "there should be no delay in the provision of abortion services".

Terminations are generally done until the stage of viability (~23.5 weeks), although this varies depending on the provider.

Induced Abortion Statistics
- Rate per 1,000 women (all ages): 13.7
- Rate per 1,000 women (age 20-24): 27.7
- Ratio of induced abortions per 100 live births (all ages): 28.3
- Ratio of induced abortions per 100 live births (age 20-24): 54.9
- 31.4% of all abortion services are accessed by women aged 20-24

Adapted from Statistics Canada, 2005, *Induced Abortion Statistics*, 82-223-XWE, page 16 of 32.

Medical Conditions in Pregnancy

Iron and Folate Deficiency Anemia

Table 7. Iron Deficiency and Folate Deficiency Anemia

	Iron Deficiency Anemia	Folate Deficiency Anemia
Etiology	Nutritional: inadequate intake Decreased iron absorption (malabsorption syndrome, antacid use) Increased iron losses (vaginal bleeding, other source of bleeding) Increased iron requirement (fetal growth, multiple gestation)	Nutritional: decreased intake Non-nutritional factors: multiple gestation, drugs (phenytoin, methotrexate), chronic hemolytic anemia, malabsorption entities (celiac sprue) Takes approximately 18 weeks of a folate-deficient diet to produce anemia
Epidemiology	Responsible for 80% of causes of non-physiologic anemia during pregnancy	Incidence varies from 0.5-25% depending on region, population, diet
Clinical Features	Same as in non-pregnant states Non-specific symptoms: pallor, fatigue, palpitations, tachycardia, dyspnea Severe anemia: angular stomatitis, glossitis	Non-specific symptoms: anorexia, nausea, vomiting, diarrhea, depression, pallor, UTI, sore mouth or tongue
Investigations	Serum iron, serum ferritin, blood smear – do not include total iron binding capacity (TIBC) since it is increased during normal pregnancy	RBC, serum folate, blood smear
Management	Prevention: 150 mg ferrous sulfate OD, 300 mg ferrous gluconate OD or 30 mg of ferrous iron OD for all pregnant women in T2 and T3 If anemic: 1 g ferrous sulfate OD (180 mg elemental Fe)	Prevention: 0.4-1 mg folic acid PO daily for 1-3 months preconceptually and throughout T1, or 5 mg folic acid per day with past history of oNTD, diabetes or anti-epileptic medication use
Complications	Maternal: angina, CHF, infection, slower recuperation, preterm labour Fetal: decreased oxygen carrying capacity leading to fetal distress, IUGR, low birth weight and hydrops	Maternal: decreased blood volume, nausea, vomiting, anorexia Fetal: neural tube defects in T1, low birth weight, prematurity
Notes	Mother needs 1 g of elemental iron per fetus; this amount exceeds normal stores + dietary intake Iron requirements increase during pregnancy due to fetal/placental growth (500 mg), increased maternal RBC mass (500 mg) and losses (200 mg) – more needed for multiple gestations	Minimum daily requirement is 0.4 mg Most often associated with iron deficiency anemia Folic acid is necessary for closure of neural tube during early fetal development (by day 28 of gestation)

Diabetes Mellitus (DM)

Classification of Diabetes Mellitus
- Type 1 and Type 2 DM (see Endocrinology, E6)
- gestational diabetes mellitus (GDM): onset of diabetes mellitus during pregnancy

Etiology
- Type 1 and Type 2 DM
- GDM: usually around 24-28 weeks GA, anti-insulin factors produced by placenta and high maternal cortisol levels create increased peripheral insulin resistance → higher fasting glucose → leading to GDM and/or exacerbating pre-existing DM

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

MANAGEMENT

A. TYPE I AND TYPE 2 DM

Preconception
- pre-plan and refer to high-risk clinic
- optimize glycemic control
- counsel patient re: potential risks and complications
- evaluate for diabetic retinopathy, neuropathy, coronary artery disease

Pregnancy
- if already on medication, generally switch to insulin therapy
  - continuing glyburide or metformin controversial
  - teratogenicity unknown for other oral anti-hyperglycemics
- tight glycemic control
  - diet management first line therapy
  - post-prandial blood glucose values seem to be the most effective at determining the likelihood of macrosomia or other adverse pregnancy outcomes
  - aim for Fasting Plasma Glucose (PG) ≤5.3 mmol/L (95 mg/dL), 1-hour post prandial PG ≤7.8 mmol/L (140 mg/dL), 2-hour post prandial PG ≤6.7 mmol/L (120 mg/dL)
  - if blood glucose not well controlled, initiate insulin therapy
  - insulin dosage may need to be adjusted in T2 due to increased demand and increased insulin resistance
- monitor as for normal pregnancy plus initial 24-hr urine protein and creatinine clearance, retinal exam, HbA_{1C}
  - HbA_{1C}: >140% of pre-pregnancy value associated with increased risk of spontaneous abortion and congenital malformations
- increased fetal surveillance (BPP, NST)

Labour
- timing of delivery depends on fetal and maternal health and risk factors (i.e. must consider size of baby, lung maturity, maternal blood glucose and blood pressure control)
- can wait for spontaneous labour if blood glucose well-controlled and BPP normal
- induce by 40 weeks
- type of delivery
  - increased risk of cephalopelvic disproportion (CPD) and shoulder dystocia with babies >4,000 g (8.8 lbs)
  - elective C/S for predicted birthweight >4,500 g (9.9 lbs) (controversial)
- monitoring
  - during labour monitor blood glucose q1h with patient on insulin and dextrose drip
  - aim for blood glucose between 3.5 to 6.5 mmol/L to reduce the risk of neonatal hypoglycemia

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

B. GESTATIONAL DIABETES MELLITUS

Risk factors for GDM:
Age >25
Obesity
Ethnicity (Aboriginal, Hispanic, Asian, African)
FHx of DM
Previous history of GDM
Previous child with birthweight >4.0 kg

Screening + Diagnosis
- at 24-28 weeks GA
- pregnant females age >25 or age <25 years with >1 risk factor (see sidebar)
- 1-hour, 50 g Oral Glucose Challenge Test (OGCT): not fasting
  - PG <7.8 mmol/L (140 mg/dL) = no GDM
  - PG ≥7.8-10.3 mmol/L = further investigation with OGTT
  - PG ≥10.3 mmol/L (185 mg/dL) = GDM
- 2-hour, 75 g Oral Glucose Tolerance Test (OGTT): fasting
  - FPG ≥5.3 mmol/L (95 mg/dL)
  - PG 1-hour ≥10.6 mmol/L (190 mg/dL)
  - PG 2-hour ≥8.9 mmol/L (160 mg/dL)
    - 2/3 of the above = GDM
    - 1/3 of the above = impaired glucose tolerance (IGT)

Management
- treat both GDM and IGT
- tight glycemic control optimal as in Type 1 and Type 2 DM
- monitoring and timing of delivery as for Type 1 and Type 2 DM
- stop insulin and diabetic diet postpartum
- follow-up with 2-hour, 75 g OGTT 6 weeks-6 months postpartum

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

Long Term Maternal Complications
- Type 1 and Type 2 DM: risk of progressive retinopathy and nephropathy
- GDM: 50% risk of developing Type 2 DM in next 20 years

Table 8. Complications of DM in Pregnancy

Maternal	Fetal
Obstetric • Hypertension/preeclampsia (especially if pre-existing nephropathy/proteinuria): insulin resistance is implicated in etiology of hypertension • Polyhydramnios: maternal hyperglycemia leads to fetal hyperglycemia, which leads to fetal polyuria (a major source of amniotic fluid)	**Growth Abnormalities** • Macrosomia: maternal hyperglycemia leads to fetal hyperinsulinism resulting in accelerated anabolism • Intrauterine growth restriction (IUGR): due to placental vascular insufficiency
Diabetic Emergencies • Hypoglycemia • Ketoacidosis • Diabetic coma	**Delayed Organ Maturity** • Fetal lung immaturity: hyperglycemia interferes with surfactant synthesis (respiratory distress syndrome)
End-organ involvement or deterioration (occur in DM1 and DM2, not in GDM) • Retinopathy • Nephropathy	**Congenital Anomalies (occur in DM1 and DM2, not in GDM)** • 2-7x increased risk of cardiac (VSD), NTD, GU (cystic kidneys), GI (anal atresia), and MSK (sacral agenesis) anomalies due to hyperglycemia *Note:* Pregnancies complicated by GDM do not manifest an increased risk of congenital anomalies because GDM develops after the critical period of organogenesis (in T1)
Other • Pyelonephritis/UTI: glucosuria provides a culture medium for *E. coli* and other bacteria • Increased incidence of spontaneous abortion (in DM1 and DM2, not in GDM): related to pre-conception glycemic control	**Labour and Delivery** • Preterm labour/prematurity: most commonly in patients with hypertension/preeclampsia. Preterm labour is associated with poor glycemic control but the exact mechanism is unknown • Increased incidence of stillbirth • Birth trauma: due to macrosomia, can lead to difficult vaginal delivery and shoulder dystocia **Neonatal** • Hypoglycemia: due to pancreatic hyperplasia and excess insulin secretion in the neonate • Hyperbilirubinemia and jaundice: due to prematurity and polycythemia • Hypocalcemia: exact pathophysiology not understood, may be related to functional hypoparathyroidism • Polycythemia: hyperglycemia stimulates fetal erythropoietin production

Hypertension in Pregnancy

Hypertension in Pregnancy

Adverse Maternal Conditions
- DBP >100 mmHg
- HELLP
- Cerebral haemorrhage
- Renal dysfunction – oliguria <500 ml/d
- Left ventricular failure, pulmonary edema
- Abruption, DIC
- **Symptoms:**
  - Abdominal pain, nausea, vomiting
  - Headaches, visual problems
  - SOB, chest pain
  - Eclampsia – convulsions

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

Preeclampsia Investigations

CBC	LDH
Liver enzymes	Albumin
INR and aPTT	Bilirubin
Cr	Urine (dip ± 24 hour collection)
Uric Acid	

- hypertensive disorders are classified as:
  1. pre-existing (<20 wks GA) – 90% cases are essential, 10% are secondary
  2. gestational (≥20 wks GA)
- preeclampsia can occur with either pre-existing or gestational hypertension

1. PRE-EXISTING HYPERTENSION

A) WITHOUT PREECLAMPSIA

Definition
- HTN (>140/90) prior to 20 weeks GA [except in a gestational trophoblastic neoplasia (GTN)], persisting postpartum
- essential hypertension is associated with an increased risk of gestational HTN, abruptio placenta, IUGR and intrauterine fetal demise (IUFD)

Management
- alpha-methyldopa 250-500 mg PO tid/qid or labetalol 100-300 mg PO bid/tid
- no ACE inhibitors, diuretics or propranolol (teratogens)
- monitor progress with serial U/S

B) WITH PREECLAMPSIA

Definition
- pre-existing hypertension with new onset proteinuria or adverse conditions or resistant hypertension
- 2-7 fold increased likelihood of developing preeclampsia/eclampsia if pre-existing maternal hypertension
- occurs early, tends to be severe (often with IUGR) and to recur with subsequent pregnancies

Management
- stabilize and deliver: only "cure" is delivery of placenta, independent of gestational age (vaginal delivery preferred)
- increased maternal monitoring: hourly input and output, urine dip q12h and hourly neurological vitals
- increased fetal evaluation: continuous FHR monitoring
- anticonvulsant therapy
  - raises seizure threshold
  - Mg sulfate 4 g IV bolus over 20 min followed by maintenance of 2-4 g/hour
  - monitor for signs of Mg toxicity: depressed deep tendon reflexes, decreased RR, anuric, hypotonic, CNS or cardiac depression
  - antagonist to Mg sulfate: calcium gluconate (10%) 10 mL (1 g) IV over 2 minutes
- antihypertensive therapy
  - lowering BP decreases the risk of stroke
  - hydralazine 5-10 mg IV bolus over 5 minutes q15-30 minutes as necessary
  - labetalol 20-50 mg IV q10 minutes
  - 2nd line: nifedipine 10-20 mg PO q20-60 minutes
  - ACE-inhibitors are contraindicated
- postpartum management
  - risk of seizure highest in first 24 hours postpartum – continue Mg sulfate for 12-24 hours after delivery
  - vitals q1h
  - consider HELLP syndrome in toxic patients
  - most return to a normotensive BP within 2 weeks

2. GESTATIONAL HYPERTENSION

Etiology
- imbalance of thromboxane (vasoconstrictor) and prostaglandin (vasodilator), arteriolar constriction, capillary damage, protein extravasation, and hemorrhage
- occurs after 20 weeks GA, except in patients with trophoblastic diseases (hydatidiform mole, hydrops, choriocarcinoma) when it occurs before 20 weeks GA

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

Clinical Evaluation of Gestational Hypertension
- in general, clinical evaluation should include the mother and fetus
- evaluation of mother:
  - RUQ pain, headache and visual disturbances are potentially ominous symptoms requiring immediate assessment
  - central nervous system
    - presence and severity of headache
    - visual disturbances – blurring, scotomata
    - tremulousness, irritability, somnolence
    - hyperreflexia
  - hematologic
    - bleeding, petechiae
  - hepatic
    - RUQ or epigastric pain
    - severe nausea and vomiting
  - renal
    - urine output and colour
  - non-dependent edema (i.e. hands and face)
- evaluation of fetus:
  - fetal movement
  - fetal heart rate tracing – NST
  - ultrasound for growth
  - biophysical profile
  - Doppler flow studies

Laboratory Evaluation of Gestational Hypertension
- hemoglobin, platelets, blood film
- PTT, INR, fibrinogen, D-dimer – especially if surgery or regional anesthetics are planned
- ALT, AST, LDH, bilirubin
- proteinuria, creatinine, uric acid
- 24-hour urine collection for total protein and creatinine clearance

Complications
- maternal
  - hemorrhagic stroke (50% of deaths)
  - left ventricular failure/pulmonary edema
  - liver and renal dysfunction
  - abruptio placentae
  - seizure
  - DIC (release of placental thromboplastin → consumptive coagulopathy)
  - HELLP syndrome (see Table 9)
    - treat with FFP infusion or plasma exchange
- fetal (2° to placental insufficiency)
  - IUGR, prematurity, abruptio placentae, IUFD

A) WITHOUT PREECLAMPSIA
Management
- bedrest in left lateral decubitus position, normal salt and protein intake
- avoid diuretics and antihypertensives
- monitor for progression
- if ≥37 weeks GA, consider induction of labour (see *Induction of Labour,* OB35)

B) WITH PREECLAMPSIA
Definition
- gestational hypertension with new onset proteinuria or one or more adverse condition(s)

Management
- see *Pre-existing Hypertension with Preeclampsia*, OB14

3. SEVERE PREECLAMPSIA
- definition: preeclampsia before 34 weeks GA with:
  - heavy proteinuria (3-5 g/day) or
  - one or more adverse condition(s)

Management of Gestational Hypertension with Seizures
- ABC's
- seizure prevention and control

Hyperemesis Gravidarum

Definition
- intractable nausea and vomiting, usually presents in T1 then diminishes; occasionally persists throughout pregnancy
- affects ~1% of pregnancies

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

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

Management
- if severe: admit to hospital, NPO initially then small frequent meals of appealing foods, correct hypovolemia, electrolyte disturbance and ketosis
- thiamine supplementation may be indicated
- TPN (if very severe) to reverse catabolic state
- non-pharmacological
  - rest
  - avoid triggers (e.g. certain odours)
  - acupressure at inner aspect of the wrists
  - ginger is effective but teratogenic effect unknown
- pharmacological options
  - Diclectin® (10 mg doxylamine succinate with vitamin B_6) can be started at 2 tablets qhs + 1 tablet qAM + 1 tablet qPM (i.e. afternoon); dosage can be increased up to 8 tablets per day
  - Gravol® can be safely used as an adjunct to Diclectin® (1 suppository bid or 25 mg PO qid)

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

Jaundice in Pregnancy

Epidemiology
- affects 1 in 1500 pregnancies

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

Etiology
- viral hepatitis (most common)
- unique to pregnancy (see Table 9)
  - cholestatic jaundice of pregnancy
  - HELLP syndrome
  - hepatic rupture, hematoma and infarct
  - acute fatty liver of pregnancy (AFLP)
  - hyperemesis gravidarum (rarely causes hepatic dysfunction)
- pre-existing conditions, see Gastroenterology, *Liver/Biliary Tract*, G31, G44

Table 9. Conditions Causing Jaundice in Pregnancy

	HELLP Syndrome	Cholestatic Jaundice of Pregnancy	Hepatic Infact, Hematoma, and Rupture	Acute Fatty Liver of Pregnancy (AFLP)
Definition	**H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelets Pathogenesis unknown	Clinical syndrome characterized by intense pruritus that precedes jaundice by 7-14 days Pathogenesis unknown, may be due to increased sensitivity to high levels of estrogen or abnormal progestational steroids	Rare consequence of preeclampsia, typically occuring in T3 Vasospasm-induced hepatic infarction can lead to hematoma formation; hematoma can lead to rupture	Form of hepatic failure with coagulopathy and encephalopathy characterized by microvesicular fatty infiltrates in liver parenchyma Pathogenesis unknown
Epidemiology	Affects 20% of women with severe preeclampsia Presents >27 weeks GA (11% sooner); up to 30% of cases present AFTER delivery and with no prevoius signs of hypertension	17-29 weeks GA High incidence in Chile and Scandinavia; rare in Asian and African populations		1 in 7000 deliveries 3rd trimester (28-40 weeks GA) Maternal mortality as high as 75%; resolution of hepatic dysfunction with delivery or termination of pregnancy
Clinical Features	Epigastric, RUQ or chest pain, N/V, symptoms of preeclampsia (headache, blurred vision, thirst) ± jaundice Atypical presentations: asymptomatic reduction in platelet count, "flu-like" symptoms	Intense pruritis (usually, worst on palms and soles of feet) ± icterus (1-2 weeks later) Steatorrhea unusual	Hepatic rupture: RUQ abdominal pain, abdominal distention, nausea/vomiting, and hypertension, followed by shock	Acute nausea/vomiting, severe upper abdominal pain preceding jaundice Confusion Preeclampsia Pruritus Range in presentation: • Mild • Fulminant: GI bleeding, hepatic coma, renal failure and true hepatic failure (coagulopathy and encephalopathy)
Investigations	AST (70-663 U/L), total bilirubin slightly increased, low platelet count (7-99), elevated LDH ± elevated D-dimers, tissue polypeptide antigen (TPA) and fibronectin, fragmented RBCs on smear Liver biopsy (rarely done): periportal hemorrhage and fibrin deposition with periportal necrosis; macro- and microvesicular fatty deposits (NOT pericentral as in AFLP)	ALT <500 IU, ALP and GGT markedly elevated (to levels consistent with moderate to severe cholestasis)	Hemoperitoneum (paracentesis, U/S, CT, MRI showing ruptured liver)	Elevated PTT and low serum fibrinogen AST>ALT Hypoglycemia Preeclampsia and HELLP features Liver biopsy to establish diagnosis: • Microvesicular fatty infiltrates of the central zone hepatocytes • Oil Red O stain on frozen tissue • Electron microscopy on glutaraldehyde fixed tissue If liver biopsy not possible, CT most useful
Management	Supportive care (in ICU) and prompt delivery	Ursodeoxycholic acid (20-25 mg/kg/day) Pruritus: cholestyramine Prophylactic vitamin K before delivery Consider induction of labour (see *Induction of Labour,* OB35)	Aggressive: rapid delivery and trauma surgery to repair liver	Early diagnosis with prompt delivery followed by maximal supportive care • ABCs, mechanical ventilation, transfusion of blood products • Hepatic encephalopathy treatment – lactulose, catharsis • Treat hypoglycemia
Notes	Complications: sepsis, multi-system organ failure, hepatic failure, DIC, death (rare)	Selenium may be protective against cholestasis Strong familial predisposition Correlates with oral contraceptive sensitivity	Complications include death (mother and fetus) if untreated	Recovery begins with delivery Persistent or increasing hyperbilirubinemia and complications: • Should not be interpreted as indications for liver transplantation • Aggressive supportive measures

Treat asymptomatic bacteriuria in pregnancy because of increased risk of progression to cystitis, pyelonephritis and probable increased risk of PRETERM LABOUR.

Urinary Tract Infection (UTI)

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

Epidemiology
- most common medical complication of pregnancy
- asymptomatic bacteriuria in 2-7% of pregnant women depending on parity and socioeconomic factors
- *note:* asymptomatic bacteriuria should be treated in pregnancy

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

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

Management
- uncomplicated UTI
  - first line: amoxicillin (250-500 mg PO q8h x 7 days)
  - alternatives: TMP-SMX (Septra®) or nitrofurantoin (avoid sulpha drugs during last 6 weeks of pregnancy due to displacement of bilirubin from albumin and increased kernicterus in the newborn)
  - follow with monthly urine cultures
- pyelonephritis
  - hospitalization and IV antibiotics

Prognosis
- complications if untreated: acute cystitis, pyelonephritis, and possible preterm premature rupture of membranes (PPROM)
- recurrence is common

Table 10. Infections During Pregnancy

Infection	Agent	Source of Transmission	Greatest Transmission Risk to Fetus	Effects on Fetus	Effects on Mother	Diagnosis	Management
Chicken Pox	Varicella zoster virus (herpes family)	To mom: direct, respiratory, To baby: transplacental	13-30 weeks GA, and 5d pre- to 2d post-delivery	Congenital varicella syndrome (limb aplasia, chorioretinitis, cataracts, cutaneous scars, cortical atrophy, IUGR, hydrops), preterm labour (prematurity)	Fever, malaise, vesicular pruritic lesions	Clinical, ± vesicle fluid culture, ± serology	VZIG for mother if exposed, decreases congenital varicella syndrome Note: Do not administer vaccine during pregnancy (live attenuated)
***CMV**	DNA virus (herpes family)	To mom: blood/organ transfusion, sexual contact To baby: transplacental, during delivery, breast milk	T1-T3	5-10% develop CNS involvement (mental retardation, cerebral calcification, hydrocephalus, microcephaly, deafness, chorioretinitis)	Asymptomatic or flu-like	Serologic screen; isolate virus from urine or secretion culture	No specific treatment; maintain good hygiene and avoid high risk situations
Erythema Infectiosum (Fifth Disease)	Parvovirus B19	To mom: respiratory, infected blood products To baby: transplacental	10-20 weeks GA	Spontaneous abortion (SA), stillbirth, hydrops in utero	Flu-like, rash, arthritis; often asymptomatic	Serology, viral PCR, maternal AFP; if IgM present, follow fetus with U/S for hydrops	If hydrops occurs, consider fetal transfusion
Hepatitis B	DNA virus	To mom: blood, saliva, semen, vaginal secretions To baby: transplacental, breast milk	T3 10% vertical transmission if asymptomatic HBsAg +ve; 85-90% if HBsAg and HBeAg +ve	Prematurity, low birth weight, neonatal death	Fever, N/V, fatigue, jaundice, elevated liver enzymes	Serologic screening for all pregnancies	Rx neonate with HBIG and vaccine (at birth, 1, 6 mos); 90% effective
***Herpes Simplex Virus**	DNA virus	To mom: intimate mucocutaneous contact, To baby: transplacental, during delivery	Delivery (if genital lesions present); less commonly in utero	Disseminated herpes (20%); CNS sequelae (35%); self-limited infection	Painful vesicular lesions	Clinical diagnosis	Acyclovir for symptomatic women, suppressive therapy at 36 wks controversial C/S if active genital lesions, even if remote from vulva
HIV	RNA retrovirus	To mom: blood, semen, vaginal secretions To baby: in utero, during delivery, breast milk	1/3 in utero, 1/3 at delivery, 1/3 breastfeeding	IUGR, preterm labour, premature rupture of membranes	See Infectious Diseases, ID29	Serology, viral PCR All pregnant women are offered screening	Triple antiretroviral therapy decreases transmission to <1% Elective C/S: no previous antiviral Rx or monotherapy only, viral load unknown or >500 RNA copies/ml, unknown prenatal care, patient request
***Rubella**	ssRNA togavirus	To mom: respiratory droplets (highly contagious) To baby: transplacental	T1	SA or congenital rubella syndrome (hearing loss, cataracts, CV lesions, MR, IUGR, hepatitis, CNS defects, osseous changes)	Rash (50%), fever, posterior auricular or occipital lymphadenopathy, arthralgia	Serologic testing; all pregnant women screened (immune if titre >1:16); infection if IgM present or >4x increase in IgG	No specific treatment; offer vaccine following pregnancy Do not administer during pregnancy (live attenuated)
Syphilis	Spirochete *(Treponema pallidum)*	To mom: sexual contact To baby: transplacental	T1-T3	Risk of PTL, multisystem involvement, fetal death	See Infectious Diseases, ID26	VDRL screening for all pregnancies; if positive, requires confirmatory testing	Pen G 2.4 M U IM 1 dose if early syphilis 3 doses if late syphilis monitor VDRL monthly If Pen G allergic: consider desensitization before treatment
***Toxoplasmosis**	Protozoa *(Toxoplasma gondii)*	To mom: raw meat, unpasteurized goat's milk, cat feces/urine To baby: transplacental	T3 (but most severe if infected in T1); only concern if primary infection during pregnancy	Congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial calcification, MR, microcephaly) NB: 75% initially asymptomatic at birth	Majority subclinical; may have flu-like symptoms	IgM and IgG serology; PCR of amniotic fluid	Self-limiting in mother; spiramycin decreases fetal morbidity but not rate of transmission

* Indicates TORCH infection

Venous Thromboembolism (VTE)

Epidemiology
- incidence 0.5-3/1,000 pregnancies occurring with approximately equal frequency in all three trimesters and postpartum

Risk Factors
- previous VTE, age >35, obesity, infection, bedrest/immobility, shock/dehydration, thrombophilias (congenital and acquired, see Hematology)

Table 11. Risk Factors for VTE Specific to Pregnancy

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

Virchow's Triad for VTE
1. Hypercoagulable state
2. Stasis
3. Endothelial damage

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

Investigations
- duplex venous Doppler sonography for DVT
- CXR and V/Q scan for PE
- due to hypercoagulability, the normal scale for D-dimer levels must be adjusted (controversial)

Management
- before initiating treatment, obtain a baseline CBC, including platelets, and aPTT
- warfarin is contraindicated during pregnancy due to its potential teratogenic effects
- unfractionated heparin
  - bolus of 5000 IU followed by an infusion of ~30 000 IU/24 hours
  - measure aPTT six hours after the bolus
  - maintain aPTT at a therapeutic level (1.5-2 times normal)
  - repeat q24h once therapeutic
  - heparin-induced thrombocytopenia (HIT) uncommon (3%) but serious complication
- compression stockings
- poor evidence to support a recommendation for or against avoidance of prolonged sitting
- VTE prophylaxis:
  - women on long-term anticoagulation: full therapeutic anticoagulation throughout pregnancy and for 6-12 weeks postpartum
  - women with a non-active PMHx of VTE: unfractionated heparin regimens suggested
- routine VTE prophylaxis:
  - insufficient evidence in pregnancy to recommend routine use of LMWH
  - current prophylaxis regimens for acquired thrombophilias (e.g. APLA syndrome) include low dose aspirin in conjunction with prophylactic heparin

Bleeding in Pregnancy Definitions
- First Trimester Bleeding: vaginal bleeding within the first 12 weeks
- Second Trimester Bleeding: <20 weeks

Approach to the Patient with Bleeding in T1/T2

History
- Risk factors for ectopic pregnancy (previous ectopic pregnancies, history of STI/PID, IUD use, previous pelvic surgery, smoking)
- Previous SA
- Recent trauma
- Characteristics of the bleeding (including any tissue passed)
- Characteristics of the pain (cramping pain suggests SA)
- History of coagulopathy
- Gynecological/obstetric history
- Dizziness (significant blood loss, may be associated with ruptured ectopic)
- Fever (may be associated with septic abortion)

Physical
- Vitals (including orthostatic changes)
- Abdomen (SFH, tenderness, presence of contractions)
- Perineum (signs of trauma, genital lesions)
- Speculum exam (cervical os open or closed, presence of active bleeding/clots/tissue)
- Pelvic exam (uterine size, adenexal mass, uterine/adenexal tenderness)

Bleeding in Pregnancy

First and Second Trimester Bleeding

Differential Diagnosis
- physiologic bleeding: spotting, due to implantation of placenta – reassure and check serial beta-hCGs
- abortion (threatened, inevitable, incomplete, complete) (see Table 12)
- abnormal pregnancy (ectopic, molar) (see Gynecology for *Molar Pregnancy*)
- trauma (post-coital or after pelvic exam)
- genital lesion (e.g. cervical polyp, neoplasms)

Investigations
- beta-hCG (lower than expected for GA in spontaneous abortion, ectopic pregnancy)
- U/S (confirm intrauterine pregnancy and fetal viability)
- CBC
- group and screen

Treatment
- IV resuscitation for hemorrhagic shock
- treat the underlying cause

Spontaneous Abortions

- see *Termination of Pregnancy*, OB11 for therapeutic abortions

Table 12. Classifications of Spontaneous Abortions

Type	History	Clinical	Management (± Rhogam®)
Threatened	Vaginal bleeding ± cramping	Cervix closed and soft U/S shows viable fetus	Watch and wait <5% go on to abort
Inevitable	Increasing bleeding and cramps ± rupture of membranes	Cervix closed until products start to expel, then external os opens	a) Watch and wait b) Misoprostol 400-800 ug PO/PV c) D&C ± oxytocin
Incomplete	Extremely heavy bleeding and cramps ± passage of tissue noticed	Cervix open	a) Watch and wait b) Misoprostol 400-800 ug PO/PV c) D&C ± oxytocin
Complete	Bleeding and complete passage of sac and placenta	Cervix open	No D&C – expectant management
Missed	No bleeding (fetal death in utero)	Cervix closed U/S may show SGA	a) Watch and wait b) Misoprostol 400-800 ug PO/PV c) D&C ± oxytocin
Recurrent	3+ consecutive spontaneous abortions		Evaluate mechanical, genetic, environmental and other risk factors
Septic	Contents of uterus infected – infrequent		D&C IV broad spectrum antibiotics

Etiology of Recurrent Pregnancy Loss

MAKE ME
Mechanical: uterine anatomy, cervical incompetence (T2)
Autoimmune: antiphospholipid antibody syndrome, lupus anticoagulant
Karyotype: both parents
Endocrine: hypothyroidism, diabetes mellitus
Maternal infection
Environment: smoking, alcohol, drugs, radiation

Management of Abortions
- Always rule out an ectopic
- Always check Rh; if negative, give Rhogam®
- Always ensure patient is hemodynamically stable

Ectopic Pregnancy

Definition
- embryo implants outside of the endometrial cavity

Epidemiology
- 1/100 pregnancies
- fourth leading cause of maternal mortality, leading cause of death in first trimester
- increase in incidence over the last 3 decades
- three commonest locations for ectopic pregnancy: ampullary (78%), isthmic (12%), fimbrial (5%)

Etiology
- 50% due to damage of fallopian tube cilia from PID
- intrinsic abnormality of the fertilized ovum
- conception late in cycle
- transmigration of fertilized ovum to contralateral tube

Risk Factors
- previous ectopic pregnancy
- demographics: older women, of African descent
- smoking
- endometriosis
- gynecologic:
  - IUD use – although decreased pregnancy rate, there is increased risk of ectopic if pregnancy occurs
  - history of PID (especially infection with *C. trachomatis*), salpingitis
  - infertility
  - clomiphene citrate (for induction of ovulation)
- previous procedures:
  - any surgery on fallopian tube (for previous ectopic, tubal ligation, etc.)
  - abdominal surgery for ruptured appendix, etc.
  - IVF pregnancies following ovulation induction (7% ectopic rate)
- structural:
  - uterine leiomyomas
  - adhesions
  - abnormal uterine anatomy (e.g. T-shaped uterus)

Clinical Features of Ectopic Pregnancy: 4Ts and 1S
- **T**emperature >38°C (20%)
- **T**enderness: abdominal (90%) ± rebound (45%)
- **T**enderness on bimanual examination, cervical motion tenderness
- **T**issue: palpable adnexal mass (50%) (half have contralateral mass due to lutein cyst)
- **S**igns of pregnancy (e.g. Chadwick's sign, Hegar's sign)

More than half of patients with ectopic pregnancy have no risk factors.

DDx of Lower Abdominal Pain
Urinary tract: UTI, kidney stones
GI: diverticulitis, appendicitis
Gyne: endometriosis, PID, fibroid (degenerating, infarcted, torsion), ovarian torsion, ovarian neoplasm, ovarian cyst, pregnancy-related

If Ectopic Pregnancy Ruptures
Acute abdomen with increasing pain
Abdominal distention
Shock

Investigations

- serial beta-hCG levels; normal doubling time with intrauterine pregnancy is 1.6-2.4 days in early pregnancy
  - rise of <20% of beta-hCG is 100% predictive of a nonviable pregnancy
  - prolonged doubling time, plateau or decreasing levels before 8 weeks implies nonviable gestation but does not provide information on location of implantation
- ultrasound
  - U/S is only definitive if fetal cardiac activity is detected in the tube or uterus
  - intrauterine sac should be visible when serum beta-hCG is
    - >1500 mIU/mL (transvaginal)
    - >6000 mIU/mL or 6 weeks gestational age (transabdominal)
  - specific finding on transvaginal U/S is a tubal ring
- culdocentesis (rarely done)
- laparoscopy (for definitive diagnosis)

Treatment

- goals of treatment: conservative (preserve tube if possible)
- surgical (laparoscopy)
  - linear salpingostomy if tube salvageable
  - salpingectomy if tube damaged or ectopic is ipsilateral recurrence
  - 15% risk of persistent trophoblast; must monitor beta-hCG titres weekly until they reach non-detectable levels
  - if patient is Rh negative give anti-D gamma globulin (Rhogam®)
  - may require laparotomy
- medical = methotrexate
  - use 50 mg/m^2 body surface area; given in a single IM dose
  - this is 1/5 to 1/6 chemotherapy dose, therefore minimal side effects (reversible hepatic dysfunction, diarrhea, gastritis, dermatitis)
  - follow beta-hCG levels weekly until beta-hCG is non-detectable
    - plateau or rising levels suggest persisting trophoblastic tissue (requires further treatment)
  - 67% success rate; up to 25% will require a 2nd dose
  - tubal patency following methotrexate treatment approaches 80%

Prognosis

- 9% of maternal deaths during pregnancy
- 40-60% of patients will become pregnant again after surgery
- 10-20% will have subsequent ectopic gestation

Interventions for Tubal Ectopic Pregnancy
Cochrane Database of Systematic Reviews 2007, Issue 1
Study: Cochrane Review of randomized controlled trials comparing treatments in women with tubal ectopic pregnancy.
Patients: Women with a diagnosis of tubal ectopic pregnancy.
Intervention: Surgery-salpingectomy/salpingostomy by open surgery or by laparoscopy, medical treatment, and expectant management.
Main outcome: Primary treatment success, defined as an uneventful decline in serum beta-hCG to undetectable levels by the initial treatment.
Results: Intramuscular MTX therapy and salpingostomy yielded similar treatment success rates (82-95% for MTX therapy vs. 80-92% for salpingostomy).

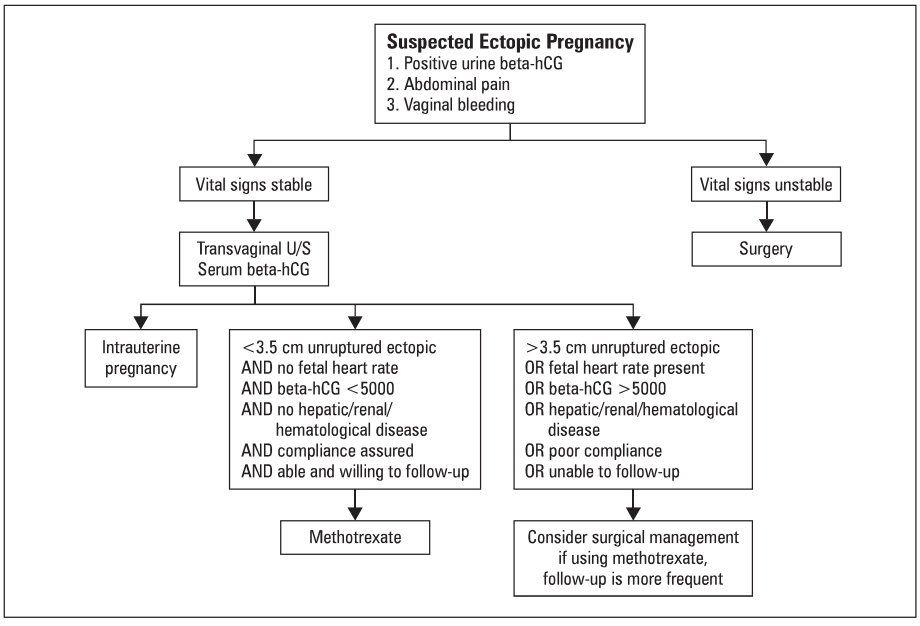


Figure 3. Algorithm for Suspected Ectopic Pregnancy

Antepartum Hemorrhage

Definition
- vaginal bleeding from 20 weeks to term

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

Placenta Previa

Definition
- abnormal location of the placenta near, partially or completely over the cervical os

Etiology
- idiopathic

Epidemiology
- incidence = 0.5-0.8% of all pregnancies

Risk Factors
- history of placenta previa (4-8% recurrence risk)
- multiparity
- increased maternal age
- multiple gestation
- uterine tumour (e.g. fibroids) or other uterine anomalies
- uterine scar due to previous abortion, C/S, D&C, myomectomy

Clinical Features
- **classification**
  - total: placenta completely covers the internal os
  - partial: placenta partially covers the internal os
  - marginal: within 2 cm of os but does not cover any part of os – causes potential risk of hemorrhage during cervical effacement and dilatation
  - low lying (NOT a previa): placenta in lower segment but clear of os (can also bleed, but usually in labour)
- **history**
  - **PAINLESS** bright red vaginal bleeding (recurrent), may be minimized and cease spontaneously, but can become catastrophic
  - mean onset of bleeding is 30 wks GA, but onset depends on degree of previa (total placenta previa bleed earlier, marginal bleed at onset of labour)
- **physical exam**
  - uterus soft and non-tender
  - presenting part high or displaced
- **complications**
  - fetal
    - perinatal mortality low but still higher than with a normal pregnancy
    - prematurity (bleeding often dictates early C/S)
    - intrauterine hypoxia (acute or IUGR)
    - fetal malpresentation
    - PPROM
    - risk of fetal blood loss from placenta, especially if incised during C/S
  - maternal
    - <1% maternal mortality
    - hemorrhage and hypovolemic shock, anemia, acute renal failure, pituitary necrosis (Sheehan syndrome)
    - placenta accreta – especially if previous uterine surgery, anterior placenta previa
    - hysterectomy

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

Levels of abnormal placental invasion:
Placenta Accreta – AT myometrium (most common)
Placenta Increta – INTO myometrium
Placenta Percreta – PASSES myometrium

Investigations
- ultrasound diagnosis (transabdominal ultrasound has 95% accuracy)
- due to development of lower uterine segment, 90-95% of previas diagnosed in T2 resolve by T3
- partial or total previas: repeat U/S at 30-32 weeks
- low-lying: repeat U/S not indicated unless recurrent bleeding

Management
- goal: keep pregnancy intrauterine until the risk of delivery < risk of not continuing pregnancy
- stabilize and monitor
  - maternal stabilization: large bore IV with hydration, O_2 for hypotensive patients
  - maternal monitoring: vitals, urine output, blood loss, bloodwork (hematocrit, CBC, INR/PTT, platelets, fibrinogen, FDP, type and cross match)
  - electronic fetal monitoring
  - U/S assessment: when fetal and maternal condition permit, determine fetal viability, gestational age and placental status/position
- Rhogam® if mother is Rh negative
  - Kleihauer-Betke test to determine extent of fetomaternal transfusion so that appropriate dose of Rhogam® can be given
- GA <37 weeks and minimal bleeding – expectant management
  - admit to hospital
  - limited physical activity, no douches, enemas, or sexual intercourse
  - consider corticosteroids for fetal lung maturity
  - delivery when fetus is mature or hemorrhage dictates
- GA ≥37 weeks, profuse bleeding or L/S ratio is >2:1 – deliver by C/S

Kleihauer-Betke Test
Quantifies fetal cells in the maternal circulation.

Abruptio Placentae

Definition
- premature separation of a normally implanted placenta after 20 weeks GA

Etiology
- most are idiopathic

Epidemiology
- incidence: 1-2% of all pregnancies

Risk Factors
- previous abruption (recurrence rate 5-16%)
- maternal hypertension (chronic or PIH in 50% of abruptions) or vascular disease
- cigarette smoking (>1 pack/day), excessive alcohol consumption, cocaine
- multiparity and/or maternal age >35 (felt to reflect parity)
- PPROM
- rapid decompression of a distended uterus (polyhydramnios, multiple gestation)
- uterine anomaly, fibroids
- trauma (e.g. motor vehicle collision, maternal battery)

Clinical Features
- **classification**
  - total (fetal death inevitable) vs. partial
  - external/revealed/apparent: blood dissects downward toward cervix
  - internal/concealed (20%): blood dissects upward toward fetus
  - most are mixed
- **presentation**
  - **PAINFUL** vaginal bleeding, uterine tenderness, uterine contractions
  - pain: sudden onset, constant, localized to lower back and uterus
  - ± fetal distress, fetal demise (15% present with demise), bloody amniotic fluid

Abruptio placentae is the most common cause of DIC in pregnancy.

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

Investigations
- clinical diagnosis: ultrasound not sensitive for abruption (sensitivity = 15%)

Table 13. Grades of Abruptio Placentae

Grade	Uterine Irritability	Maternal Hemodynamics	Maternal Fibrinogen	FHR
Mild	Mild	Normal	Normal	Normal
Moderate	Moderate-severe ± tetany	BP with postural drop Increased HR	Decreased	Distress: decreased variability Late decelerations
Severe	Tetany	Decreased BP, decreased HR	Extremely decreased	Absent

Management
- maternal stabilization: large bore IV with hydration; O_2 for hypotensive patients
- maternal monitoring: vitals, urine output, blood loss, bloodwork (hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match)
- electronic fetal monitoring
- blood products on hand (red cells, platelets, cryoprecipitate) because of DIC risk
- Rhogam® if Rh negative
  - Kleihauer-Betke test may confirm abruption
- mild abruption:
  - GA <37 weeks: use serial Hct to assess concealed bleeding, deliver when fetus is mature or hemorrhage dictates
  - GA ≥37 weeks: stabilize and deliver
- moderate to severe abruption:
  - hydrate and restore blood loss and correct coagulation defect if present
  - vaginal delivery if no contraindication and no evidence of fetal or maternal distress OR fetal demise
  - C/S if live fetus and fetal or maternal distress develops with fluid/blood replacement, labour fails to progress or if vaginal delivery otherwise contraindicated

Table 14. Comparison of Abruptio Placenta and Placenta Previa

Abruptio Placenta	Placenta Previa
Abdominal PAIN and/or backache	PAINLESS
Uterine TENDERNESS	NO tenderness
INCREASED uterine tone	Uterus SOFT
Uterine IRRITABILITY/CONTRACTIONS	No uterine irritability/contractions
Usually NORMAL fetal presentation	Malpresentation and/or high presenting part
FHR may be ABSENT or abnormal tracing	FHR usually NORMAL
Shock and anemia OUT OF PROPORTION to apparent blood loss	Shock and anemia CORRESPOND to apparent blood loss
May have COAGULOPATHY	Coagulopathy very UNCOMMON initially

Vasa Previa

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

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

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

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

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

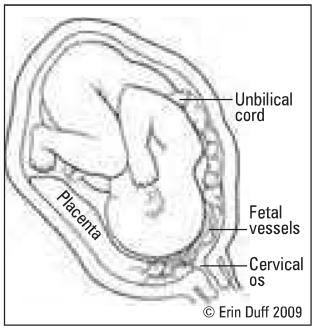


Figure 4. Vasa Previa

Multiple Gestation

Epidemiology

- incidence of twins is 1/80 and triplets 1/6400 in North America
- 2/3 of twins are dizygotic (fraternal)
  - risk factors for dizygotic twins: IVF, increased maternal age, newly discontinued OCP, ethnicity (e.g. certain African regions)
- monozygous twinning occurs at a constant rate worldwide (1/250)
- determine zygosity by number of placentas, thickness of membranes, sex, blood type

The P's of Multiple Gestation Complications

Increased rates of:

- **P**uking
- **P**allor (anemia)
- **P**reeclampsia/**P**IH
- **P**ressure (compressive symptoms)
- **P**TL/**P**ROM/**P**PROM
- **P**olyhydramnios
- **P**lacenta previa/abruptio
- **P**PH/APH
- **P**rolonged labour
- Cord **P**rolapse
- **P**rematurity
- Mal **P**resentation
- **P**erinatal morbidity and mortality
- **P**arental distress
- **P**ostpartum depression

Clinical Features

Table 15. Complications Associated with Multiple Gestation

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

*Most common cause of perinatal mortality in multiple gestation

Management

- U/S determination of chorionicity must be done within first trimester (ideally 8-12 weeks GA)
- increased antenatal surveillance
  - nonstress test (NST) weekly from 24 weeks GA
  - serial U/S q 2-3 weeks from 28 weeks GA to assess growth
  - Doppler flow studies weekly if discordant fetal growth
  - BPP as needed
- vaginal examinations in third trimester to check for cervical dilatation
- may attempt vaginal delivery if twin A presents as vertex, otherwise C/S (40-50% of all twin deliveries, 15% of cases have twin A delivered vaginally and twin B delivered by C/S)
- mode of delivery depends on fetal weight, GA, presentation

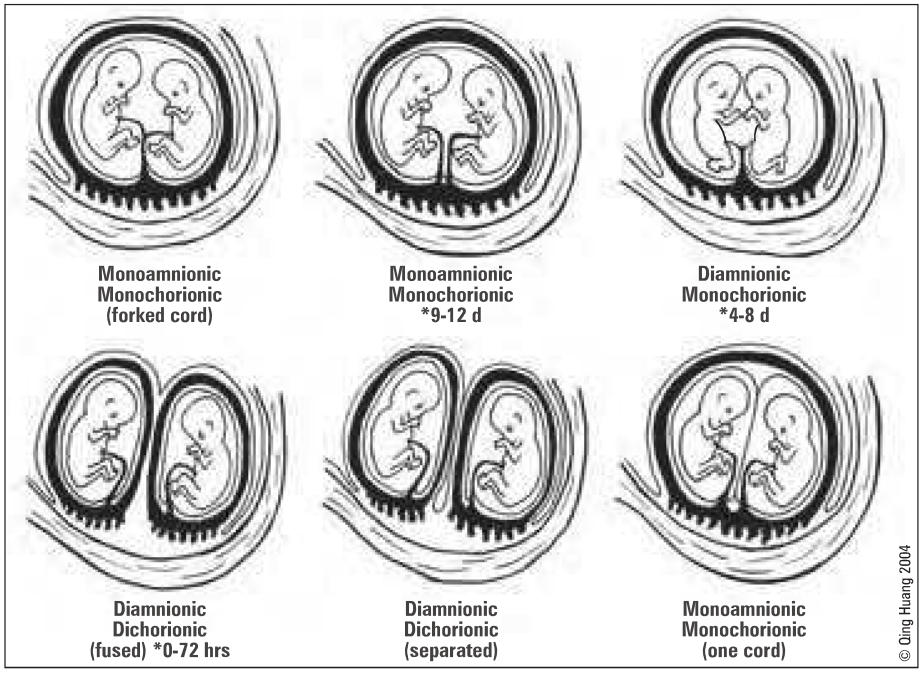


Figure 5. Classification of Twin Pregnancies *Indicates time of cleavage

Twin-Twin Transfusion Syndrome

Epidemiology
- 10% of monochorionic twins

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

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

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

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

Growth Discrepancies

Intrauterine Growth Restriction (IUGR)

Definition
- common definition: infant weight <10th percentile for GA
- other definitions: infant <2500 g, Ponderal Index: birth weight (gm)/crown-heel lenght (cm) x 100

Etiology/Risk Factors
- maternal causes
  - malnutrition, smoking, drug abuse, alcoholism, cyanotic heart disease, Type 1 DM, SLE, pulmonary insufficiency, previous IUGR
- maternal-fetal
  - any disease causing placental insufficiency
  - includes gestational HTN, chronic HTN, chronic renal insufficiency, gross placental morphological abnormalities (infarction, hemangiomas)
- fetal causes: TORCH infections, multiple gestation, congenital anomalies

TORCH
Toxoplasmosis
Others: e.g. syphilis
Rubella
CMV
HSV

• See Table 10, OB19

Clinical Features
- symmetric/Type I (20%) – occurs early in pregnancy
  - inadequate growth of head and body
  - **head:abdomen ratio** may be normal (>1 up to 32 weeks; =1 at 32-34 weeks; <1 after 34 weeks GA)
  - usually associated with congenital anomalies or TORCH infections
- asymmetric/Type II (80%) – occurs late in pregnancy
  - brain is spared, therefore head:abdomen ratio increased
  - usually associated with placental insufficiency
  - more favorable prognosis than Type I
- complications
  - prone to meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation
  - greater risk of perinatal morbidity and mortality

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

Investigations
- symphysis-fundus height (SFH) measurements at every antepartum visit
- if mother at high risk or SFH lags >2 cm behind GA:
  - anatomy U/S for biparietal diameter (BPD), head and abdomen circumference, femur length and fetal weight, amniotic fluid volume (decrease associated with IUGR)
  - ± BPP
  - Doppler analysis of umbilical cord blood flow

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

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

Macrosomia

Definition
- infant weight >90th percentile for a particular GA or >4000 g

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

Clinical Features
- increased risk of perinatal mortality
- cephalopelvic disproportion (CPD) and birth injuries (shoulder dystocia, fetal bone fracture) more common
- complications of DM in labour (see *Medical Conditions in Pregnancy,* OB11)

Investigations
- serial SFH
- further investigations if mother at high risk or SFH >2 cm ahead of GA
- U/S predictors
  - polyhydramnios
  - third trimester abdominal circumference (AC) >1.5 cm/week
  - head circumference (HC)/AC ratio <10th percentile
  - femur length (FL)/AC ratio <20th percentile

Management
- prophylactic C/S is a reasonable option where estimated fetal weight (EFW) >5000 g in nondiabetic women and EFW >4500 g in diabetic women
  - no evidence that prophylactic C/S improves outcomes
- early induction of labour is not recommended for non-diabetic mothers
- risks and benefits of early induction (risk of C/S vs. risk of dystocia) must be weighed in diabetic mothers, as current research is unclear

Polyhydramnios/Oligohydramnios

Table 16. Polyhydramnios and Oligohydramnios

	Polyhydramnios	Oligohydramnios
Definition	Amniotic fluid volume (AFV) >2,000 cc at any stage in pregnancy U/S criteria: >8 x 8 cm (3.1 x 3.1 in) pocket of amniotic fluid	Amniotic fluid index of 5 cm (2 in) or less • Important sign of chronic placental insufficiency
Etiology	Idiopathic most common (40%) Maternal: • Type 1 DM: abnormalities of transchorionic flow Maternal-fetal: • Chorioangiomas • Multiple gestation • Fetal hydrops (increased erythroblastosis) Fetal: • Chromosomal anomaly (up to 2/3 of fetuses have severe polyhydramnios) • Respiratory: cystic adenomatoid malformed lung • CNS: anencephaly, hydrocephalus, meningocele • GI: tracheoesophageal fistula, duodenal atresia, facial clefts (interfere with swallowing)	Early onset oligohydramnios: • Decreased production: renal agenesis or dysplasia, urinary obstruction, posterior urethral valves (male), chronic hypoxemia and IUGR resulting in shunting away from the kidneys to ensure perfusion of the brain • Increased loss: prolonged amniotic fluid leak (although most often labour ensues) Late onset oligohydramnios: • Amniotic fluid normally decreases after 35 weeks • Common in post-term pregnancies • U/S Doppler studies (umbilical cord and uterine artery)
Epidemiology	Occur in 0.2 to 1.6% of all pregnancies	Occur in ~4.5% of all pregnancies Severe form in <0.7% Common in pregnancies >41 weeks (~12%)
Clinical Features and Complications	Pressure symptoms from overdistended uterus (dyspnea, edema, hydronephrosis) Uterus large for dates, difficulty palpating fetal parts and hearing fetal heart tones Cord prolapse, placental abruption, malpresentation, preterm labour, uterine dysfunction and PPH	Cord compression Increased risk of adverse fetal outcomes Early onset: • 15-25% have fetal anomalies • Amniotic fluid bands (T1) can lead to Potter's facies, limb deformities, abdominal wall defects Late onset: • Pulmonary hypoplasia • Marker for infants who may not tolerate labour well
Management	Determine underlying cause: • Screen for maternal disease/infection • Complete fetal U/S evaluation Depends on severity: • Mild to moderate cases require no treatment • If severe, hospitalize and consider therapeutic amniocentesis	Always warrants admission and investigation: • Rule out rupture of membranes (ROM) • Fetal monitoring (NST, CTG, BPP) • U/S Doppler studies (umbilical cord and uterine artery) Maternal hydration with oral or IV fluids to help increase amniotic fluid Vesicoamniotic shunt: if etiology is related to fetal obstuctive uropathy; however, pulmonary function may not be restored with restoration of amniotic fluid Injection of fluid via amniocentesis will improve condition for ~1 wk – may be most helpful for visualizing any associated fetal anomalies Consider delivery if term Amnio-infusion may be considered during labour via intra-uterine catheter
Prognosis	Two to five-fold increase in risk of perinatal mortality	Poorer with early onset High mortality related to congenital malformations and pulmonary hypoplasia when diagnosed during T2

Normal Labour and Delivery

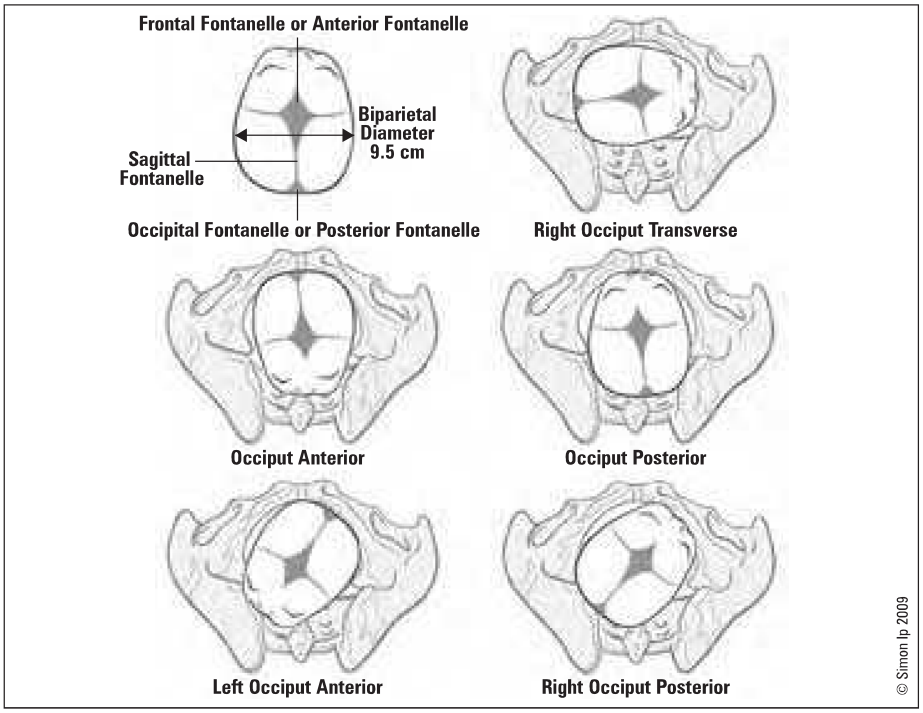


Figure 6. Fetal Positions

The Fetus

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

Presenting Parts include:
Occiput for vertex
Sacrum for breech
Mentum for face

- **fetal presentation**
  - fetal part presenting at pelvic outlet
    - breech (complete, frank, footling) – see Figure 8, OB40
    - cephalic (vertex, face, asynclitic)
    - transverse (shoulder)
    - compound (fetal extremity prolapses along with presenting part)
  - all except vertex are considered malpresentations (see *High Risk Labour and Delivery*, OB37)

Fetal lie: long axis of fetus compared to long axis of uterus

Fetal presentation: fetal part at pelvic outlet

Fetal position: position of presenting part relative to pelvis

- **fetal position**
  - position of presenting part of the fetus relative to the maternal pelvis
    - occiput anterior (OA): most common presentation ("normal") – left OA most common
    - 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**
  - flexion/extension of fetal head relative to shoulders
    - brow presentation: head partially extended (requires C/S)
    - face presentation: head fully extended
      - mentum posterior always requires C/S, mentum anterior will deliver vaginally

- **station**
  - position of presenting part relative to ischial spines – determined by vaginal exam
    - at ischial spines = station 0 = engaged
    - cm above (–5 → –1) or cm below (+1 → +5)

The Cervix

- dilatation: latent phase: 0-3 cm; active phase: 4-10 cm
- effacement: thinning of the cervix by percentage or length of cervix (cm)
- consistency: firm vs. soft
- position: posterior vs. anterior
- application: contact between the cervix and presenting part (i.e. well or poorly applied)
- see Bishop score (Table 20, OB36)

Definition of Labour

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

Analgesic and Anaesthetic Techniques in Labour and Birth

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

Non-pharmacologic Pain Relief Techniques

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

Pharmacologic Methods

- nitrous oxide (e.g. self-administered Entonox®)
- narcotics (usually combined with anti-emetic)
- pudental nerve block
- perineal infiltration with local anesthetic
- regional anaesthesia (epidural block)

Four Stages of Labour

Course of Normal Labour

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

First Stage of Labour

- latent phase
  - uterine contractions typically infrequent and irregular
  - slow cervical dilatation (usually to 3-4 cm) and effacement
- active phase
  - rapid cervical dilatation to full dilatation (nulliparous ~1.2 cm/h, multiparous ~1.5 cm/h)
  - phase of maximum slope on cervical dilatation curve (see Figure 9, OB43)
  - painful, regular contractions q2-3 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
- women may choose a comfortable position that enhances pushing efforts and delivery
  - upright (semi-sitting, squatting) and LLDP are supported in the literature
- progress measured by descent

Signs of Placental Separation
1. Gush of blood
2. Lengthening of cord
3. Uterus becomes globular
4. Fundus rises

Continuous Support for Women During Childbirth
Cochrane Database of Systematic Reviews 2007, Issue 3
Study: Systematic review of 16 RCTs from 11 countries, 13,391 women in labour.
Intervention: Continuous support during labour vs. usual care.
Outcome: Effects on mothers and their babies.
Results: Continuous intrapartum support increased likehood of shorter labour, spontaneous vaginal birth, decrease in analgesia use, and a decrease in dissatisfaction with childbirth experience. Greatest benefit when provider is not a health care professional.

Third Stage of Labour
- separation and expulsion of the placenta
- can last up to 30 minutes before intervention indicated
- start oxytocin IV drip or give 10 U IM after delivery of anterior shoulder in anticipation of placental delivery, otherwise give after delivery of placenta
- routine oxytocin administration in third stage of labour can reduce the risk of PPH by >40%

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

The Cardinal Movements of the Fetus During Delivery

Figure 7. Cardinal Movements of Fetus During Delivery
Adapted from illustration in *Williams Obstetrics,* 19th Ed.

Fetal Monitoring in Labour

- see online **Fetal Heart Rate Tutorial**

Vaginal Exam

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

Intrapartum Fetal Monitoring

- intermittent fetal auscultation with Doppler device q15-30 minutes for one minute in first stage active phase following a contraction, q5 minutes during second stage when pushing has begun
- continuous electronic FHR monitoring reserved for non-reassuring auscultation, prolonged labour, and labour which is induced or augmented
  - routine use of continuous electronic monitoring shown to lead to higher intervention rates and no improvement in outcome for the neonate
  - techniques for continuous monitoring include external (Doppler) vs. internal (fetal scalp electrode) monitoring
- fetal scalp sampling should be used in conjunction with electronic FHR monitoring and contraction monitoring (cardiotocometry – CTG) to resolve the interpretation of non-reassuring patterns

Electronic Fetal Heart Rate (FHR) Monitoring

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

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

- **Variability**
  - physiologic variability is a normal characteristic of FHR
  - effect of vagus nerve on fetal heart
  - normal variability indicates fetal acid-base status is acceptable
  - can only be assessed by electronic fetal monitoring (CTG)
  - variability decreases intermittently even in healthy fetus

- **Periodicity**
  - accelerations: increase of ≥15 bpm lasting ≥15 seconds, in response to fetal movement or uterine contraction (or ≥10 bpm lasting ≥10 sec if <32 wks GA)
  - decelerations: 3 types, described in terms of shape, onset, depth, duration recovery, occurrence, and impact on baseline FHR and variability (see Table 18)

Table 17. Factors Affecting Fetal Heart Rate

	Fetal Tachycardia (FHR >160)	Fetal Bradycardia (FHR <110)	Decreased Variability
Maternal Factors	Fever Hyperthyroidism Anemia	Hypothermia Hypotension Hypoglycemia	Infection Dehydration
Fetal Factors	Arrhythmia Anemia	Rapid descent Dysrhythmia Heart block	CNS anomalies Dysrhythmia Inactivity/sleep cycle, preterm fetus
Drugs	Sympathomimetics	β-blockers Anesthetics	Narcotics, sedatives Magnesium sulphate, β-blockers
Uteroplacental	Early hypoxia (abruption, HTN) Chorioamnionitis	Late hypoxia (abruption, HTN) Acute cord prolapse Hypercontractility	Hypoxia

Fetal Scalp Blood Sampling

- indicated when non-reassuring fetal heart rate (NRFHR) is suggested by clinical parameters including heavy meconium or moderately to severely abnormal FHR patterns, including unexplained low variability, repetitive late decelerations, complex variable decelerations, fetal cardiac arrythmias
  - pH ≥7.25: normal, repeat if abnormal FHR persists
  - pH 7.21-7.24: repeat assessment in 30 minutes or consider delivery if rapid fall since last sample
  - pH ≤7.20: indicates fetal acidosis, delivery is indicated
- contraindications
  - known or suspected fetal blood dyscrasia (hemophilia, von Willebrand disease)
  - active maternal infection (HIV, genital herpes)

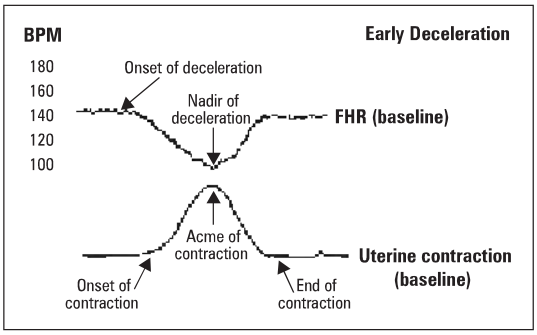
Continuous Cardiotocography (CTG) as a Form of Electronic Fetal Monitoring (EFM) for Fetal Assessment during Labour
Cochrane Database of Systematic Reviews 2006, Issue 3
Purpose: To examine the effectiveness of continuous fetal heart monitoring (cardiotocography) during labour on improving health outcomes.
Methods: Systematic review comparing continuous fetal monitoring with no monitoring, intermittent auscultation, and intermittent monitoring.
Results: 12 trials (37 000 women) meeting search criteria were identified, of which 2 trials were high quality. Continuous electronic fetal heart monitoring did not have an effect on overall perinatal death rate compared to intermittent auscultation, with a relative risk (RR) or 0.85, 95% CI 0.59-1.23. Continuous monitoring also lead to increased incidence of C-section (RR 1.66, 95% CI 1.30 to 2.13, n=18,761, 10 trials) and instrument assisted vaginal delivery (RR 1.16, 95% CI 1.01 to 1.32, n = 18,151, nine trials). These results appeared consistent regardless if pregnancy was high risk, low risk, or pre-term.
Summary: Continuous fetal cardiotocography does not significantly improve infant mortality or other standards of infant well-being. It increases the incidence of C-section and instrument assisted vaginal delivery.

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

Approach to the Management of Abnormal FHR
Ensure fetal tracing
Call for help
Change position to LLDP
100% O_2 by mask
Stop oxytocin
Correct maternal hypotension
Fetal scalp pH/fetal scalp electrode
Vaginal exam to rule out cord prolapse
Rule out fever, dehydration, drug effects, prematurity
Amnioinfusion or tocolytics in selected cases
C/S when necessary

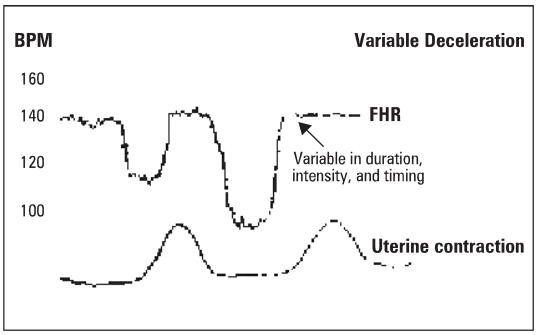
Table 18. Comparison of Decelerations

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



Variable Decelerations
- Variable in shape, onset, and duration
- Most common type of periodicity seen during labour
- Often with abrupt drop in FHR; usually no effect on baseline FHR or variability
- Due to cord compression or, in second stage, forceful pushing with contractions

Complicated Variable Decelerations
- To <70 bpm for >60 sec
- Loss of variability or decrease in baseline after deceleration
- Biphasic deceleration
- Slow return to baseline
- Baseline tachycardia or bradychardia



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

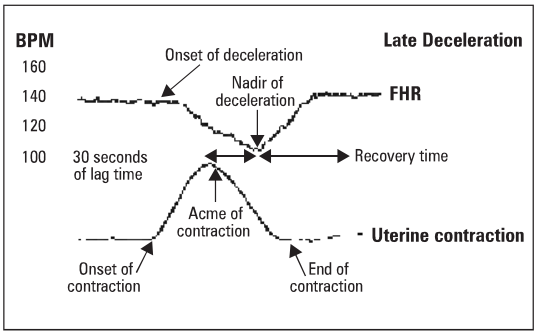


Table 19. Classification of Intrapartum EFM Tracings

	Normal Tracing (Category 1)	Atypical Tracing* (Category 2)	Abnormal Tracing* (Category 3)
Baseline	110-160 bpm	Bradycardia 100-100 bpm Tachycardia >160 for 30-80 min Rising baseline	Bradycardia <100 bpm Tachycardia >160 bpm for >80 min Erratic baseline
Variability	6-25 bpm ≤5 bpm for <40 min	≤5 bpm for 40-80 min	<5 bpm for >80 min ≥25 bpm for >10 min
Decelerations	None Early decelerations Occasional uncomplicated variable decelerations	Repetitive (≥3) uncomplicated variable decelerations Occasional late decelerations Any prolonged deceleration 2-3 min	Repetitive (≥3) complicated variable decelerations Repetitive late decelerations Any prolonged deceleration >3 min
Accelerations	Accelerations spontaneous or during scalp stimulation	Absent with scalp stimulation	Nearly absent
Action	EFM may be interrupted for ≤30 min if mother/fetus stable	Further assessment required	Action required: review clinical situation, obtain scalp pH, prepare for possible delivery

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

Fetal Oxygenation

- uterine contractions during labour decrease uteroplacental blood flow, which results in reduced oxygen delivery to the fetus
- most fetuses tolerate this reduction in flow and have no adverse effects
- distribution of oxygen to the fetus depends on maternal, uteroplacental and fetal factors
- **maternal factors**
  - decreased maternal oxygen carrying capacity
    - significant anemia (iron deficiency, hemoglobinopathies)
    - carboxyhemoglobin (smokers)
  - decreased uterine blood flow
    - hypotension (blood loss, sepsis)
    - regional anesthesia
    - maternal positioning
  - chronic maternal conditions
    - vasculopathies (lupus, Type 1 DM, chronic HTN)
    - antiphospholipid syndrome
    - cyanotic heart disease
    - COPD
- **uteroplacental factors**
  - uterine hypertonus
    - hyperstimulation secondary to oxytocin, prostaglandins or normal labour
    - placental abruption
  - uteroplacental dysfunction
    - placental abruption
    - placental infarction (dysfunction marked by IUGR, oligohydramnios, abnormal Doppler studies)
    - chorioamnionitis
    - placental edema (diabetes, hydrops)
    - placental senescence (post dates)
- **fetal factors**
  - cord compression
    - oligohydramnios
    - cord prolapse or entanglement
  - decreased fetal oxygen carrying capability
    - significant anemia (isoimmunization, feto-maternal bleed)
    - carboxyhemoglobin (exposure to smokers)
- **fetal response to hypoxia/asphyxia**
  - decreased movement, tone, and breathing activities
  - redistribution of fetal blood flow
    - increased flow to brain, heart, and adrenals
    - decreased flow to kidneys, lungs, gut, liver and peripheral tissues
    - increase in blood pressure
  - transient fetal bradycardia followed by fetal tachycardia
  - anaerobic metabolism (decreased pH)

Induction of Labour

Definition

- artificial initiation of labour before its spontaneous onset for the purpose of delivery of the fetus and placenta

Induction vs. Augmentation

Induction is the artificial initiation of labour.

Augmentation promotes contractions when spontaneous contractions are inadequate.

Prerequisites for Labour Induction

- capability for C/S if necessary
- maternal
  - short, thin, soft, anterior cervix with open os ("inducible" or "ripe")
  - if cervix is not ripe, use prostaglandin vaginal insert (Cervidil®), prostaglandin gel (Prepidil®), or Foley catheter
- fetal
  - reassuring fetal heart tracing
  - cephalic presentation
  - adequate fetal monitoring available
- likelihood of success determined by Bishop score (see Table 20)
  - cervix considered unfavourable if <6
  - cervix favourable if ≥6
  - score of 9-13 associated with high likelihood of vaginal delivery

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

Table 20. Bishop Score

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

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

Indications
- post-date pregnancy (generally >41 weeks)
- maternal factors
  - significant antepartum hemorrhage
  - gestational HTN
  - other maternal medical problems, e.g. diabetes, renal or lung disease
- maternal-fetal factors
  - isoimmunization, PROM, chorioamnionitis, post-term pregnancy
- fetal factors
  - suspected fetal jeopardy as evidenced by biochemical or biophysical indications
  - fetal demise, severe IUGR

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

Contraindications
- maternal
  - prior classical or inverted-T incision or uterine surgery (e.g. myomectomy)
  - unstable maternal condition
  - gross CPD (although diagnosis cannot be made until active labour)
  - active maternal genital herpes
  - invasive cervical carcinoma
  - pelvic structure deformities
- maternal-fetal
  - placenta previa or vasa previa
  - cord presentation
- fetal
  - fetal distress, malpresentation, preterm fetus without lung maturity

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

Use of Prostaglandins in Cervical Ripening and Induction Intravenous Prostaglandin for Induction of Labour
- Prostaglandin E2 and F2 alpha can be used for cervical ripening and induction of labour. A meta-analysis comparing intravenous prostaglandin with oxytocin concluded that intravenous prostaglandin was no more likely to result in vaginal delivery (RR 0.85). Prostaglandins were associated with significantly more maternal side effects including gastrointestinal problems, thrombophlebitis and pyrexia. Currently, there is not enough evidence to draw any conclusions about the relative effects of prostaglandins vs. oxytocin and the choice is between the patient and the physician.
- Intravaginal prostaglandins are associated with higher rate of uterine hypertonus, uterine hyperstimulation, and fetal heart rate abnormalities.
- Prostaglandins are associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction.

Cochrane Review. The Cochrane Library, 2000. Issue 2

Induction Methods

CERVICAL RIPENING

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

Methods
- intravaginal prostaglandin PGE2 gel (Prostin® gel): long and closed cervix with no ROM
  - recommended dosing interval of prostaglandin gel is every 6 to 12 hours up to 3 doses
- intravaginal PGE2 (Cervidil®): long and closed cervix, may use if ROM
  - continuous release, can be removed if needed
  - controlled release PGE2
- Foley catheter placement to mechanically dilate the cervix
- hydroscopic dilators, osmotic dilators (laminaria)
- misoprostol: synthetic methylated PGE1 (not commonly used)

INDUCTION OF LABOUR

Amniotomy
- artificial rupture of membranes (amniotomy) to stimulate PG synthesis and secretion; may try this as initial measure if cervix is dilated
- few studies address the value of amniotomy alone for induction of labour
- amniotomy plus intravenous oxytocin: more women delivered vaginally at 24 hours than amniotomy alone (relative risk = 0.03) and had fewer instrumental vaginal deliveries (relative risk = 5.5)

Oxytocin
- oxytocin (Pitocin®): 10 U in 1L NS, run at 0.5-2 mU/min IV increasing by 1-2 mU/min q20-60 min to a max of 36-48 mU/min
  - reduces rate of unsuccessful vaginal deliveries within 24 hours when used alone (8.3% vs. 54%, RR 0.16)
  - ideal dosing regime of oxytocin is not known
  - current recommendations: use the minimum dose to achieve active labour and increase every 30 minutes as needed
  - reassessment should occur once a dose of 20 mU/min is reached
- potential complications
  - hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus)
  - uterine muscle fatigue, uterine atony (may result in PPH)
  - vasopressin-like action causing anti-diuresis

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

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

Augmentation of Labour

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

High Risk Labour and Delivery

Preterm Labour (PTL)

Definition
- labour occurring between 20 and 37 weeks gestation

Etiology
- idiopathic (most common)
- **maternal**: infection (recurrent pyelonephritis, untreated bacteriuria, chorioamnionitis), genital infection (bacterial vaginosis is associated with a twofold increase in relative risk of preterm birth), HTN, DM, chronic illness, mechanical factors, previous obstetric, gynecological and abdominal surgeries, socio-environmental (poor nutrition, smoking, drugs, alcohol, stress)
- **maternal-fetal**: PPROM (common), polyhydramnios, placenta previa or abruption, placental insufficiency
- **fetal**: multiple gestation, congenital abnormalities of fetus, fetal hydrops
- **uterine**: incompetent cervix, excessive enlargement (hydramnios), malformations (leiomyomas, septate uterus)

Epidemiology
- preterm labour complicates about 10% of pregnancies

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

PTL Recurrence
Definition: 3 or more consecutive lost pregnancies prior to 20th week of gestation
- 15% sporadic loss of 1 pregnancy
- 2% experience 2 consecutive pregnancy losses
- 0.4-1% experience 3 consecutive pregnancy losses

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

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

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

B. Suppression of Labour – Tocolysis
- does not inhibit preterm labour completely, but may buy time to allow for betamethasone valerate (Celestone®) and/or transfer to appropriate centre
- requirements (all must be satisfied)
  - preterm labour
  - live, immature fetus, intact membranes, cervical dilatation of <4 cm
  - absence of maternal or fetal contraindications
- contraindications
  - maternal: bleeding (placenta previa or abruption), maternal disease (hypertension, diabetes, heart disease), preeclampsia or eclampsia, chorioamnionitis
  - fetal: erythroblastosis fetalis, severe congenital anomalies, fetal distress/demise, IUGR, multiple gestation (relative)
- tocolytic procedure
  - ensure availability of necessary personnel and equipment to assess mother and fetus during labour and care for baby of the predicted GA if therapy fails
  - if no contraindications present, agent used depends on clinical situation
  - should be used only for <48 hr and/or until transfer to an appropriate facility for care of the premature infant
  - proven efficacy
    - first line: calcium channel blockers: nifedipine
    - second line: prostaglandin (PG) synthesis inhibitors: indomethacin
    - some emerging evidence for use of progesterone
  - no proven efficacy
    - nitroglycerin patch: vasodilator and smooth muscle relaxant
    - magnesium sulfate (if diabetes or cardiovascular disease present)

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

D. Cervical Cerclage
- definition: placement of cervical sutures, wires or synthetic tape at the level of the internal os, usually at the end of the first trimester and removed in the third trimester
- indications: cervical incompetence (i.e. cervical dilation and effacement in the absence of increased uterine contractility)
  - emerging evidence indicates that progesterone suppositiories are superior to cerclage in preventing preterm labor late in pregnancy
- diagnosis of cervical incompetence
  - obstetrical Hx: silent cervical dilation
  - ability of cervix to hold an inflated Foley catheter during a hysterosonogram
- proven benefit in the prevention of PTL in women with primary structural abnormality of the cervix (e.g. conization of the cervix, connective tissue disorders)
- benefit is variable in those with secondary cervical incompetence causing premature ripening of the cervix (e.g. infection, abnormal placentation)

Prognosis
- prematurity is the leading cause of perinatal morbidity and mortality
  - 30 weeks or 1500 g (3.3 lb) = 90% survival
  - 33 weeks or 2000 g (4.4 lb) = 99% survival
- morbidity due to asphyxia, hypoxia, sepsis, respiratory distress syndrome (RDS), intraventricular cerebral hemorrhage, thermal instability, retinopathy of prematurity, bronchopulmonary dysplasia, necrotizing enterocolitis

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

Premature Rupture of Membranes (PROM)

Definitions
- premature ROM (PROM or amniorrhexis): rupture of membranes prior to labour at any GA
- prolonged ROM: >24 hours elapsed between rupture of membranes and onset of labour
- preterm ROM: ROM occurring before 37 weeks gestation (associated with PTL)
- preterm premature ROM (PPROM): rupture of membranes before 37 weeks AND prior to onset of labour

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

Clinical Features
- history of fluid gush or continued leakage

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

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

Management
- admit for expectant management and monitor vitals q4h, daily BPP and WBC count
- avoid introducing infection with examinations (do NOT do a bimanual exam)
- cultures (cervix for GC, lower vagina for GBS)
- assess fetal lung maturity by L/S ratio of amniotic fluid
  - consider administration of betamethasone valerate (Celestone®) to accelerate maturity if <32 weeks and no evidence of infection
  - consider tocolysis for 48h to permit administration of steroids if PPROM induces labour
- weigh degree of prematurity vs. risk of amnionitis and sepsis by remaining in utero
  - <24 weeks: consider termination (poor outcome due to pulmonary hypoplasia)
  - 24-25 weeks: individual consideration with counselling of parents re: risks to preterm infants
  - 26-34 weeks: expectant management as prematurity complications are significant
  - 34-36 weeks: "grey zone" where risk of death from RDS and neonatal sepsis is the same
  - ≥37 weeks: induction of labour since the risk of death from sepsis is greater than RDS
- if not in labour or labour not indicated, consider antibiotics (controversial)
  - studies show broad spectrum coverage increases the time to onset of labour from PROM by 5-7 days with no increase in maternal or neonatal morbidity or mortality
- deliver urgently if evidence of fetal distress and/or chorioamnionitis

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

A. Complete Breech

B. Frank Breech

C. Footling Breech

Figure 8. Types of Breech Presentation

Breech Presentation

Definition
- fetal buttocks or lower extremity is the presenting part (see Figure 8)
- complete (10%): flexion at hips and knees
- frank (60%): flexion at hips, extension at knees
  - most common type of breech presentation
  - most common breech presentation to be delivered vaginally
- footling (30%): may be single or double with extension at hip(s) and knee(s) so that foot is the presenting part

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

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

Clinical Features
- noted by Leopold's maneuvers (see Figure 2) and U/S
  - PPV of Leopold's maneuvers is only 30%

Management
- external cephalic version (ECV): repositioning of fetus within uterus under U/S guidance
  - overall success rate of 65%
  - criteria: >37 weeks, singleton, unengaged presenting part, reactive NST
  - contraindications: previous T3 bleed, prior classical C/S, previous myomectomy, oligohydramnios, PROM, placenta previa, abnormal U/S, suspected IUGR, hypertension, uteroplacental insufficiency, nuchal cord
  - risks: abruption, cord compression
  - method: tocometry, followed by ultrasound guided transabdominal manipulation of fetus with consistent fetal heart monitoring
  - if patient Rh negative, give Rhogam® prior to procedure
  - good prognostic factors (for a successful version)
    - multiparous, good fluid volume, small baby, skilled obstetrician
- pre- or early labour ultrasound to assess type of breech presentation, fetal growth, estimated weight, altitude of fetal head; if ultrasound unavailable, recommend C-section
- trial of labour and elective C-section should be presented as options with the risks and benefits outlined; obtain informed consent
- contraindications to vaginal breech delivery:
  - cord presentation
  - clinically inadequate maternal pelvis
  - fetal anomaly incompatible with vaginal delivery
- criteria for vaginal breech delivery:
  - frank or complete breech, GA >36 weeks
  - EFW 2500-3800 g based on clinical and U/S assessment (5.5–8.5 lb)
  - fetal head flexed
  - continuous fetal monitoring
  - 2 experienced obstetricians, assistant, and anesthetist present
  - ability to perform emergency C-section within 30 minutes if required
- method for vaginal breech delivery:
  - encourage effective maternal pushing efforts
  - at delivery of after-coming head, assistant must apply suprapubic pressure to flex and engage fetal head
  - delivery can be spontaneous or assisted; avoid fetal traction
  - apply fetal manipulation only after spontaneous delivery to level of umbilicus
- C/S recommended if: the breech has not descended to the perineum in the second stage of labour after 2 hours, in the absence of active pushing, or if vaginal delivery is not imminent after 1 hour of active pushing

Vaginal Delivery of Breech Presentation
SOGG Clinical Practice Guideline, JOGC, June 2009
Objective: To discuss risks and benefits of trial of labour versus planned C-section, with selection criteria, management and delivery techniques for trial of vaginal breech birth.
Evidence: Randomized trials, prospective cohort studies and select cohort studies from Medline search for long-term outcomes and epidemiology of vaginal breech delivery.
Summary: Higher risk of perinatal mortality and short-term neonatal morbidity can be associated with vaginal breech birth as compared to elective C-sections. However, careful case selection (including term singleton breech fetuses and clinically adequate maternal pelvis) and labour management may achieve a similar safety level as elective C-sections (approx. 2 per 1000 births perinatal mortality, approx. 2% short-term neonatal morbidity). Specific protocols for vaginal breech delivery should be followed: continuous fetal heart monitoring, assessment for adequate progress in labour, no induction of labour recommended, emergency C-section available if required and health care providers with requisite skills and experience. Informed consent for the preferred delivery method should be obtained.

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

Vaginal Birth After Caesarean (VBAC)

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

Contraindications
- 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 (9 lb)
- non-vertex presentation or placenta previa
- inadequate facilities or personnel for emergency C/S

Vaginal Delivery After C-Section (VBAC)
- Rate of VBAC ranges from 60-82%
- No significant difference in maternal deaths or hysterectomies between VBAC or C-section
- Uterine rupture more common in VBAC group
- Evidence regarding fetal outcome is lacking

Safety of vaginal birth after caesarean section: a systematic review. *Obstet Gynecol* 2004; 103(3):420-9

Prolonged Pregnancy

Definition
- pregnancy beyond 42 weeks GA

Epidemiology
- 41 weeks GA: up to 27%
- 42 weeks GA: 4-14%

Etiology
- most cases idiopathic
- anencephalic fetus with no pituitary gland
- placental sulfatase deficiency (X-linked recessive condition in 1/2000-1/6000 infants)

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

Management
- GA 40-41 weeks – expectant management
  - no evidence to support induction of labour (IOL) or C/S unless other risk factors for morbidity are present (see prognosis)
- GA >41 weeks – offer induction of labour (IOL) if vaginal delivery is not contraindicated
  - IOL shown to decrease C/S, fetal heart rate changes, meconium staining, macrosomia and death when compared with expectant management
- GA >41 weeks and expectant management elected – serial fetal surveillance:
  - fetal movement count by the mother
  - AFV ± NST (modified BPP)

Prognosis
- if >41 weeks, perinatal mortality 2-3x higher (due to progressive uteroplacental insufficiency)
- morbidity increased with hypertension in pregnancy, DM, abruption, IUGR and multiple gestation

Intrauterine Fetal Death

Definition
- fetal death in utero after 20 weeks GA

Epidemiology
- 1% of pregnancies

DIC: Generalized coagulation and fibrinolysis leading to depletion of coagulation factors

Obstetrical Causes
- Abruption
- PIH
- Fetal demise
- PPH

DIC-specific Bloodwork
- Platelets
- aPTT and PT
- FDP (fibrin degradation products)
- Fibrinogen

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

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

Clinical Features
- decreased perception of fetal movement by mother
- SFH and maternal weight not increasing
- absent fetal heart tones (not diagnostic)
- high maternal serum alpha-fetoprotein (MSAFP)

Management
- diagnosis: absent cardiac activity and fetal movement on U/S required for diagnosis
- determine secondary cause
  - maternal: HbA_{1C}, Kleihauer-Betke, VDRL, ANA, antibody screens, INR/PTT, serum/urine toxicology screens, cervical and vaginal cultures, TORCH screen
  - fetal: chromosomes, cord blood, skin biopsy, genetics evaluation, autopsy
  - placenta: pathology, bacterial cultures

Treatment
- induction of labour
- monitor for maternal coagulopathy [10% risk of disseminated intravascular coagulation (DIC)]
- parental psychological care:
  - referral to grieving support programs
  - encourage partner to stay for support, ask couple to hold the newborn (inform that bruising, facial marks pay be present)
  - make neonatal footprint, take a photo of the newborn, encourage to name the child
  - early follow-up within 2 weeks to assess mental well-being (depression, anxiety)
  - comprehensive discussion within 3 months about final investigation and post-mortem results, help make plans for future pregnancies

Complications of Labour and Delivery

Meconium in Amniotic Fluid

Epidemiology
- usually not present early in labour
- in general, meconium may be present in up to 25% of all labours; usually NOT associated with poor outcome, but extra care is required at time of delivery to avoid aspiration

Maternal Mortality Causes
- Thrombo-embolism
- Cardiac event
- Suicide
- Sepsis
- Ectopic pregnancy
- Hypertension
- Amniotic-fluid embolism
- Hemorrhage

* In Canada (2005), lifetime risk of maternal death is 1 in 11,000

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

Clinical Features
- timing: early (prior to ROM) or late (after ROM with clear fluid)
- consistency
  - thin meconium: light green or yellow, not usually associated with poor outcome
  - thick meconium: dark green or black, pea-soup consistency, associated with lower APGARs and increased risk of meconium aspiration

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

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

Abnormal Progression of Labour (Dystocia)

Definition
- expected patterns of descent of the presenting part and cervical dilatation fail to occur in the appropriate time frame; can occur in all stages of labour (see Figure 9)
- during active phase: >4 hrs of <0.5 cm/hr
- during 2nd phase: >1 hr with no descent during active pushing

Etiology

- **P**ower (leading cause): contractions (hypotonic, incoordinate), inadequate maternal explusive efforts
- **P**assenger: fetal position, attitude, size, anomalies (hydrocephalus)
- **P**assage: pelvic structure (cephalopelvic disproportion), maternal soft tissue factors (tumours, full bladder or rectum, vaginal septum)
- **P**syche: hormones released in response to stress can bring about dystocia
  - psychological and physiological stress should be evaluated as part of the management once dystocia has been diagnosed

The 4 Ps of Dystocia
Power
Passenger
Passage
Psyche

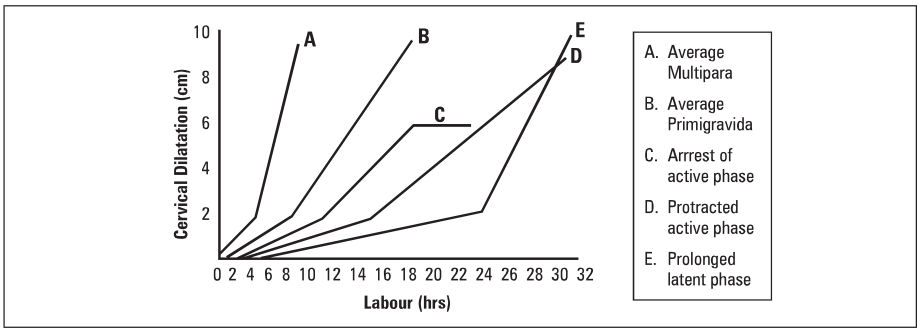


Figure 9. Normal and Abnormal Courses of the First Stage of Labour

The 4 Types of Pelvis
GAAP
Gynecoid (50%) – commonest, obstetrically ideal
Anthropoid (25%)
Android (20%)
Platypelloid (5%)

Arrest Disorder (Curve C)

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

Protraction Disorders (Curve D)

- protraction of dilatation: slope of cervical dilatation <1.2 cm/hr in primigravidas or <1.5 cm/hr in multigravidas
- protraction of descent: a rate of descent of <1.0 cm/hr in primigravidas or 2.0 cm/hr in multigravidas
- treatment: oxytocin augmentation if contractions are inadequate ± amniotomy

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

Prolonged Latent Phase (Curve E)

- ≥20 hrs in primigravidas or ≥14 hrs in multigravidas during which labour has not progressed to the active phase
- most often due to false labour (avoid amniotomy for fear of false labour and increased risk of intrauterine infection)
- premature or excessive use of sedation or analgesia may play a role
- careful search for factors of CPD should be made
- management: oxytocin augmentation if diagnosis of labour is certain, otherwise rest ± sedation

Risks of Dystocia

- inadequate progression of labour is associated with an increased incidence of:
  - maternal stress
  - maternal infection
  - postpartum hemorrhage
  - need for neonatal resuscitation

Umbilical Cord Prolapse

Definition

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

Etiology/Epidemiology

- increased incidence with prematurity/PROM, fetal malpresentation (~50% of cases), low-lying placenta, polyhydramnios, multiple gestation, CPD
- incidence: 0.17-0.63%

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

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

Treatment
- emergency C/S
- O_2 to mother, monitor fetal heart
- alleviate pressure of the presenting part on the cord by placing digit in vagina (maintain this position until C/S)
- keep cord warm and moist by replacing it into the vagina ± applying warm saline soaks
- position mother in Trendelenburg or knee-to-chest position
- if fetal demise or too premature (<22 weeks), allow labour and delivery

Shoulder Dystocia

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

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

Risk Factors
- maternal: obesity, diabetes, multiparity
- fetal: prolonged gestation, macrosomia
- labour
  - prolonged 2nd stage
  - prolonged deceleration phase (8-10 cm)
  - instrumental midpelvic delivery

Approach to the Management of Shoulder Dystocia

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

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

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

Treatment
- goal: to displace anterior shoulder from behind symphysis pubis; follow a stepwise approach of maneuvers until goal achieved (see sidebar)
- other options
  - cleidotomy (deliberate fracture of neonatal clavicle)
  - Zavanelli maneuver: replacement of fetus into uterine cavity and emergent C/S
  - symphysiotomy
  - abdominal incision and shoulder disimpaction via hysterotomy – subsequent vaginal delivery

Prognosis
- 1% risk of long term disability for infant

Uterine Rupture

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

Clinical Features
- prolonged fetal bradycardia – most common presentation
- acute onset abdominal pain
- hyper or hypotonic uterine contractions
- vaginal bleed

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

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

Amniotic Fluid Embolus

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

Etiology/Epidemiology
- rare intrapartum or immediate postpartum complication
- 60-80% maternal mortality rate, accounts for 10% of all maternal deaths
- leading cause of maternal death in induced abortions and miscarriages
- 1/8000-1/80,000 births

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

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

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

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

Chorioamnionitis

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

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

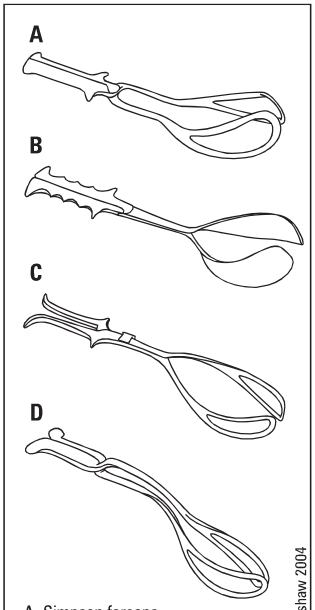
Clinical Features of Chorioamnionitis
Temperature
Tachycardia (maternal or fetal)
Tenderness (uterine)
Foul discharge

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 and purulent cervical discharge

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

Risk Factors for Primary and Subsequent Anal Sphincter Lacerations
Am J Obstet Gynecol 2007; 196(4):344.
Objective: Assess effects of pregnancy, delivery method and parity on risk of primary and secondary anal sphincter laceration in women with 1st vaginal delivery (VD), VBAC or 2nd VD.
Methods: Retrospective cohort study of all deliveries at one hospital from 1995-2002.
Summary: 20,674 live singleton deliveries were included. Women with first VD and VBAC both had OR 5.1 for laceration compared to 2nd VD. Factors that significantly increased risk of laceration for all 3 groups were: forceps and midline episiotomy. 2nd stage of labor >2 h only increased risk for 1st VD. Factors that had no significant increase in risk: infant birth weight >3500g, vacuum delivery. Women with prior anal sphincter laceration are at 3 times increased risk for subsequent sphincter laceration, compared with women with prior vaginal delivery without sphincter laceration.



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

Figure 10. Types of Forceps

Prerequisites for Operative Vaginal Delivery

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

Treatment
- IV antibiotics (ampicillin and gentamicin and anaerobic coverage if C/S)
- expedient delivery regardless of gestational age

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

Operative Obstetrics

Operative Vaginal Delivery

Definition
- forceps or vacuum extraction

Indications
- fetal
  - 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 (e.g. cardiac/cerebrovascular disease)
  - exhaustion, lack of cooperation and excessive analgesia may impair pushing effort

Forceps

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

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

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

Types of Forceps (see Figure 10)
- Simpson forceps for OA presentations
- Kielland (rotational) forceps when rotation of head to OA is recessing
- Piper forceps for breech

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

Vacuum Extraction

- traction instrument used as alternative to forceps delivery; aids maternal pushing

Advantages
- easier to apply
- less anesthesia required
- less maternal soft-tissue injury compared to forceps

Disadvantages
- contraindicated if fetus at risk for coagulation defect
- suitable only for vertex presentations
- maternal pushing required
- contraindicated in preterm delivery
- EFW must be ≥2500 g
- vacuum can lose suction and dislodge, especially if CPD present (note that conversely, on occasion vacuums can be used to overcome CPD and achieve vaginal birth)

Specific Complications of Vacuum Extraction for Fetus
- increased incidence of cephalohematomata and retinal hemorrhages compared to forceps
- subglial hemorrhage, subaponeurotic hemorrhage, soft tissue trauma

Limits for Trial of Vacuum
- Any combination of 3 pulls and/or pop-offs
- 20 minutes with no delivery

Lacerations

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

Episiotomy

Definition
- incision in the perineal body at the time of delivery
  - essentially a controlled second degree laceration
- **midline:** incision through central tendinous portion of perineal body and insertions of superficial transverse perineal and bulbocavernosus muscle
  - better healing but increased risk of deep tear
- **mediolateral:** incision through bulbocavernosus, superficial transverse perineal muscle, and levator ani
  - reduced risk of extensive tear but poorer healing and more pain
- easier to repair

Indications
- to reduce chance of third or fourth degree tear
- to relieve obstruction of the unyielding perineum
- instrumental delivery
- controversy over whether it is preferable to make a cut or let the perineum tear as needed
  - current evidence suggests letting perineum tear and then repair as needed (restricted use)

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

Restrictive vs. Routine Episiotomies with Vaginal Births
Episiotomy for vaginal birth. *Cochrane Database of Systematic Reviews* 2009; Issue 1
Study: This systematic review and meta-analysis of 8 RCTs assessed the effects of restrictive (only done for fetal indications or if severe perineal trauma was judged to be imminent) and routine (liberally done to prevent any tear) use of episiotomy during vaginal birth.
Patients: Of the 2709 patients in the routine episiotomy group, 2035 (75%) women had episiotomies. In the restrictive episiotomy group, 776 (28%) of the 2733 women had episiotomies.
Results: Restrictive episiotomies appear to have less severe perineal trauma (RR 0.67), less suturing (RR 0.71), and fewer healing complications at 7 days (RR 0.69) compared to routine episiotomies. There is no difference for pain measures, dyspareunia, urinary incontinence, and severe vaginal or perineal trauma, but there was an increased risk of anterior perineal trauma (RR 1.84) with restrictive episiotomy. Similar results were obtained when comparing restrictive versus routine mediolateral versus midline episiotomy.
Conclusions: Compared to routine use, restrictive use of episiotomy during vaginal delivery appears to be more beneficial.

Caesarean Delivery

Epidemiology
- incidence 20-25%

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

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

Risks/Complications
- anesthesia
- hemorrhage (average blood loss ~1000 cc)
- infection (UTI, wound, endometritis)
- injury to surrounding structures (bowel, bladder, ureter, uterus)
  - single dose prophylactic antibiotic should be used (e.g. cefazolin 1-2 g)
- thromboembolism
- increased recovery time/hospital stay
- maternal mortality (<0.1%)

Puerperal Complications

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

Postpartum Hemorrhage (PPH)

Definition
- loss of >500 mL of blood at the time of vaginal delivery, or >1000 mL with C/S
- early – within first 24h postpartum
- late – after 24h but within first 6 weeks

Epidemiology
- incidence 5-15%

Etiology (4 T's)

Uterine atony is the most common cause of PPH.

DDx of Early PPH – 4 T's
1. **T**one (atony)
2. **T**issue (retained placenta, clots)
3. **T**rauma (laceration, inversion)
4. **T**hrombin (coagulopathy)

DDx of Late PPH
1. Retained products
2. ± endometritis
3. Sub-involution of uterus

1. Tone
- uterine atony
  - most common cause of PPH
  - avoid by giving oxytocin with delivery of the anterior shoulder or placenta
  - occurs within first 24 hours
- due to:
  - labour (prolonged, precipitous, induced, augmented)
  - uterus (infection, over-distention)
  - placenta (abruption, previa)
  - maternal factors (grand multiparity, gestational HTN)
  - halothane anesthesia

2. Tissue
- retained placental products
- retained blood clots in an atonic uterus
- gestational trophoblastic neoplasia

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

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

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

Management
- ABCs
- 2 large bore IVs and crystalloids
- CBC, coagulation profile, cross and type 4 units pRBCs
- treat underlying cause

Medical Therapy
- oxytocin 20 U/L NS or RL IV continuous infusion
  - in addition can give 10 U intramyometrial (IMM) after delivery of the placenta
- methylergonavine maleate (ergotamine) 0.25 mg IM/IMM q5min up to 1.25 mg; can be given as IV bolus of 0.125 mg (may exacerbate HTN)
- carboprost (Hemabate®) (synthetic PGF-2 alpha analog) 0.25 mg IM/IMM q15 min to max 2 mg (major prostaglandin side effects and contraindicated in cardiovascular, pulmonary, renal and hepatic dysfunction)

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

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

RETAINED PLACENTA

Definition
- placenta undelivered after 30 minutes postpartum

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

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

Clinical Features
- incomplete placenta removed
- risk of postpartum hemorrhage and infection

Investigations
- explore uterus
- assess degree of blood loss

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

UTERINE INVERSION

Definition
- turning inside out (inversion) of the uterus through cervix ± vaginal introitus

Etiology/Epidemiology
- often iatrogenic (excess cord traction with fundal placenta)
- excessive use of uterine tocolytics
- more common in grand multiparous (lax uterine ligaments)
- 1/1500-1/2000 deliveries

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

Management
- urgent management essential, call anesthesia
- ABCs – initiate IV crystalloids
- can use tocolytic drug (e.g. terbutaline) or nitroglycerin IV to relax uterus and aid replacement
- replace uterus without removing placenta
- remove placenta manually and withdraw slowly
- IV oxytocin infusion (only after uterus replaced)
- re-explore uterus
- may require GA ± laparotomy

Postpartum Pyrexia

Etiology of Post-Partum Pyrexia

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

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

Etiology
- **W**ind (atelectasis, pneumonia), **W**ater (UTI), **W**ound (C/S incision or episiotomy site), **W**alking (pelvic thrombophlebitis, DVT), **B**reast (engorgement, mastitis – *S. aureus*), **W**omb (endometritis)

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

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

Risk Factors for Endometritis
C-section, intrapartum chorioamnionitis, prolonged labour, prolonged ROM, multiple vaginal examinations

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

VENOUS THROMBOEMBOLISM
- see OB20

Mastitis

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

Table 21. Lactational versus Non-Lactational Mastitis

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

Postpartum Mood Alterations

POSTPARTUM BLUES
- 85% of new mothers, onset day 3-10; extension of the "normal" hormonal changes and adjustment to a new baby
- self-limited, should resolve by 2 weeks
- manifested by mood lability, depressed affect, increased sensitivity to criticism, tearfulness, fatigue, irritability, poor concentration/despondency

POSTPARTUM DEPRESSION (PPD)
- definition: major depression occurring in a woman within 6 months of childbirth (see Psychiatry, PS10)
- epidemiology: 10-20%, risk of recurrence 50%
- risk factors
  - personal or family history of depression (including PPD)
  - prenatal depression or anxiety
  - stressful life situation
  - poor support system
  - unwanted pregnancy
  - colicky or sick infant
- clinical features: suspect if the "blues" last beyond 2 weeks, or if the symptoms in the first two weeks are severe (e.g. extreme disinterest in the baby, suicidal or homicidal/infanticide ideation)
- assessment: Edinburgh Postnatal Depression Scale or other
- treatment: antidepressants, psychotherapy, supportive care, ECT if refractory
- prognosis: interferes with bonding and attachment between mother and baby so it can have long term effects

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

Postpartum Care

Postpartum Office Visit at 6 Weeks

Care of Baby
- assess weight, feeding, immunization
- encourage breastfeeding if no contraindications

Care of Mother (The 10 Bs)
- **B**e careful: do not use douches or tampons for 4-6 weeks post-delivery
- **B**e fit: encourage gradual increases in walking, Kegel exercises
- **B**irth control: assess for use of contraceptives; breastfeeding is NOT an effective method of birth control
- **B**ladder: assess for urinary incontinence, maintain high fluid intake
- **B**leeding: (see *Lacerations,* OB47), 300 μg of RhIG should be given if Rh+ fetus and Rh– mother or extensive bleeding at delivery
- **B**lood pressure: especially if gestational HTN
- **B**lood tests: glucose, CBC (for anemia as sign of hematomas, retained placenta)
- **B**lues: see *Postpartum Mood Alterations,* OB51
- **B**owel: fluids and high-fibre foods, bulk laxatives; for hemorrhoids/perineal tenderness: pain meds, doughnut cushion, Sitz baths, ice compresses
- **B**reast and pelvic exam: watch for *Staphyloccoccal* or *Streptococcal* mastitis/abscess, ± Pap smear at 6 weeks

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

The acronym "**BUBBLES**" for what to ask about when rounding on postpartum care. Modify this for C/S or vaginal delivery
Baby care and breastfeeding (latch, amount)
Uterus – firm or boggy?
Bladder function – Voiding well? Dysuria?
Bowel function – Passing gas or stool? Constipated?
Lochia or discharge – Any blood?
Episiotomy/laceration/incision – Pain controlled?
Symptoms of VTE – Dyspnea? Calf pain?

Breastfeeding Problems
- inadequate milk: consider domperidone
- breast engorgement: cool compress, manual expression/pumping
- nipple pain: clean milk off nipple after feeds, moisture cream, topical steroid if needed
- mastitis: treat promptly (see OB50)
- inverted nipples: makes feeding difficult
- maternal medications: may require pediatric consultation (see OB54)

Bladder Dysfunction
- pelvic floor prolapse can occur after vaginal delivery
- stress or urge urinary incontinence common
- increased risk with instrumental delivery or prolonged second stage
- conservative management: pelvic floor retraining with Kegel exercises, vaginal cones or pessaries, lifestyle modifications (e.g. limit fluid, caffeine intake)
- surgical management: minimally invasive procedures (tension-free vaginal tape, transobturator tape, midurethral sling) (see Gynecology, GY34)

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

Drug and Food Safety During Pregnancy and Breastfeeding

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

Antibiotics

Drug Resources during Pregnancy and Breastfeeding
- Motherisk at the Hospital for Sick Children in Toronto: www.motherisk.org
- Hale, T. Medications and Mothers' Milk, 11th Edition. Pharmasoft Publishing, 2004.

- safest: ampicillin, cephalosporins
- erythromycin: maternal liver damage (acute fatty liver)
  - used only if contraindication to penicillin use
- tetracyclines: stain infant's teeth, may affect long bone development
- sulpha drugs: anti-folate properties, therefore theoretical risk in T1; risk of kernicterus in T3
- metronidazole: anti-metabolite, therefore theoretical risk in T1
- chloramphenicol: grey baby syndrome (fetal circulatory collapse 2° to toxic accumulation)
- fluoroquinolones: risk of cartilage damage (in dog and rat studies)

Other Medications and Substances

- analgesics: acetaminophen preferable to ASA or ibuprofen

Documented adverse effects, contraindicated:
- ACE inhibitors: fetal renal defects, IUGR, oligohydramnios
- tetracycline: see above
- retinoids (e.g. Accutane®): CNS, craniofacial, cardiac, and thymic anomalies
- DES (and other estrogenic or androgenic compounds): vaginal adenosis, adenocarcinoma, uterine malformation in females exposed to DES in utero
- misoprostol: Mobius syndrome (congenital facial paralysis with or without limb defects)

Documented adverse effects, weigh benefits vs. risks and consider medication change:
- anticonvulsants
  - phenytoin associated with fetal hydantoin syndrome in 5-10% (IUGR, mental retardation, facial dysmorphogenesis, congenital anomalies)
  - valproate associated with oNTD in 1%
  - carbamazepine associated with oNTD in 1-2%
  - generally recommended to remain on the lowest dose anticonvulsant appropriate for their condition
- lithium: Ebstein's cardiac anomaly, goitre, hyponatremia
- warfarin: increased incidence of spontaneous abortion, stillbirth, prematurity, IUGR
  - fetal warfarin syndrome (nasal hypoplasia, epiphyseal stippling, optic atrophy, mental retardation, intracranial hemorrhage)

Substances
- alcohol: increased incidence of abortion and stillbirth, congenital anomalies
  - fetal alcohol syndrome (growth retardation, CNS involvement and facial anomalies)
- cigarette smoke: decreased birth weight, placenta previa/abruption, spontaneous abortion, preterm labour, stillbirth
- cocaine: microcephaly, growth retardation, prematurity, MR

Immunizations

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

Postpartum
- rubella vaccine for all non-immune mothers
- hepatitis B vaccine should be given to infant within 12h of birth if maternal status unknown or positive – follow-up doses at 1 and 6 months

Food

Caffeine
- diuretic and stimulant properties
- readily crosses placenta
- possible risk for miscarriage and fetal growth retardation at high doses (>200-300 mg/day); note some of this presumed risk may be due to confounders, such as cigarette smoking
- based on a meta-analysis, consumption should be limited to no more than 150 mg per day from all sources during pregnancy and lactation

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

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

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

Food Borne Illnesses
- microbiological contamination of food may occur through cross-contamination and/or improper food handling
  - listeriosis (*Listeria monocytogenes*) and toxoplasmosis (*Toxoplasma gondii*) are of concern during pregnancy
  - avoid consumption of raw meats, fish, poultry, raw eggs, and unpasteurized dairy products
  - wash all raw fruit and vegetables thoroughly
  - avoid soft cheeses and pates as they may be sources of *Listeria*
- chemical contamination of food
  - current guideline for mercury of 0.5 ppm in fish is considered protective for the general population, including pregnant women
  - Health Canada advises pregnant women to limit consumption of top predator fish such as shark, swordfish and fresh/frozen tuna (not canned tuna) to one meal per month

Radiation

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

- ionizing radiation exposure is considered teratogenic at high doses
  - if indicated for maternal health, should be done
- imaging not involving direct abdominal/pelvic high dosage is not associated with adverse effects
  - higher dosage to fetus: plain x-ray of lumbar spine/abdomen/pelvis, barium enema, CT abdomen, pelvis, lumbar spine
- most investigations involve minimal radiation exposure (see Table 22)
- radioactive isotopes of iodine are contraindicated
- no known adverse effects from U/S or MRI

Table 22. Approximate Fetal Doses from Common Diagnostic Procedures

Examination	Estimated Fetal Dose (rad)	Number of Exams Safe in Pregnancy
Plain Film		
Abdomen	0.245	20
Pelvis	0.040	125
Lumbar spine	0.359	13
Thoracic spine	0.009	555
Chest (2 views)	0.00007	71 429
CT		
Abdomen (10 slices)	2.600	1
Pelvis (1 slice with scout film)	0.250	20
Lumbar spine (5 slices)	3.500	1
Chest	0.2-0.6	20

Adapted from Valentin, 2000.

Breastfeeding and Drugs

Breastfeeding: Contraindicated Drugs

BREAST
Bromocriptine/Benzodiazepines
Radioactive isotopes/Rizatriptan
Ergotamine/Ethosuximide
Amiodarone/Amphetamines
Stimulant laxatives/Sex hormones
Tetracycline/Tretinoin

- safe
  - penicillins, aminoglycosides, cephalosporins
  - oral contraceptive use (low dose) – OCP will decrease quantity but not affect composition of breast milk
  - medroxyprogesterone acetate
- avoid
  - chloramphenicol (bone marrow suppression)
  - metronidazole (mutagenic in vitro)
  - sulphonamides (hemolysis with G6PD deficiency)
  - nitrofurantoin (hemolysis with G6PD deficiency)
  - tetracycline
  - lithium
  - anti-neoplastics and immunosuppressants
  - psychotropic drugs (relative)

Common Medications

Table 23. Common Medications

Drug Name (Brand Name)	Dosing Schedule	Indications/Comments
betamethasone valerate (Celestone®)	12 mg IM q24h x 2 doses	Enhancement of fetal pulmonary maturity for PTL
carboprost (Hemabate®)	0.25 mg IM/IMM q15min; max 2 mg	Treatment of uterine atony
dexamethasone	6 mg IM q12h x 4 doses	Enhancement of fetal pulmonary maturity for PTL
dinoprostone (Cervidil®: PGE_2 impregnated thread)	10 mg PV (remove after 12h) max of 3 doses	Induction of labour Advantage: can remove if uterine hyperstimulation
doxylamine succinate (Diclectin®)	2 tabs qhs + 1 tab qAM + 1 tab qPM max of 8 tabs/day	Each tablet contains 10 mg doxylamine succinate with vitamin B_6 Used for hyperemesis gravidarum
folic acid	0.4-1 mg PO OD x 1-3 mo preconception and T1 5 mg PO OD with past Hx of NTD	Prevention of oNTD
methotrexate	50 mg/m^2 IM or 50 mg po x 1 dose	For ectopic pregnancy or medical abortion
methylergonavine maleate (Ergotamine®)	0.25 mg IM/IMM q5min up to 1.25 mg or IV bolus 0.125 mg	Treatment of uterine atony
misoprostol (Cytotec®)	800-1000 μg PR x 1 dose 400 μg PO x 1 dose or 800 μg PV x 1 dose, 3 to 7 days after methotrexate	For treatment of PPH For medical abortion Also used for NSAID-induced ulcers (warn patients of contraindications)
oxytocin (Pitocin®)	0.5-2.0 mU/min IV, or 10 U/L N/S incr. by 1-2 mU/min q20-60min max of 36-48 mU/min 10 U IM at delivery of anterior shoulder and of placenta 20 U/L NS or RL IV continuous infusion	Augmentation of labour (also induction of labour) Prevention of uterine atony Treatment of uterine atony
PGE_2 gel (Prostin® gel)	0.5 mg PV q6-12h; max of 3 doses	Induction of labour
Rh IgG (Rhogam®)	300 μg IM x 1 dose	Given to Rh negative women • Routinely at 28 wks GA • Within 72 hrs of birth of Rh +ve fetus positive • Positive Kleihauer-Betke test • With any invasive procedure in pregnancy • Ectopic pregnancy • Antepartum hemorrhage • Miscarriage or TA (dose: 50 μg IM only)

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

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

References

American College of Obstetricians and Gynecologists www.acog.org
The Society of Obstetricians and Gynaecologists of Canada www.sogc.org
ABC of labour care: Care of the newborn in the delivery room. BMJ 1999; 318: 1403-1406.
ABC of labour care: Obstetric emergencies. BMJ 1999; 318: 1342-1345.
ABC of labour care: Operative delivery. BMJ 1999; 318: 1260-1264.
ABC of labour care: Induction. BMJ 1999; 318: 995-998.
ABC of labour care: Labour in special circumstances. BMJ 1999; 318: 1124-1127.
ABC of labour care: Physiology and management of normal labour. BMJ 1999; 318: 793-796.
ABC of labour care: Place of birth. BMJ 1999; 318: 721-723.
ABC of labour care: Preterm labour and premature rupture of membranes. BMJ 1999; 318: 1059-1062.
ABC of labour care: Relief of pain. BMJ 1999; 318: 927-930.
ABC of labour care: Unusual presentations and positions and multiple pregnancy. BMJ 1999; 318: 1192-1194.
Alfirevic Z. (2002). Oral misoprostol for induction of labour (Cochrane Review). The Cochrane Library, Issue 2.
Antenatal Corticosteroid Therapy for Fetal Maturation. SOGC Clinical Practice Guidelines Policy Statement, December 1995: 53.
Baskett, T. Essential Management of Obstetric Emergencies. 3rd ed. Clinical Press, Bristol, 1999.
Bastian LA, Piscitelli JT. Is this patient pregnant? Can you reliably rule in or rule out early pregnancy by clinical examination? JAMA. 1997 Aug 20; 278(7): 586-91. Review
Berghella V, et al. (2004). Cerclage for prevention of preterm birth in women with a short cervix found on transvaginal examination: A randomized trial. Amer J Obs Gyn. 191:1311-7.
Boucher, M. Mode of Delivery for Pregnant Women infected by the Human Immunodeficiency Virus. SOGC Clinical Practice Guidelines. April 2001: 101.
Boucher, M. and Gruslin, A. The Reproductive Care of Women living with Hepatitis C Infection. SOGC Clinical Practice Guidelines. October 2000: 96.
Bricker L, Luckas M. (2002). Amniotomy alone for induction of labour (Cochrane Review). The Cochrane Library, Issue 2.
Carol, Blenning. An Approach to the Postpartum Office Visit. American Family Physician. Dec 2005, 72(12); 2491 – 2496.
Chodirker et al. Canadian Guidelines for Prenatal Diagnosis Part 1 & Part 2. SOGC Clinical Practice Guidelines. July 2001 and June 2000. No 105.
Chyu JK, Strassner HT. Prostaglandin E2 for cervical ripening: a randomized comparison of cervidil vs. prepidil. AMJ Obstet Gynecol 1997. 177: 606-11.
Crane, J. (2001). Induction of labour at term. SOGC Clinical Practice Guideline, 107:1-12.
Fetal health Surveillance in Labour – Parts 1 and 2. SOGC Clinical Practical Guideline. January 1996, 45.
Ford HB, Schust DJ. (2009) Recurrent Pregnancy Loss: Etiology, Diagnosis, and Therapy. Rev Obstet Gynecol. 2009 Spring; 2(2): 76–83.
Gagnon A, and Wilson R. Obstetrical complications associated with abnormal maternal serum markers analytes. SOGC Clinical Practice Guidelines. Oct 2008, No 216; 918-932.
Guidelines for exercise in pregnancy. SOGC Clinical Practical Guidelines. Committee Opinion. June 2003.
Guidelines for the Management of Nausea and Vomiting in Pregnancy. SOGC Clinical Practice Guidelines Committee Opinion. November 1995: 12.
Goldenberg et al. Epidemiology and causes of preterm birth. The Lancet. Jan 2008, Vol 371; 75 – 84.
Guise JM, Berlin M, McDonagh M, Osterweil P, Chan B, Helfand M. Safety of vaginal birth after caesarean: a systematic review. Obstet Gynec. 2004 Mar; 103(3):420-9.
Hannah, M. Post-term Pregnancy. SOGC Clinical Practice Guidelines Committee Opinion. March 1997: 15.
Healthy Beginnings: Guidelines for Care during Pregnancy and Childbirth. SOGC Clinical Practice Guidelines Policy Statement. December 1998: 71.
Hennessey MH, Rayburn WF, Stewart JD, Likes EC. Preeclampsia and induction of labour: a randomized comparision of prostaglandin E2 as an intracervical gel, with oxytocin immediately, or as a sustained – release vaginal insert. AMJ Obstet Gynecol. 1998. 179(5): 1204-9.
Hod M, Bar J, Peled Y, Fried S, Katz I, Itzhak M, Ashkenazi S, Schindel B, Ben-Rafael Z. Antepartum management protocol. Timing and mode of delivery in gestational diabetes. Obstet Gynecol. 2009 Jan;113(1):206-17.
Howarth GR, Botha DJ. (2002). Amniotomy plus intravenous oxytocin for induction of labour (Cochrane Review). The Cochrane Library, Issue 2.
Kelly AJ, Tan B. (2002). Intravenous oxytocin alone for cervical ripening and induction of labour (Cochrane Review). The Cochrane Library, Issue 2.
Kent, N. Prevention and Treatment of Venous Thromboembolism (VTE) in Obstetrics. SOGC Clinical Practice Guidelines. September 2000: 95.
Koren, G. Motherisk Update: Caffeine Diring Pregnancy? Canadian Family Physician. April 2000. 46 : 801-3.
Kotaska et al. Vaginal Delivery of Breech Presentation. SOGC Clinical Practice Guidelines. June 2009. No. 226; 557 – 566.
Langlois S, Ford J, and Chitayat D. Carrier Screening for Thalassemia and hemoglobinopathies in Canada. SOGC Technical Update. Oct 2008, No 217; 950-959.
Langlois S, Wilson R. Carrier Screening for Genetic Disorders in Individuals of Ashkenazi Jewish Descent. SOGC Clinical Practice Guidelines. Apr 2006, No 177; 324-332.
Ling, F. and Duff, P. Obstetrics and Gynecology, Principles for practice. USA: McGraw-Hill Companies, 2002.
Luckas M, Bricker L. (2000). Intravenous prostaglandin for induction of labour (Cochrane Review). The Cochrane Library, Issue 2.
Meltwer, S., Leiter, L., Daneman, D., Gerstein, HC., Lau, D., Ludwig S., Yale, JF., Zinman, B., Lillie, D. 1998 Clinical Practice Guidelines for the Management of Diabetes in Canada. CMAJ. 1998, 159 (8 suppl): S1-29.
Menezes EV, Yakoob MY, Soomro T, Haws RA, Darmstadt GL, Bhutta ZA. (2009) Reducing stillbirths: prevention and management of medical disorders and infections during pregnancy. BMC Pregnancy Childbirth. 2009; 9(Suppl 1): S4.
Midmer D, Biringer A, Carroll JC, Reid AJ, Wilson L, Stewart D, Tate M, Chalmers B. A Reference Guide for Providers: The ALPHA Form – Antenatal Psychosocial Health Assessment Form, 2nd Edition. Toronto: University of Toronto, Faculty of Medicine, Department of Family and Community Medicine, 1996.
Ministry of Health and Long Term Care and Canadian Medical Association. Antenatal Record 1. Ontario.
Ministry of Health and Long Term Care and Canadian Medical Association. Antenatal Record 2. Ontario.
Money, D and Dobson, S. The prevention of early-onset group B streptococcal disease. SOGC Clinical Practice Guidelines. September 2004.
Lowder JL, Burrows LJ, Krohn MA, Weber AM. Risk factors for primary and subsequent anal sphincter lacerations: a comparison of cohorts by parity and prior mode of delivery. Am J Obstet Gynecol. 2007 Apr;196(4):344.e1-5.
Mount Sinai Hospital. First Trimester Combined Screening Program. 2001.
North York General Hospital Genetics Program. Integrated Prenatal Screening. 1999.
Ottinyer WS, Menara MK, Brost BC. A randomized control trial of prostaglandin E2 intracervical gel and a slow release vaginal for preinduction cervical ripening. AMJ Obstet Gynecol. 1998. 179(2): 349-53.
Schuurmans, N., MacKinnon, C., Lane C., Etches, D. Prevention and Management of Postpartum Hemorrhage. SOGC Clinical Practice Guidelines. April 2000: 88.
SOGC. Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline, Sept. 2007.
SOGC. Conservative Management of Urinary Incontinence, No. 186. Dec 2006.
SOGC. Midurethral Minimally Invasive Sling Procedures for Stress Urinary Incontinence. No 213. Aug 2008.
Statistics Canada. 2005. Induced Abortion Statistics, 82-223-XWE, page 16 of 32.
Stewart, D. A broader context for maternal mortality. CMAJ, Jan 31 2006, 174 (3); 302 – 303.
Stewart JD, Rayburn WF, Farmer KC, Ciles EM, Schipaul AH Jr., Stanley JR. Effectiveness of prostaglandin E2 intracervical gel (prepidil) with immediate oxytocin vs. vaginal insert (cervidil) for induction of labour. AMJ Obstet Gynecol 1998. 179: 1175-80.
Summers A, Langlois S, Wyatt P, Wilson D. Prenatal Screening for Fetal Aneuploidy. SOGC Clinical Practice Guidelines. Feb 2007, No 187; 146-161.
Van den Hof, M. and Crane, J. Ultrasound Cervical Assessment in Predicting Preterm Birth. SOGC Clinical Practice Guidelines. May 2001: 102.