OBSTETRICS

Dr. P. Bernstein Denise Bateman and Kelly Grant, editors Lopita Banerjee, associate editor

NORMAL OBSTETRICS	NORMAL LABOUR AND DELIVERY 33 The Fetus The Cervix Definition of Labour
PRENATAL CARE	Four Stages of Labour The Cardinal Movements of Fetus During Delivery
Gestation-Dependent Management Prenatal Diagnosis	ABNORMAL LABOUR
FETAL MONITORING	Abnormal Progress of Labour Shoulder Dystocia Breech Presentation
MULTIPLE GESTATION. 11 Background Classification Complications	Vaginal Birth After Cesarean (VBAC) Uterine Rupture Amniotic Fluid Embolus
Management MEDICAL CONDITIONS IN PREGNANCY 12 Urinary Tract Infection	OPERATIVE OBSTETRICS 41 Indications for Operative Vaginal Delivery Forceps
Iron Deficiency Anemia Folate Deficiency Anemia Diabetes Mellitus	Vacuum Extraction Lacerations
Gestational Diabetes Hypertensive Disorders of Pregnancy Hyperemesis Gravidarum	Episiotomy Cesarean Delivery
Isoimmunization Infections During Pregnancy	OBSTETRICAL ANESTHESIA 42 Pain Pathways During Labour Analgesia
SURGICAL CONDITIONS IN PREGNANCY 22 Acute Surgical Conditions Nonemergent Surgical Conditions	Anesthesia NORMAL PUERPERIUM
ANTENATAL HEMORRHAGE	Definition Post-Delivery Examination Breast
Third Trimester Bleeding Placenta Previa Abruptio Placentae	Uterus Lochia
Vasa Previa GROWTH DISCREPANCIES	PUERPERAL COMPLICATIONS
Intra-Uterine Growth Restriction Macrosomia Polyhydramnios Oligohydramnios	Postpartum Pyrexia Postpartum Hemorrhage Postpartum Mood Alterations
ANTENATAL COMPLICATIONS	DRUGS CONTRAINDICATED IN
Rupture of Membranes Umbilical Cord Prolapse Chorioamnionitis Post-Date Pregnancy Intrauterine Fetal Death	Immunizations Breast Feeding and Drugs

DEFINITIONS

Gravidity ☐ the total number of pregnancies of any gestation • includes abortions, ectopic pregnancies, and hydatidiform moles • twins count as one pregnancy
Parity ☐ the number of pregnancies that have been carried to > 20 weeks • twins count as one • grand multiparity is parity of 4 or more ☐ four digit number (T P A L) • 1st digit: number of term infants delivered (> 37 weeks) • 2nd digit: number of premature infants delivered (20 to 37 weeks) • 3rd digit: number of abortions (< 20 weeks)
• 4th digit: number of living children Trimesters T1: 0 to 12 weeks T2: 12 to 28 weeks T3: 28 to 40 weeks normal pregnancy term: 37 to 42 weeks
Abortion ☐ loss of intrauterine pregnancy prior to viability of fetus • < 20 weeks and/or < 500 g fetal weight • includes induced ("therapeutic") and spontaneous ("miscarriage")
Stillbirth ☐ loss of intrauterine pregnancy after 20 weeks and/or > 500 g fetal weight
Stillbirth Rate ☐ the annual number of stillbirths per 1000 total births
Perinatal Mortality Rate ☐ the annual number of stillbirths and early neonatal deaths (in the first seven days of life) per 1000 total births ☐ causes • prematurity • congenital anomalies
Neonatal Mortality Rate the annual number of deaths of liveborn infants within 28 days per 1000 live births
Infant Mortality Rate ☐ the annual number of deaths of liveborn infants in the first year of life per 1000 live births (includes neonatal mortality)
Maternal Mortality Rate ☐ the annual number of deaths of women while pregnant or within 90 days of pregnancy per 100 000 live births • direct: from obstetrical causes such as ectopic, PIH, PPH, infection, PE • indirect: from pre-existing illness or by accident
Birth Rate ☐ the annual number of live births per 1000 population
Fertility Rate ☐ the annual number of live births per 1000 women aged 15-44 years

DIAGNOSIS OF PREGNANCY

Symptoms amenorrhea nausea and/or vomiting breast tenderness urinary frequency fatigue
Signs softening of the cervix (Goodell sign): 4-6 weeks bluish discoloration of the cervix and vagina due to engorgement of pelvic vasculature (Chadwick sign): 6 weeks uterine enlargement softening of the isthmus (Hegar sign): 6-8 weeks
Investigations □ ßhCG • positive in the serum at 9 days post-conception • positive in the urine 28 days after LMP □ transvaginal ultrasound • 5 weeks: gestational sac visible (ßhCG = 1200 -1500 mIU/mL) • 6 weeks: fetal pole seen • 7-8 weeks: fetal heart tones visible □ transabdominal U/S • intrauterine pregnancy visible at ßhCG = 5000 mIU/mL
MATERNAL PHYSIOLOGY
General Principles progesterone induces relaxation of smooth muscle, among other effects physiologic changes are more pronounced in multiple gestations
Cardiovascular System ☐ increased cardiac output, heart rate, and blood volume (hyperdynamic circulation) ☐ decreased blood pressure (especially diastolic, maximal in T2) due to decreased peripheral vascular resistance ☐ blood flow to the uterus, kidneys, breasts, and skin increases with gestational age ☐ enlarging uterus compresses IVC and pelvic veins leading to risk of hypotension (by decreasing venous return) as well as varicose veins, hemorrhoids and leg edema (because of increased venous pressure)
Hematologic System □ apparent decrease in hemoglobin and hematocrit due to hemodilution • plasma volume increases more than RBC mass □ increased risk of DVT and PE secondary to hypercoagulable state • increase in factors I, VII, VIII, IX, X, XIII • decrease in factors XI, XIII and antithrombin III activity • venous stasis from uterine compression of veins □ increased leukocyte count but impaired function • 5000 to 12 000/uL in pregnancy • up to 25 000/uL in labour/postpartum • often have improvement in autoimmune conditions
Respiratory System increased oxygen consumption by 20% increased sensitivity to carbon dioxide (progesterone effect on respiratory centre) results in hyperventilation and respiratory alkalosis compensated by increased renal excretion of serum bicarbonate 50% increase in minute ventilation decreased total lung capacity, FRC and residual volume vital capacity unchanged increased tidal volume by 35-50% increased alveolar ventilation by 65%

Gastrointestinal System ☐ increased gastroesophageal reflux • decreased sphincter tone • delayed gastric emptying • increased intraabdominal pressure ☐ increased stasis in gallbladder ☐ decreased GI motility and constipation ☐ upward displacement of appendix • appendicitis may have atypical presentation in pregnancy ☐ hemorrhoids caused by constipation and elevated venous pressure
Genitourinary System ☐ increased GFR 50% (therefore decreased BUN and serum creatinine) but no change in urine output because of increased reabsorption in tubules ☐ glycosuria can be physiologic; with increase in GFR the threshold for glucose reabsorption can be surpassed ☐ increased urinary frequency ☐ physiologic dilatation of ureters and renal pelvis (R > L) due to progesterone-induced smooth muscle relaxation and uterine enlargement ☐ increased incidence of UTI and pyelonephritis due to stasis • asymptomatic bacteriuria more likely to become a clinically significant infection (i.e. pyelonephritis) in pregnancy and therefore should be treated
 Endocrine System □ estrogen • main estrogen is estradiol (E3) • production involves an intricate pathway, requiring maternal, placental and fetal contributions • sudden decline may indicate fetal compromise
 progesterone produced by corpus luteum during first 7 weeks, thereafter synthesized by the placenta maintains the endometrium absolutely necessary for continuation of pregnancy human chorionic gonadotropin (hCG) produced by placental trophoblastic cells peptide hormone composed of two subunits: alpha (common to all glycoproteins) and beta (specific to hCG) has LH-like actions: maintains the corpus luteum serum ßhCG positive 8-9 days after ovulation plasma levels double every 1-2 days, peak (8-10 weeks) and then fall to a plateau until delivery rule of 10's
 relaxin produced by the corpus luteum/ovary relaxes symphysis pubis and other pelvic joints helps soften and dilate the cervix inhibits uterine contraction

 Ca⁺⁺ metabolism total maternal Ca⁺⁺ decreased due to decreased albumin free ionized (i.e. active) proportion remains the same due to increased PTH which results in increased bone resorption and gut absorption bone turnover increased but no loss of bone density because estrogen counteracts the PTH effect by inhibiting resorption
Neurologic System ☐ carpal tunnel syndrome and Bell's palsy more common
Integumentary System □ pigmentation changes (fade after delivery) • increased pigmentation of perineum and areola • chloasma (pigmentation changes under eyes and bridge of nose) • linea nigra (midline abdominal pigmentation) • spider angiomas • palmar erythema □ striae gravidarum (fade but seldom disappear)

PRENATAL CARE

PRECONCEPTION COUNSELLING ☐ folic acid to prevent NTD's (0.4 to 1 mg daily in all women, 4 mg if past NTD) ☐ genetic history and risk factors ☐ modify medications, alcohol, smoking ☐ rubella immunity ☐ proper nutrition ☐ use of prenatal vitamin and iron supplementation ☐ impact on family and occupation (maternity/paternity leave) ☐ domestic violence (50% of domestic violence begins in pregnancy) ☐ depression / mental health
INITIAL VISIT ☐ generally after 12 weeks
History ☐ determine GA by dates from the first day of the LMP (if regular periods and sure dates) ☐ if LMP unsure, get a dating ultrasound ☐ determine EDC using the Naegele Rule • first day of LMP + 7 days - 3 months • e.g. LMP = 1 Apr. 1999, EDC = 8 Jan. 2000 • modify appropriately for longer or shorter cycles ☐ obtain obstetric history of all previous pregnancies (GTPAL) ☐ obtain relevant medical, social, and family history ☐ counselling (see Preconception Counselling Section) • drug use, alcohol consumption, smoking • breastfeeding
Physical □ complete physical exam □ baseline BP (very important for relating subsequent changes) □ baseline weight □ pelvic exam
 Investigations □ bloodwork • CBC, blood group and type, Rh antibodies • rubella titre, VDRL, HBsAg routine; HIV serology should be offered to all
 R&M, C&S asymptomatic bacteriuria in 5% of pregnant women if untreated 25-30% will get a UTI in pregnancy (increased risk of preterm labour)

 pelvic exam Pap smear (if none within 6 months), culture for GC and chlamydia
SUBSEQUENT VISITS ☐ for low-risk, uncomplicated pregnancy • q monthly until 28 weeks • q 2 weeks from 28 to 36 weeks • q weekly from 36 weeks until delivery
With Every Visit ☐ estimate GA ☐ urine dip for glucose and protein
weight gainexpect gain of roughly 1 lb/month in first half of
 pregnancy, 1 lb/week in second half of pregnancy average weight gain 25-35 lbs with only 40% of weight gain products of conception
□ blood pressure □ symphyseal-fundal height measurement: SFH should be within 2 cm of gestational age in weeks between 20 and 37 weeks, i.e. SFH = 20 cm @
20 weeks • 12 weeks fundus @ pubic symphysis • 20 weeks @ umbilicus
• 37 weeks @ sternum ☐ differential diagnosis of uterus incorrect size for dates (accurate dates essential)
 maternal—> DM maternal-fetal—> poly/oligo-hydramnios, multiple gestation
 fetal—> abnormal karyotype, ľUGR, fetal anomaly examination of abdomen for lie, position and presentation (Leopold maneuvers) in T3
☐ fetal heart tones starting at ~ 12 weeks if using doppler U/S

Table 1. Gestation-Depe	
Gestational Age (weeks)	Management Issues
10-12	CVS
15-16 or up to term	Amniocentesis
16	MSS
16-18	U/S for dates and structural assessment
	Quickening (fetal movement felt by mother)
26 -28	50 g oral glucose challenge test (OGCT)
28	Repeat CBC
	Rhogam to Rh negative woman
36	Rh antibody screen if indicated; GBS screen
6 (postpartum)	Follow-up visit
4 1	discuss contraception
	 breast exam and pelvic exam, Pap
	depression/mental health

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

 differential diagnosis of low MSAFP GTN incorrect GA missed abortion
 chromosomal anomalies (e.g. Trisomy 18, 21) 80% of Down's babies born to women under 35 years, so MSS is a valuable screening tool MSS has a 6-7% false positive rate
detection rate of Trisomy 21 with the 3 markers is 2-3 times higher than with MSAFP alone, however will still miss 20-30% of Trisomy 21 pregnancies in older women and will not reliably detect other chromosomal anomalies that occur more frequently in older women so amniocentesis should still be offered to high risk women
Group B Streptococcus ☐ danger of vertical transmission (neonatal sepsis, meningitis or pneumonia) ☐ indications for antibiotic prophylaxis (intrapartum ampicillin or clindamycin in pen-allergic - guidelines controversial) • positive GBS screen based on vaginal cultures taken at 36-38 weeks or
 GBS status unknown and one of the following risk factors previous GBS bacteriuria even if treated previous infant with GBS infection preterm labour PROM > 12 hours
 maternal intrapartum temperature > 37.7°C fetal tachycardia
PRENATAL DIAGNOSIS
Indications maternal age > 35 (increased risk of some chromosomal anomalies) abnormal MSS or ultrasound past history of pregnancy with chromosomal anomaly or genetic disease either parent a known carrier of a genetic disorder or balanced translocation
 three or more miscarriages family history of chromosomal anomaly, genetic disorder, birth defect, or undiagnosed mental retardation consanguinity
Amniocentesis ultrasound-guided transabdominal extraction of amniotic fluid at 15-16 weeks gestation to identify genetic problems such as trisomies during 3rd trimester for assessment of fetal lung maturity
 L/S ratio: if > 2:1, fetal lungs are mature enough that RDS less likely to occur used to quantitate amniotic fluid bilirubin concentration in Rh-isoimmunized pregnancies
 advantages screen for NTD (acetyl cholinesterase and amniotic AFP)
 more accurate genetic testing disadvantages 0.5% risk of spontaneous abortion populate take 10.14 days EISH geneticables in 79 hours
 results take 10-14 days; FISH available in 72 hours in women over 35 years, the risk of chromosomal anomaly (1/180) is greater than the increased risk of miscarriage from the procedure, so it is offered routinely
Chorionic Villus Sampling (CVS) ☐ needle through abdomen or catheter through cervix at 10-12 weeks ☐ advantages
 enables pregnancy to be terminated earlier more rapid karyotyping, DNA tests, chromosome status, biochemical assay (results in 48 hours; do not have to wait for culture) increasing availability of probes to allow diagnosis of genetic abnormalities (i.e. FISH)
 disadvantages 1-2% risk of spontaneous abortion
 does not screen for neural tube defects (NTD) risk of limb injury poor test because of genetic mosaicism

ANTENATAL MONITORING
Fetal Movements □ assessed by • maternal perception (quickening) • choose a time when baby is normally active to count movements • if < 6 movements in 2 hours, notify MD • 10 movements in 12 hour period is lower limit of normal (32 weeks and over) • palpation • U/S
Ultrasound □ routinely done at 16-20 weeks to assess fetal growth and anatomy □ earlier or subsequent U/S only when medically indicated • confirm intrauterine pregnancy • identify multiple pregnancy • past history of early fetal losses • bleeding or other complications • measure fetal growth and identify IUGR • placental localization • determine gestational age (most accurately determined through measurement of crown-rump length prior to 11-12 weeks gestational age)
Non-Stress Test (NST) □ constant fetal heart rate (FHR) tracing using an external doppler to assess fetal heart rate and its relationship to fetal movement (see Intrapartum Fetal Cardiotocography) □ indicated when there is any suggestion of uteroplacental insufficiency or suspected fetal distress □ reactive NST (normal) • observation of two accelerations of FHR > 15 bpm from the baseline lasting ≥ 15 seconds in 20 minutes □ nonreactive NST (abnormal) • one or no FHR acceleration of at least 15 bpm and 15 seconds duration associated with fetal movement in 40 minutes • if no observed accelerations or fetal movement in the first 20 minutes, stimulate fetus (fundal pressure, acoustic/vibratory stimulation) and continue monitoring for 30 minutes • if NST nonreactive then perform BPP

Biophy	/sical Profil	le	(BPI	(د

☐ consists of NST and 30 minute ultrasound assessment of the fetus ☐ five scored parameters of BPP (see Table 2) ☐ scores

8-10 perinatal mortality rate 1: 1000 repeat BPP as clinically indicated
6 perinatal mortality 31:1000 repeat BPP in 24 hours
0-4 perinatal mortality rate 200:1000 deliver fetus if mature
AFV a marker of chronic hypoxia, all other parameters indicative of acute hypoxia

Table 2. Scoring of the Biophysical Profile		
Parameter	Normal (2)	Abnormal (0)
AFV	fluid pocket of 2 cm in 2 axes	oligohydramnios
NST	reactive	nonreactive
breathing	at least one episode of breathing lasting at least 30 seconds	no breathing
limb movement	three discrete movements	two or less
fetal tone	at least one episode of limb extension followed by flexion	no movement

INTRA-PARTUM MONITORING

Vaginal Exam ☐ membrane status ☐ cervical effacement (thinning), dilatation, consistency, position, application ☐ fetal presenting part, position, and station ☐ bony pelvis size and shape
Intrapartum Fetal Cardiotocography (CTG) ☐ external (doppler) vs. internal (scalp electrode) monitoring ☐ describe in terms of baseline FHR, variability (short term, long term) and periodicity (accelerations, decelerations) (see Table 3) ☐ baseline FHR • normal range is 120-160 bpm • a parameter of fotal well being us, dictross
 a parameter of fetal well-being vs. distress □ variability short term - beat to beat (requires scalp monitor) long term - described with respect to frequency and amplitude of change in baseline frequency is defined as number of times in a 1 minute period with an increase or decrease of at least 5 bpm lasting 5 seconds (average frequency is 3) amplitude is based on difference between highest and lowest FHR within a 1 minute period (11-25 bpm is average)
 periodicity accelerations excursion of 15 bpm or more lasting for at least 15 seconds, in response to fetal movement or uterine contraction decelerations describe in terms of shape, onset, depth, duration, recovery, occurrence, and impact on baseline FHR and variability early decelerations (see Figure 1) uniform shape with onset early in contraction, returns to baseline by end of contraction; slow gradual deceleration often repetitive, no effect on baseline FHR or variability due to vagal response to head compression benign, usually seen with cervical dilatation of 4-7cm variable decelerations (see Figure 2) most common type of periodicity seen during labour
 variable in shape, onset and duration may or may not be repetitive often with abrupt rapid drop in FHR, usually no effect on baseline FHR or variability due to cord compression or, in second stage, forceful pushing with contractions benign unless repetitive, with slow recovery, or when associated with other abnormalities of FHR late decelerations (see Figure 3) uniform (symmetric) in shape, with onset late in contraction, lowest depth after peak of contraction, and returns to baseline after end of contraction may cause decreased variability and change in baseline FHR must see 3 in a row, all with the same shape to define as late deceleration due to fetal hypoxia and acidemia, maternal hypotension, or uterine hypertonus usually a sign of uteroplacental insufficiency (ominous)
 usually a sign of deteroplacental insufficiency (offinious) manage with position change to left lateral decubitus, oxygen, stopping oxytocin, C/S

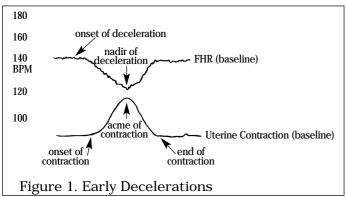
Table 3. Factors Affecting Fet	al Heart Rate	
Fetal Tachycardia (FHR > 160)	Fetal Bradycardia (FHR < 120)	Decreased Variability
fetal or maternal anemia fetal arrhythmia early hypoxia (abruption, PIH) chorioamnionitis maternal fever sympathomimetics (i.e. ritodrine) hyperthyroidism / thyrotoxicosis	uterine hypercontractility congenital heart block late hypoxia (abruption, PIH) maternal hypotension maternal use of beta blockers rapid descent during labour acute cord prolapse	hypoxia narcotics magnesium sulphate CNS anomalies of fetus fetal inactivity / sleep (< 20 min) maternal dehydration infection

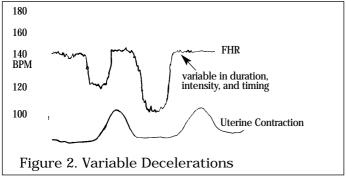
Approach to Abnormal FHR
☐ if external monitor, ensure fetal tracing and not maternal
☐ change position of mother give 100% oxygen by mask and discontinue oxytocin rule out cord prolapse consider fetal scalp electrode to assess beat-to-beat variability and fetal scalp blood sampling if abnormality persists immediate delivery if recurrent prolonged bradycardia Fetal Scalp Blood Sampling
☐ indicator of fetal distress
☐ > 7.25 is normal
☐ < 7.25 indicates that test should be repeated in 30 minutes
☐ < 7.20 indicates fetal acidosis severe enough to warrant immediate delivery Meconium in the Amniotic Fluid
☐ usually not present early in labour
☐ may occur prior to ROM or after rupture has occurred with passage of

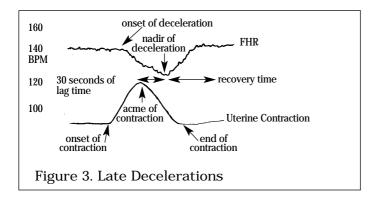
clear fluid classified as thick or thin

thin meconium appears as a lightly stained yellowish or greenish fluid thick meconium appears dark green or black and may have pea-soup

associated with lower APGARS and increased risk of meconium aspiration
call pediatrics to delivery
may indicate undiagnosed breech
increasing amount during labour may be a sign of fetal distress







MULTIPLE GESTATION

BACKGROUND

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

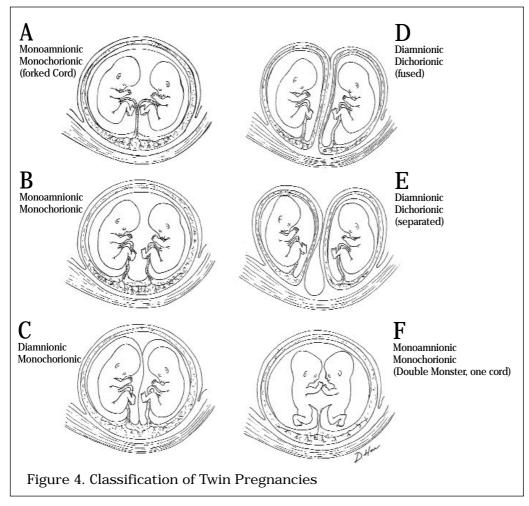


Table 4. Complications Associated with Multiple Gestation		
Maternal	Maternal-Fetal	Fetal
hyperemesis gravidarum DM preeclampsia / PIH PPH (uterine atony) placental abruption anemia (increased iron and folate needs) increased physiological stress on all systems increased compressive symptoms	increased PROM / PTL polyhydramnios umbilical cord prolapse placenta previa	prematurity* IUGR malpresentation congenital anomalies twin-twin transfusion (DA/MC) increased perinatal morbidity and mortality twin interlocking (twin A breech, twin B vertex) single fetal demise
C-section		_
* most common cause of perinatal mortality in multiple gestation		

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

MEDICAL CONDITIONS IN PREGANCY

URINARY TRACT INFECTION □ occurs more frequently in pregnancy and puerperium □ most common medical complication of pregnancy □ due to increased urinary stasis from mechanical and hormonal (progesterone) factors □ organisms • same as non-pregnant woman, also GBS □ may be symptomatic or asymptomatic; treat all □ risk of acute cystitis, pyelonephritis, and possible PPROM □ treatment of asymptomatic bacteruria or acute cystitis • amoxicillin first line • alternatives are TMP-SMX (Septra) or nitrofurantoin (avoid during last 6 weeks of pregnancy) • follow with monthly urine cultures • recurrence common □ treatment of pyelonephritis during pregnancy requires hospitalization and IV antibiotics
IRON DEFICIENCY ANEMIA □ iron requirements increase during pregnancy (mother needs 1000 mg of elemental iron per fetus; this amount exceeds normal stores) • fetus (500 mg) • RBC mass (500 mg) • losses (200 mg)
Etiology inadequate iron intake iron malabsorption bleeding, vaginal or other source multiple gestation concurrent antacid use (may prevent iron absorption)

Complications ☐ maternal: angina, CHF, infection, slower recuperation, preterm labour ☐ fetal: decreased oxygen carrying capacity leading to fetal distress, IUGR, low birth weight and hydrops
Diagnosis □ CBC, blood film, serum ferritin (changes in ferritin stores first sign of anemia) □ microcytic, hypochromic anemia with decreased ferritin □ morphology not good indicator because of RBC half life □ TIBC not reliable because increased during pregnancy
Treatment ☐ prevention • dietary iron and iron mobilized from stores insufficient to meet demands • adequate iron intake (30 mg elemental iron/day) for all women ☐ oral supplement of 200 mg/day of elemental iron if anemic ☐ monitor
FOLATE DEFICIENCY ANEMIA ☐ most often associated with iron deficiency anemia ☐ necessary for closure of neural tube during early fetal development ☐ minimum daily requirement is 0.5 mg ☐ takes approximately 18 weeks of folate deficient diet to produce anemia ☐ leafy green vegetables good source of dietary folate
Etiology Imalnutrition Imalabsorption (e.g. sprue) Inchronic hemolytic anemia (e.g. SCD) Imultiple gestation Immedications (i.e. phenytoin, trimethoprim-sulfamethoxazole, oral contraceptives)
Complications ☐ maternal: smaller blood volume, nausea, vomiting, anorexia ☐ fetal: NTD in T1, low birth weight, prematurity
Diagnosis □ suspect if iron deficiency anemia fails to respond to treatment □ CBC, blood film, red blood cell folate levels □ megaloblastic anemia and hypersegmented neutrophils on smear □ glossitis and skin roughness □ NO neurologic symptoms (unlike B12 deficiency) □ elevated serum iron and transferrin saturation
Treatment ☐ 1 mg folic acid PO daily ☐ 4 mg folic acid per day with past history of neural tube defect
DIABETES MELLITUS
Incidence \Box 2-3% of pregnancies are complicated by diabetes mellitus
Normal Physiology in Pregnancy ☐ in early pregnancy (T1) insulin secretion is increased and its anabolic actions are potentiated, decreasing fasting maternal glucose levels and promoting maternal energy storage ☐ in later pregnancy (T2,T3) insulin resistance develops ☐ anti-insulin factors: human placental lactogen (increased secretion with growth of the placenta) and cortisol ☐ result: higher fasting glucose and enhanced lipolysis (increased FFA, TG, lipids, ketones) to supply energy for fetal growth

Classification of Diabetes Mellitus (DM) Insulin Dependent DM (Type I) Non-Insulin Dependent DM (Type II) Gestational Diabetes: DM diagnosed during pregnancy	
Complications of Pregnancy in the Diabetic maternal hyportonsion/PET polyhydramnios, pyolonophritis/LEU	
 hypertension/PET, polyhydramnios, pyelonephritis/UTI ketoacidosis, diabetic coma, worsening retinopathy in Type I or Type II, NOT in GDM ☐ fetal	
 maternal hyperglycemia leads to fetal hyperinsulinism; accelerated anabolism and macrosomia result increased congenital anomalies and miscarriage from preconception or T1 hyperglycemia cardiac (VSD), neural tube, genitourinary, gastrointestinal and MSK (sacral agenesis) defects 	
IUGR if mother has end-organ damage delayed fetal lung maturity preterm labour/prematurity	
 increased incidence of stillbirth pregnancies complicated by GESTATIONAL diabetes do not manifest an increased risk of congenital anomalies because it develops later (i.e. after T1) 	
 neonatal macrosomia and associated birth trauma, hypoglycemia, hyperbilirubinemia and jaundice, hypocalcemia, polycythemia, and RDS 	
 see prior to pregnancy to optimize glycemic control (will reduce risk of congenital anomalies) since oral hypoglycemics are contraindicated, Type II's must be switched to insulin counsel re: potential complications and risks advise preconception folic acid see early and date pregnancy consult internist and dietitian to manage insulin and diet measure hemoglobin A₁C early in T1 or preconception if possible; this gives an indication of glycemic control during embryogenesis and can be used to estimate risk of birth defects initial evaluations: 24 hour urine (protein and creatinine clearance), retinal exam, ECG, urine C&S, hemoglobin A₁C throughout pregnancy monitor BP, urine dip (protein/glucose/ketones), weight gain, blood glucose (self-monitor) every visit and occasional urine C&S and hemoglobin A₁C in early pregnancy transfer of glucose and amino acids to the fetus results in a tendency toward maternal hypoglycemia nausea and vomiting may reduce food intake, therefore may need to decrease insulin dose 	
 office visits q 2 weeks MSAFP (at 16 weeks) and 3 detailed U/S examinations consider fetal echocardiography to exclude congenital heart defect admit for blood sugar control if needed in the second half of pregnancy, the diabetogenic action of placental hormones outweigh the continuous siphoning of glucose by the fetus demand for insulin is increased, hence insulin dosages need to be increased 	
 T3 office visits q 1 week fetal surveillance (BPP, NST); frequency depends on risk < 36 weeks = q weekly > 36 weeks = q weekly or biweekly 	

 timing of delivery dependent upon fetal and maternal risk factors can wait for spontaneous labour if glucose well-controlled and BPP normal induce by 40 weeks 	
 labour increased risk of CPD, shoulder dystocia with babies weighing over 4000 g 	
 elective C-section for predicted birthweights of greater than 4500 g (controversial) 	
during labour monitor sugars q1h with patient on insulin and dextrose drip and aim for blood sugar of 3.5 to 6.5 to reduce the risk of neonatal hypoglycemia Prostrortum On postpartum On the postpartum On the postpartum of the postpartum	
 postpartum increased risk of hypoglycemia once eating a regular diet, resume insulin at two-thirds of prepregnancy dose and monitor q6h 	
GESTATIONAL DIABETES ☐ glucose intolerance that is present only during pregnancy ☐ genetic predisposition to the development of glucose intolerance exists in this population of women ☐ 50% risk of developing Type II DM in next 20 years	
Risk Factors age > 30 previous history of high blood glucose, GDM, or macrosomic infant (> 4.5 kg) positive family history (GDM, Type II DM, macrosomic infant) excessive weight gain in pregnancy, prepregnancy obesity baby > 4.5 kg or large for GA previous unexplained stillbirth previous congenital anomaly early preeclampsia or polyhydramnios repeated vaginal candidiasis member of high risk ethnic group multiple gestation	
Diagnosis □ screen at 26 weeks (or earlier) with 50 g oral glucose challenge test if risk factors or glycosuria are present □ > 7.8 mmol/L at 1 hour is abnormal □ confirm with 3 hour 100 g oral glucose tolerance test (OGTT) • need 2 out of 4 values to be abnormal to diagnose GDM • fasting: > 5.8 mmol/L • 1 hour: > 10.6 mmol/L • 2 hour: > 9.2 mmol/L • 3 hour: > 8.1 mmol/L	
Management of Gestational Diabetes controversial aim to achieve normal blood sugars post-prandial (i.e. < 6.7 mmol/L) start with diabetic diet if blood sugars 2 hours post-prandial are > 6.7, add insulin (Humulin) oral hypoglycemic agents contraindicated in pregnancy fetal monitoring and timing of delivery same as for DM above insulin and diabetic diet should be stopped post-partum follow-up testing recommended postpartum because of increased risk of overt diabetes (i.e. OGTT at 6 weeks postpartum)	
HYPERTENSIVE DISORDERS OF PREGNANCY	
Classification ☐ preeclampsia/eclampsia ☐ chronic hypertension ☐ chronic hypertension with superimposed preeclampsia/eclampsia ☐ transient or gestational hypertension	

Preeclampsia/Preeclamptic Toxemia/Eclampsia (PET) hypertension accompanied by proteinuria and/or non-dependent edema with onset > 20 weeks • BP > 140/90 mmHg or an increment of 30 mmHg systolic and 15 mmHg diastolic over a nonpregnant or T1 BP • non-dependent edema (e.g. face, hands) is generalized and usually associated with excessive weight gain (> 2 kg/week) • proteinuria is defined as > 1+ protein on random dipstick analysis or > 300 mg in a 24 hour urine collection □ 50% of all hypertension in pregnancy □ due to an imbalance of thromboxane (vasoconstrictor) and prostaglandin (vasodilator), causing generalized arteriolar constriction □ resultant vasospasm damages capillaries, leading to protein extravasation and hemorrhage	
Conditions Associated with Preeclampsia/Eclampsia maternal factors • 80-90% of cases in primagravidas • past history or family history of preeclampsia/eclampsia • diabetes, chronic hypertension, or renal disease	
 extremes of maternal age fetal factors IUGR hydatidiform mole > 1 fetus fetal hydrops 	
Fetal Complications □ mainly due to placental insufficiency • fetal loss • IUGR • prematurity • abruptio placentae	
Maternal Complications cerebral hemorrhage (50% of deaths) left ventricular failure/pulmonary edema liver and renal dysfunction abruption seizures DIC release of placental thromboplastin, leading to a	
consumptive coagulopathy HELLP • hemolysis, elevated liver enzymes, low platelets • may only respond to fresh frozen plasma with plasma exchange	
Mild Preeclampsia PET uncomplicated by neurologic symptoms or criteria for a diagnosis of severe PET	
Severe Preeclampsia □ PET complicated by at least two of the following • BP > 160/110 • congestive heart failure • pulmonary edema or cyanosis • proteinuria > 5 g/24 hours or > 2+ on dipstick • elevated serum creatinine • oliguria (< 400 mL/24 hours) • thrombocytopenia (< 100 000 - 150 000/mL) • ascites • RUQ or epigastric pain (subcapsular hemorrhage) • elevated liver enzymes • hyperbilirubinemia • headache (cerebral artery vasospasm) • visual disturbances (i.e. scotomas, loss of peripheral vision) • hyperreflexia, clonus • IUGR	

Eclampsia ☐ grand mal seizures in a woman with preeclampsia
Management of Mild Preeclampsia □ maternal evaluation • history and physical examination (see above criteria) • laboratory • CBC and electrolytes • renal function tests —> BUN, creatinine, uric acid • liver enzymes and coagulation studies —> PT, PTT, FDP • urinalysis for protein and casts • 24 hour urine for protein and creatinine clearance
□ fetal evaluation of FHR, NST, BPP □ management with bed rest in left lateral decubitus position (reduces abdominal vessel compression) □ normal dietary salt and protein intake □ no use of diuretics/antihypertensives
Management of Severe Preeclampsia ☐ stabilize and deliver; the only "cure" is delivery ☐ admit and complete maternal evaluation (same as for mild) • keep NPO ☐ start W each and time
□ start IV, cross and type □ Foley catheter to monitor urine output □ maternal monitoring • hourly input and output, check urine q 12 hours for protein • vitals and DTR q 1 hour
 Italia and DTR q Thou If etal evaluation NST followed by continuous electronic fetal monitoring until delivery
 anticonvulsant therapy given to increase seizure threshold baseline magnesium blood level magnesium sulphate (4g IV push) followed by maintenance of 2-4 g/hour
 excretion of magnesium sulfate is via kidney therefore patients with oliguria require a lower infusion rate signs of magnesium toxicity (> 13 mg % serum level) depression of DTR depression of RR < 10/minute
 decreased muscle tonicity CNS or cardiac depression antagonist to magnesium sulphate is calcium gluconate (10%) 10 mL IV if respiratory arrest occurs antihypertensive therapy
 decreasing the BP decreases the risk of stroke (indicated only if BP > 140-170/90-110) first line: hydralazine 5 - 10 mg IV push over 5 minutes q 15 - 30 minutes until desired effect (an arteriolar vasodilator with minimal venous effect)
 controls BP for hours not days (deliver as soon as possible) next dose is given ~6 hours later with BP readings q 15 minutes also used in postpartum state if BP remains elevated and urinary output < 25 mL/hour second line: labetalol 20 - 50 mg IV q 10 minutes third line: nifedipine (Adalat) 10 -20 mg po q 20 - 60 minutes (puncture capsule and swallow liquid)
 postpartum management all antepartum therapy and monitoring continued until stable risk of seizure highest in first 24 hours postpartum continue magnesium sulfate for 12-24 hours after delivery the patient who continues to remain in serious condition may have HELLP
 most women return to a normotensive BP within 2 weeks but BP may worsen transiently in that time

Management of Eclampsia ☐ airway, breathing, circulation ☐ seizure control and prevention (see Neurology Notes) • do not attempt to shorten or abolish the initial convulsion • prevent maternal injury and maintain adequate oxygenation • minimize risk of aspiration, auscultate lungs after every seizure • give adequate magnesium sulphate as soon as convulsion has ended • correct maternal acidemia (obtain post-ictal blood gases) • some use valium for seizure control
Chronic Hypertension ☐ features • history of hypertension (> 140/90) before gestation • detection of hypertension prior to 20 weeks gestation (unless there is a GTN) • persistence of hypertension postpartum • strong family history of hypertension • most gravidas have essential hypertension, associated with an increased risk of preeclampsia or eclampsia, abruptio placenta, IUGR and IUD
 management methyldopa and/or labetalol no ACE inhibitors, diuretics, propranolol
Chronic Hypertension with Superimposed Preeclampsia/ Eclampsia □ 2-7 fold increased likelihood of developing preeclampsia/ eclampsia if pre-existing maternal hypertension □ tends to recur □ occurs early in pregnancy, tends to be severe, often with IUGR
Transient or Gestational Hypertension ☐ hypertension alone that develops during the latter half of pregnancy or during the first 24 hours after delivery and disappears within 10 days following parturition ☐ monitor for signs of preeclampsia/eclampsia
HYPEREMESIS GRAVIDARUM
Definition ☐ intractable nausea and vomiting to extent of weight loss, dehydration and electrolyte imbalance, acid-base disturbance and if severe, hepatic and renal damage ☐ usually present in T1 then diminishes; persists throughout pregnancy in a minority
Etiology □ presently thought to be multifactorial with hormonal, immunologic and psychologic components □ high or rapidly rising BhCG or estrogen levels are implicated
Maternal Complications ☐ Mallory Weiss tears ☐ Wernicke's encephalopathy, if protracted course ☐ death
Fetal Complications □ usually none □ IUGR is 15x more common in women losing > 5% of prepregnant weight
Differential Diagnosis of Nausea and Vomiting hyperemesis is a diagnosis of exclusion Gl inflammation/infection appendicitis cholecystitis henatitis

 gastroenteritis pancreatitis PUD fatty liver of pregnancy pyelonephritis thyrotoxicosis multiple gestation GTN (see Gynecology Notes) HELLP syndrome
Investigations ☐ labs (CBC, lytes, BUN and creatinine, urinalysis, LFTs) ☐ ultrasound (to R/O molar pregnancy, multiple pregnancy and to assess liver, pancreas, gallbladder, etc)
Treatment • early recognition is important • if severe, admit to hospital • NPO initially, then small frequent meals of appealing foods • correct hypovolemia, electrolyte imbalance and ketosis • thiamine, if indicated • TPN if severe to reverse catabolic state • consider emotional support, dietary and psychologic counselling □ pharmacological options • dimenhydrinate (Gravol) • vitamin B6 and doxylamine succinate (Diclectin) □ non-pharmacological options • accupressure at inner aspect of the wrists, just proximal to the flexor crease has been shown to significantly reduce symptoms of nausea and vomiting • avoid triggers (i.e. certain smells)
ISOIMMUNIZATION □ antibodies produced against a specific RBC antigen as a result of antigenic stimulation with RBC of another individual □ most common is anti-Rh Ab produced by a sensitized Rh-negative mother □ other antibodies can lead to fetal red blood cell hemolysis • much less common and no prophylaxis is available
Pathogenesis maternal-fetal circulation normally separated by placental barrier upon first exposure, initially IgM and then IgG antibodies are produced; IgG antibodies cross the placental barrier sensitization routes incompatible blood transfusion previous fetal-maternal transplacental hemorrhage invasive procedure while pregnant therapeutic abortion, D&C, amniocentesis complications anti-Rh Ab can cross the placenta and cause fetal hemolysis resulting in fetal anemia, CHF, edema, and ascites severe cases can lead to fetal hydrops (total body edema), or erythroblastosis fetalis
Diagnosis □ routine screening at first visit for blood group, Rh status, antibodies □ Ab titres < 1:16 considered benign □ Ab titres > 1:16 necessitates amniocentesis (correlation exists between amount of biliary pigment in amniotic fluid and severity of fetal anemia) from 24 weeks onwards □ Liley curve is used to determine bilirubin level and appropriate management (see below) □ Kleihauer-Betke test can be used to determine extent of fetomaternal hemorrhage • fetal red blood cells are identified on a slide treated with citrate phosphate buffer • adult hemoglobin is more readily eluted through cell membrane in presence of acid

	Rhogam binds to Rh Ag of fetus and prevents it from contacting maternal immune system Rhogam must be given to all Rh negative women • at 28 weeks • within 48 hours of the birth of an Rh positive fetus • positive Kleihauer-Betke test • with any invasive procedure in pregnancy • in the case of ectopic pregnancy • with miscarriage, therapeutic abortion • antepartum hemorrhage if Rh neg and Ab screen positive, follow mother with serial monthly
Tr	Ab titres throughout pregnancy +/– serial amniocentesis as needed (Rhogam of no benefit) reatment falling biliary pigment warrants no intervention (usually indicative
	of fetus which is unaffected or mildly affected) rising or stable biliary pigment on serial amniocentesis must be compared to a standard table which is divided into 3 zones based on severity of hemolysis (Liley Curve) intrauterine transfusion of O-negative packed red blood cells may be required for severely affected fetus or early delivery of the fetus for exchange transfusion
IN	FECTIONS DURING PREGNANCY
	protozoal infection (<i>Toxoplasma gondii</i>) incidence: 1/1000 pregnancies source: raw meat, unpasteurized goat's milk, cat urine/feces greatest risk of transmission in T3 severity of fetal infection greatest in T1 75% asymptomatic at birth, but may later develop sequelae risk of congenital toxoplasmosis (chorioretinitis, hydrocephaly, intracranial calcifications, MR, microcephaly) if primary maternal infection during pregnancy diagnosis based on serologic testing for both IgM and IgG confirmation of diagnosis based on presence of IgM antibodies in cord blood self-limiting infection, spiramycin (macrolide) decreases fetal morbidity
	nbella RNA togavirus with transmission by respiratory droplets (highly contagious) maternal infection during pregnancy (greatest in T1) may cause spontaneous abortion or Congenital Rubella Syndrome: hearing loss, cataracts, cardiovascular lesions, MR, symmetric IUGR, hepatitis, CNS defects and osseous changes diagnosis best made by serologic testing all pregnant women screened for rubella immunity (rubella titer > 1:16 = immune) non-immune • should be offered vaccination following pregnancy (not a contraindication for breast feeding) • rubella vaccine should be avoided before (3 months) or during pregnancy since it is an attenuated live vaccine
	tomegalovirus DNA virus (herpes family) transmission: blood transfusion, organ transplant, sexual contact, breast milk, transplacental, or direct contact during delivery congenital infection can occur from primary or re-infection of the mother increased fetal morbidity with primary infection risk of transmission constant across trimesters 5-10% of fetuses infected in utero will develop neurologic involvement (MR, cerebral calcification, hydrocephalus or microcephaly, deafness, chorioretinitis) diagnosis: isolation of virus in urine culture (or culture of other secretions), serologic screening for antibodies

Herpes ☐ DNA herpes virus ☐ transmission: intimate mucocutaneous contact ☐ primary infection during pregnancy increases risk of neonatal complications
□ 50% transmission if primary infection, 4% transmission if secondary recurrence
☐ infection to fetus may occur in utero but more commonly occurs
during delivery ☐ C-section if active genital lesions present within 4-6 hours of ROM, even if lesions remote from vulvar area
Syphilis ☐ Treponema pallidum ☐ may have transplacental transmission ☐ serological tests
Hepatitis B □ transmitted via blood, saliva, vaginal secretions, semen, breast milk, transplacental □ fetal infection most likely with T3 maternal infection □ risk of vertical transmission 10% if asymptomatic HBsAg +ve □ risk of vertical transmission 85-90% if HBsAg +ve and HBcAg +ve □ chronic active hepatitis increases risk of prematurity, low birth weight, neonatal death □ treatment of neonate with Hep B immune globulin (HBIG) and vaccine (at birth, one and six months) is 90% effective □ vaccine safe during pregnancy
Erythema Infectiosum (Fifth Disease) □ parvovirus B19 □ febrile illness with bilateral erythema of cheeks ("slapped cheek" rash) followed by maculopapular rash of trunk and extremities □ fetus of infected woman may develop hydrops in utero • follow fetus with weekly U/S (if hydrops occurs, consider fetal transfusion) □ risk of intrauterine death 1-12 weeks after infection
HIV ☐ offer screening to all women ☐ risk of vertical transmission 12 to 28%; more likely if maternal CD4 count < 300 ☐ risks to infected mom include decreased CD4 count, cancer, increased opportunistic infection (PCP, TB, CMV, toxoplasmosis, mycoplasma) ☐ care of HIV positive patient ● PCP prophylaxis with Bactrim if CD4 < 200 ● AZT shown to decrease transmission to fetus from 25% to 8% risk ● exclude cervical dysplasia ● toxoplasmosis and CMV antibodies

Group B Streptococccus (see Prenatal Care Section)

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

ACUTE SURGICAL	CONDITIONS
I incidence is approximate	taly 1 in 500 pragnancias

incidence is approximately 1 in 500 pregnancies
generally manage acute surgical condition regardless of

☐ pregnancy substantially increases complications associated with surgery

NON-EMERGENCY SURGICAL CONDITIONS

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

ANTENATAL HEMORRHAGE

FIRST AND SECOND TRIMESTER BLEEDING

Differential Diagnosis

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

Table 6. Types of Abortions			
Туре	History	Cervix	Management*
threatened	vaginal bleeding +/- cramps	closed - intact membranes	U/S shows viable fetus
inevitable	bleeding + cramps +/- ruptured membranes	open > 2 cm	D&C +/- oxytocin
incomplete	heaviest bleeding + cramps; soft abdomen; may have passage of tissue	open	D&C +/- oxytocin
complete	bleeding + complete sac and placenta passed	open	no D&C
missed	fetal death and retention of products; presents as pregnancy not progressing	closed	D&C +/- oxytocin
habitual	3 or more consecutive spontaneous abortions		evaluate environmental factors (smoking, alcohol, heavy caffeine), uterine anatomy, karyotype of both parents, TSH, antiphospholipid antibodies (including lupus anticoagulant and anticardiolipin antibodies)
therapeutic	for genetic, medical, and psychological reasons		see below
septic	contents of uterus infected before, during or after abortion		D&C IV wide spectrum antibiotics oxygen
* Rhogam to all Rl	h negative mothers	1	'

THERAPEUTIC ABORTIONS

- □ medical management
 < 9 weeks use methotrexate plus misoprostol (experimental)
 > 12 weeks use prostaglandins intra- or extra-amniotically, or IM
 □ surgical management
- - < 12-16 weeks use dilatation and curettage
 > 16 weeks use dilatation and evacuation
- complications
 - perforation of uterus
 hemorrhage
 laceration of cervix

 - · risk of sterility
 - infection usually due to retained products, occasionally endometritis
 - Asherman's syndrome (fibrosis of the uterus)

THIRD TRIMESTER BLEEDING

Di	ifferential Diagnosis
	placenta previa
	abruptio placentae vasa previa
	vasa previa
	bloody show (shedding of cervical mucous plug)
	marginal sinus bleeding
	marginal sinus bleeding cervical lesion (cervicitis, polyp, ectropion, cervical cancer)
	uterine rupture
	other: bleeding from bowel or bladder, placenta accreta, abnormal
	coagulation
	NB - do NOT perform a vaginal exam until placenta previa has been
	ruled out by U/S

PLACENTA PREVIA □ abnormal location of the placenta at or near the internal cervical os □ 1/200 at time of delivery □ many are low lying in early pregnancy but due to development of lower uterine segment appear to "move upward" as pregnancy nears term □ 95% of previas diagnosed in T2 resolve by T3; repeat U/S at 30-32 weeks GA
Classification ☐ total
 placenta completely covers the internal os partial placenta partially covers the internal os marginal placenta reaches margin but does not cover any part of the internal os low lying (NOT a previa) placenta in lower segment but clear of os can also bleed, usually later (i.e. in labour)
Etiology □ unknown but many associated conditions and risk factors • multiparity • history of placenta previa (4-8% recurrence risk) • multiple pregnancy • increased maternal age • uterine scar due to previous abortion, C-section, D&C, myomectomy • uterine tumour (e.g. fibroids) or other uterine anomalies
Fetal Complications perinatal mortality low but still higher than with a normal pregnancy prematurity (bleeding often dictates early C/S) intrauterine hypoxia (acute or IUGR) fetal malpresentation PPROM risk of fetal blood loss from placenta, especially if incised during C/S
Maternal Complications < 1% maternal mortality hemorrhage and hypovolemic shock anemia acute renal failure pituitary necrosis (Sheehan syndrome) PPH (because lower uterine segment is atonic) hysterectomy placenta accreta
Clinical Features □ recurrent, PAINLESS bright red vaginal bleeding • onset of bleeding depends on degree of previa (i.e. complete bleed earlier) • mean GA is 30 weeks; one third present before • initially, bleeding may be minimal and cease spontaneously but can be catastrophic later • bleeding at onset of labour can occur with marginal placenta previa □ uterus soft and non-tender □ presenting part high or displaced □ diagnosed by U/S (95% accuracy with transabdominal)
Management ☐ maternal stabilization; large bore IV with hydration ☐ electronic fetal monitoring

 □ maternal monitoring • vitals, urine output, blood loss • bloodwork including hematocrit, CBC, PTT/PT, platelets, fibrinogen, FDP, type and cross match □ when fetal and maternal condition permit, perform careful U/S examination to determine fetal viability, gestational age and placental status/position □ Rhogam given if mother is Rh negative □ management decision depends on • previa characteristics (amount of bleeding, degree of previa) • fetal condition (GA, level of distress, presentation) • uterine activity □ expectant management and observation of mother and fetus if the initial bleeding episode is slight and GA < 37 weeks • admit to hospital • limited physical activity • no douches, enemas, or sexual intercourse • consider corticosteriods for fetal lung maturity • delivery when fetus is mature or hemorrhage dictates □ delivery if bleeding is profuse, GA > 36 weeks, or L/S ratio is 2:1 or greater • usually C-section (incision site dictated by location of previa)
ABRUPTIO PLACENTAE ☐ premature separation of a normally implanted placenta after 20 weeks gestation ☐ incidence = 0.5-1.5%
Classification total (fetal death inevitable) vs. partial external/revealed/apparent; blood dissects downward toward cervix internal/concealed (20%); blood dissects upward toward fetus most are mixed
Etiology □ unknown, but associated with • maternal hypertension (chronic or PIH) in 50% of abruptions • multiparity • previous abruption (recurrence rate 10%) • PROM • maternal age > 35 (felt to reflect parity) • maternal vascular disease • cigarette smoking • alcohol consumption • uterine distension (polyhydramnios, multiple gestation) • short cord • trauma • sudden decompression of the uterus (twins) • uterine anomaly, fibroids
Fetal Complications perinatal mortality 25-60% prematurity intrauterine hypoxia
Maternal Complications < 1% maternal mortality DIC (in 20% of abruptions) acute renal failure anemia hemorrhagic shock pituitary necrosis (Sheehan syndrome) amniotic fluid embolus
Clinical Features PAINFUL vaginal bleeding; blood may be bright red or dark or clotted uterine tenderness and increased tone

ANTENATAL HEMORRHAGE ... CONT.

 □ degree of anemia may not correlate with degree of observed blood loss □ fetal distress; loss of variability, late decelerations (see Fetal Monitoring Section) □ 15% present with fetal demise
Diagnosis □ clinical □ U/S NOT helpful except to rule out placenta previa
Management initial management • maternal stabilization, IV hydration • fetal monitoring • monitor maternal vitals, urine output • blood for hemoglobin, platelets, PT/PTT, fibrinogen, FDP, cross and type • blood products on hand (red cells, platelets, cryoprecipitate) because of DIC risk • Rhogam if Rh negative □ mild abruption and GA < 36 weeks • close observation of fetal well-being and amount of bleeding • limited physical activity • serial Hct to assess concealed bleeding • delivery when fetus is mature or when hemorrhage dictates □ mild abruption and GA > 36 weeks • stabilization and delivery □ moderate to severe abruption • hydrate and restore blood loss and correct coagulation defect if present • vaginal delivery if no evidence of fetal or maternal distress and if cephalic presentation OR with dead fetus • labour must progress actively □ severe abruption and live fetus • C-section if fetal or maternal distress develops with fluid/blood replacement, labour fails to progress or non-cephalic fetal presentation
VASA PREVIA ☐ incidence 1 in 5000 ☐ occurs with velamentous insertion of cord into membranes of placenta; unprotected fetal vessels pass over the cervical os ☐ since bleeding is from fetus a small amount of blood loss can have catastrophic consequences • presents with painless vaginal bleeding and fetal distress (tachy- to bradyarrhythmia) ☐ Apt test (NaOH mixed with the blood) can be done immediately to determine if the source of the bleeding is fetal (supernatant turns pink) or maternal (supernatant turns yellow) ☐ Wright stain on blood smear and look for nucleated red blood cells (in cord not maternal blood) ☐ management is STAT C-section ☐ 50% perinatal mortality, increasing to 75% if membranes rupture (most infants die of exsanguination)

INTRA-UTERINE GROWTH RESTRICTION

Definition ☐ infants whose weight is < 10th %ile for a particular GA ☐ weight not associated with any constitutional or familial cause ☐ prone to problems such as meconium aspiration, asphyxia, polycythemia, hypoglycemia, and mental retardation ☐ greater risk of perinatal morbidity and mortality
Etiology maternal causes poor nutrition, cigarette smoking, drug abuse, alcoholism, cyanotic heart disease, severe DM, SLE, pulmonary insufficiency maternal-fetal
 any disease which causes placental insufficiency leading to inadequate transfer of substrate across the placenta includes PIH, chronic HTN, chronic renal disease, gross placental morphological abnormalities (infarction, hemangiomas) fetal causes
TORCH infections, multiple gestation, congenital anomalies Clinical Features
□ symmetric/Type I (20%) • occurs early in pregnancy • inadequate growth of head and body although the head:abdomen ratio may be normal • usually associated with congenital anomalies or TORCH
 asymmetric/Type II (80%) occurs late in pregnancy brain is spared therefore the head:abdomen ratio is increased usually associated with placental insufficiency more favorable prognosis than Type I
Diagnosis ☐ clinical suspicion ☐ SFH measurements at every antepartum visit ☐ more thorough assessment if mother is in high risk category or if SFH lags > 2 cm behind GA ☐ U/S exam should include assessment of BPD, head and abdomen circumference, head:body ratio, femur length and fetal weight ☐ doppler analysis of umbilical cord blood flow
 Management □ prevention via risk modification prior to pregnancy ideal □ most important consideration is accurate menstrual history and GA in which to assess the above data □ modify controllable factors: smoking, alcohol, nutrition □ bed rest (in LLD position) □ serial BPP (monitor fetal growth) □ delivery when extrauterine existence is less dangerous than continued intrauterine existence or if GA > 34 weeks with significant oligohydramnios □ liberal use of C-section since IUGR fetus withstands labour poorly
MACROSOMIA
Definition \Box fetal weight > 90th %ile for GA, > 4000 g at term
Clinical Features □ maternal associations • obesity • DM • past history of macrosomic infant • prolonged gestation • multiparity

Diagnosis ☐ maternal history for associated conditions ☐ serial examination (SFH) ☐ investigations (U/S) ☐ U/S predictors: polyhydramnios, T3 AC growth >1.5 cm/week, HC/AC ratio < 10th percentile, FL/AC ratio <20th percentile
Complications ☐ increased risk of perinatal mortality ☐ fetopelvic disproportion and shoulder dystocia more common ☐ complications of DM in labour (see Medical Illnesses in Pregnancy Section)
POLYHYDRAMNIOS
Definition ☐ amniotic fluid volume > 2 L at any stage in pregnancy ☐ > 8 cm x 8 cm pocket on U/S ☐ 1/250 deliveries ☐ up to 2/3 of fetuses with severe polyhydramnios have chromosomal problems
Causes idiopathic
Complications cord prolapse placental abruption malpresentation preterm labour uterine dysfunction and postpartum hemorrhage increased perinatal mortality rate
Clinical Features □ pressure symptoms from overdistended uterus (dyspnea, edema, hydronephrosis) □ uterus large for dates, difficulty palpating fetal parts and hearing fetal heart tones
Management ☐ find cause (40% idiopathic) ☐ complete fetal U/S evaluation ☐ mild to moderate cases require no treatment ☐ screen for maternal disease/infection as indicated ☐ if severely symptomatic, hospitalize and consider therapeutic amniocentesis ☐ admit if contracting or cervix dilating
OLIGOHYDRAMNIOS
Definition ☐ amniotic fluid index of 5 cm or less
Etiology of Early Onset Oligohydramnios decreased production • renal agenesis or dysplasia, urinary obstruction, posterior urethral valves (male)

 □ increased loss • prolonged amniotic fluid leak (although most often labour ensues) □ 15-25% of cases have fetal anomalies
Fetal Complications ☐ cord compression ☐ T1 onset • Potter's facies • limb deformities • abdominal wall defects ☐ onset at > 20 weeks • pulmonary hypoplasia
Late Pregnancy Onset Oligohydramnios ☐ amniotic fluid normally decreases after 35 weeks ☐ common in post-term pregnancies ☐ may be a marker for infants who may not tolerate labour well
Management ☐ oligohydramnios is an important sign of chronic placental insufficiency and always warrants admission and investigation • rule out ROM (see below) • fetal monitoring (NST, CTG, BPP) • consider delivery if at term

ANTENATAL COMPLICATIONS

PRETERM LABOUR
Definition ☐ labour occurring between 20 and 37 weeks gestation ☐ complicates about 10% of pregnancies ☐ prematurity is the leading cause of perinatal morbidity and mortality • at 30 weeks or 1500 g = 90% survival • at 33 weeks or 2000 g = 99% survival ☐ major causes of morbidity = asphyxia, sepsis, RDS ☐ intrapartum asphyxia may lead to cerebral hemorrhage
Etiology idiopathic (most common) maternal • prior history of premature delivery (recurrence risk of 17-40%) • preeclampsia/hypertension • placenta previa or abruption • uncontrolled diabetes • recurrent pyelonephritis and untreated bacteriuria • maternal genital tract infection • chorioamnionitis • other medical illness (heart disease, renal disease, severe anemia, systemic infection, chronic vascular disease) • maternal age < 18 years or > 40 years • fibroids or other uterine anomalies • incompetent cervix • history of abortions or stillbirths • surgical (intra-abdominal surgery, cholecystitis, peritonitis) • previous incision into uterus or cervix (C/S, conization) • low socioeconomic class • lack of prenatal care • poor nutrition • low prepregnancy weight • smoking • drug addiction (alcohol, cocaine) • stress/anxiety/fatigue

 □ maternal-fetal • PPROM (a common cause) • polyhydramnios □ fetal
multiple gestation congenital abnormalities of fetus
Requirements for Consideration of Labour Suppression (Tocolysis) live fetus fetal immaturity intact membranes cervical dilatation of 4 cm or less absence of maternal or fetal contraindications (see below) availability of necessary personnel and equipment to assess mother and fetus during labour and care for baby of the predicted GA if therapy fails
Maternal Contraindications to Tocolysis ☐ bleeding (placenta previa or abruption) ☐ maternal disease (hypertension, diabetes, heart disease) ☐ preeclampsia or eclampsia ☐ chorioamnionitis
Fetal Contraindications to Tocolysis erythroblastosis fetalis severe congenital anomalies fetal distress/demise IUGR, multiple gestation (relative)
Diagnosis ☐ regular contractions (2 in 10 minutes) ☐ cervix > 2 cm dilated or 80% effaced OR documented change in cervix
Prevention ☐ good prenatal care ☐ identify pregnancies at risk ☐ treat silent vaginal infection or UTI ☐ patient education ☐ the following may help but evidence for their effectiveness is lacking
Management ☐ initial • transfer to appropriate facility
 hydration (NS @ 150 mL/hour) bed rest in left lateral decubitus position sedation (morphine) avoid repeated pelvic exams (increased infection risk) U/S examination of fetus (for GA, BPP, position) prophylactic antibiotics; controversial but may help delay delivery aggressiveness depends on the GA tocolytic agents - if no contraindications present have no impact on neonatal morbidity or mortality but may buy time to allow celestone use or to transfer to
 appropriate centre beta-mimetics: ritodrine, terbutaline magnesium sulphate (if diabetes or cardiovascular disease present) calcium channel blockers: nifedipine PG synthesis inhibitors (2nd line agent): indomethacin

Enhancement of Pulmonary Maturity most effective between 28 and 34 weeks gestation treatment: betamethasone valerate (Celestone) 12 mg IM q12h times 2 wait 24 hours for delivery specific maternal contraindications
RUPTURE OF MEMBRANES
Premature ROM ☐ rupture of membranes prior to the onset of labour at any GA
Prolonged ROM ☐ if 24 hours elapse between rupture of membranes and onset of labour
Preterm ROM ☐ ROM occurring before 37 weeks gestation (associated with PTL)
PPROM ☐ preterm premature rupture of membranes (not in labour)
Associated Conditions congenital anomaly infection
Causes ☐ idiopathic (most common) ☐ frequently associated with • multiparity • cervical incompetence • infection: cervicitis, vaginitis, STD, UTI • multiple gestation • family history of PROM • low socioeconomic class/poor nutrition • and other risk factors associated with PTL (see above)
Complications ☐ cord prolapse ☐ intrauterine infection (chorioamnionitis) ☐ premature delivery
Diagnosis ☐ history of fluid gush or continued leakage ☐ avoid introducing infection with examinations (do not do a digital pelvic exam) ☐ sterile speculum exam • pooling of fluid in the posterior fornix • may observe fluid leaking out of cervix on valsalva ☐ amniotic fluid turns nitrazine paper blue (low specificity as can be blood, urine or semen) ☐ ferning (high salt content of amniotic fluid evaporates and looks like ferns under microscope) ☐ U/S
Management ☐ cultures (cervix for GC, lower vagina for GBS) ☐ dependent upon gestational age; must weigh degree of prematurity vs risk of amnionitis and sepsis by remaining in utero • < 24 weeks consider termination (poor outlook due to pulmonary hypoplasia) • 26-34 weeks: expectant management as prematurity complications significant • 34-36 weeks: "grey zone" where risk of death from RDS and neonatal sepsis is the same

 > 36 weeks: induction of labour since the risk of death from sepsis is greater than RDS □ assess fetal lung maturity by L/S ratio of amniotic fluid □ consider administration of betamethasone valerate (Celestone) to accelerate maturity □ if not in labour or labour not indicated, consider antibiotics (controversial) □ admit and monitor vitals q4h, daily BPP and WBC count 	
UMBILICAL CORD PROLAPSE □ descent of the cord to a level adjacent to or below the presenting part causing cord compression between presenting part and pelvis □ visible or palpable cord □ FHR changes (variable decelerations, bradycardia or both) □ increased incidence with prematurity/PROM, fetal malpresentations, low-lying placenta, polyhydramnios, multiple gestation, CPD	
Management ☐ "STAT" C-section ☐ adjunctive measures • alleviate pressure of the presenting part on the cord • keep cord warm and moist by replacing it into the vagina and/or applying warm saline soaks	
CHORIOAMNIONITIS ☐ definition: infection of the chorion, amnion and amniotic fluid ☐ risk factors: prolonged ROM, long labour, multiple vaginal exams during labour, internal monitoring, bacterial vaginosis and other vaginal infections ☐ clinical features: maternal fever, maternal or fetal tachycardia, uterine tenderness, foul cervical discharge, leukocytosis, presence of leukocytes or bacteria in amniotic fluid ☐ management: blood and amniotic fluid cultures, IV antibiotics (ampicillin and gentamycin) ☐ expedient delivery regardless of gestational age	
POST-DATE PREGNANCY	
Definition and Clinical Features □ pregnancy beyond 42 weeks (10% of pregnancies) □ accurate dating essential □ etiology unknown □ morbidity increased with hypertension/PET, DM, abruption, IUGR and multiple gestation	
Complications □ perinatal mortality 2-3 x higher □ oligohydramnios □ meconium passage; risk of meconium aspiration □ asphyxia □ macrosomia □ placental insufficiency; infarction of aging placenta □ postmaturity syndrome: 10-20% of post-term pregnancies (fetal weight loss, reduction in subcutaneous fat, scaling, dry skin from placental insufficiency)	
Management ☐ fetal movement count by the mother ☐ BPP twice weekly from 40 weeks ☐ delivery after 41 weeks if not already in labour since perinatal mortality is higher secondary to progressive uteroplacental insufficiency ☐ if BPP < 10/10 at any time, deliver ☐ decreased tolerance for asphyxia intrapartum	

INTRAUTERINE FETAL DEATH ☐ incidence = 1% of pregnancies
Etiology unknown in 50% hypertension, DM erythroblastosis fetalis congenital anomalies umbilical cord or placental complications intrauterine infection antiphospholipid Ab's
Clinical Features history
 • decreased perception of fetal movement by mother □ examination • SFH and maternal weight not increasing
 absent fetal heart tones (not diagnostic) investigations absent cardiac activity and fetal movement on U/S required for diagnosis high MSAFP
Management ☐ labour induction (see Abnormal Labour Section) ☐ must monitor for maternal coagulopathy (10% risk of DIC) ☐ psychologic aspects of fetal loss ☐ investigations to determine cause ☐ subsequent pregnancies high risk
NORMAL LABOUR AND DELIVERY
THE FETUS
1112 1 2 1 0 0
Fetal Lie ☐ refers to the orientation of the long axis of the fetus with respect to the long axis of the uterus • longitudinal • transverse • oblique ☐ transverse/oblique often due to uterine anomalies (C-section if they don't convert)
Fetal Lie □ refers to the orientation of the long axis of the fetus with respect to the long axis of the uterus • longitudinal • transverse • oblique □ transverse/oblique often due to uterine anomalies (C-section if they don't convert) Fetal Presentation □ refers to the fetal part presenting at pelvic outlet • breech (complete, frank, footling) • cephalic
Fetal Lie □ refers to the orientation of the long axis of the fetus with respect to the long axis of the uterus • longitudinal • transverse • oblique □ transverse/oblique often due to uterine anomalies (C-section if they don't convert) Fetal Presentation □ refers to the fetal part presenting at pelvic outlet • breech (complete, frank, footling) • cephalic • vertex • brow • face
Fetal Lie □ refers to the orientation of the long axis of the fetus with respect to the long axis of the uterus • longitudinal • transverse • oblique □ transverse/oblique often due to uterine anomalies (C-section if they don't convert) Fetal Presentation □ refers to the fetal part presenting at pelvic outlet • breech (complete, frank, footling) • cephalic • vertex • brow

Attitude ☐ refers to flexion/extension of fetal head relative to shoulders • brow presentation: head partially extended (requires C-section) • face presentation: head fully extended (mentum posterior always requires C-section, mentum anterior will deliver vaginally)	
Station ☐ defined by position of presenting part relative to ischial spines • at ischial spines = station 0 = engaged	
THE CERVIX ☐ dilatation • latent phase: 0-3 cm • active phase: 4-10 cm ☐ effacement • thinning of the cervix (25%-50%-100%) ☐ consistency • soft vs. hard ☐ position • posterior vs. anterior ☐ application • contact between the cervix and presenting part	
DEFINITION OF LABOUR □ regular, painful uterine contractions increasing in frequency, accompanied by progressive DILATATION and EFFACEMENT of cervix, and normally associated with DESCENT of presenting part • preterm (> 20 but < 37 weeks GA) • term (37-42 weeks) • post-term (> 42 weeks) □ Braxton-Hick contractions • irregular and painless, occur throughout pregnancy and not associated with any dilatation, effacement or descent	
FOUR STAGES OF LABOUR	
First Stage of Labour (see Table 7) □ latent phase • uterine contractions typically infrequent and irregular • slow cervical dilatation (usually to 3-4 cm) and effacement □ active phase • rapid cervical dilatation to full dilatation (nulliparous ~1.2 cm/h and ~1.5 cm/h in multiparas) • phase of maximum slope on Friedman curve (see Figure 6) • painful, regular contractions ~q 2 min, lasting 45-60 seconds • contractions strongest at fundus, weakest at lower segment	
Second Stage of Labour from full dilatation to delivery of the baby mother feels a desire to bear down and push with each contraction progress measured by descent	
Third Stage of Labour □ separation and expulsion of the placenta □ can last up to 30 minutes before intervention indicated □ signs of placenta separation: gush of blood, lengthening of cord, uterus becomes globular	
Fourth Stage of Labour ☐ first postpartum hour ☐ monitor vital signs and bleeding +/- oxytocin ☐ repair lacerations ☐ ensure uterus is contracted (palpate uterus and monitor uterine bleeding) ☐ 3rd and 4th stages of labour most dangerous to the mother (i.e. hemorrhage)	

Table 7. 0	Course of Normal La	bour
Stage	Nulliparous	Multiparous
first second third	6-18 hours 30 minutes-3 hours 5-30 minutes	2- 10 hours 5-30 minutes 5-30 minutes

THE CARDINAL MOVEMENTS OF THE FETUS DURING DELIVERY

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

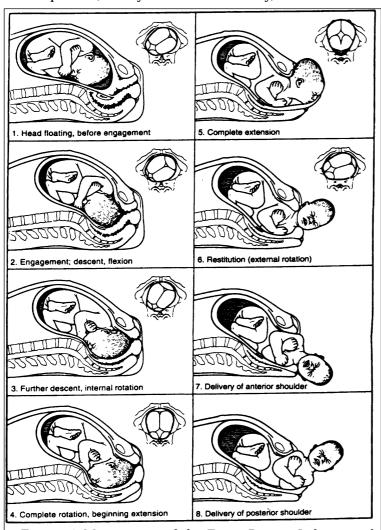


Figure 5. Movements of the Fetus During Labour and Delivery, Left Occiput Anterior Position

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

INDUCTION OF LABOUR

Definition the artificial initiation of labour to maintain maternal health or to remove the fetus from a potentially harmful environment Perecquisites For Labour Induction maternal short anterior cervix with open os ("inducible" or "ripe") if cital fetal adequate fetal monitoring available cephalic presentation good fetal health Indications maternal factors perganany-induced hypertension naternal medical problems, e.g. diabetes, renal or lung disease maternal-fetal factors pregnancy-induced hypertension prost challension post-tension induced hypertension post-tension induced hypertension prost challension induced hypertension prost challension induced hypertension prior classical incision or complete transection of the uterus unstable maternal condition goss CPD maternal prior classical incision or complete transection of the uterus unstable maternal genital herpes maternal prior classical incision or complete transection of the uterus unstable maternal genital herpes maternal classical incision or complete transection of the uterus unstable maternal genital herpes maternal prior classical incision or complete transection of the uterus distress dis	
maternal short anterior cervix with open os ("inducible" or "ripe") fetral short anterior cervix is not ripe, use prostaglandin (PG) gel (see below) fetal adequate fetal monitoring available cephalic presentation good fetal health Indications maternal factors pregnancy-induced hypertension maternal factors pregnancy-induced hypertension maternal factors historiamunization PROM chorioamnionitis Promise historiamunization PROM chorioamnionitis post-term pregnancy fetal factors suspected fetal geopardy as evidenced by biochemical or biophysical indications letal demise letal demise prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete prior classical incision or effacement prior classica	☐ the artificial initiation of labour to maintain maternal health or to
adequate fetal monitoring available cephalic presentation pood fetal health Indications maternal factors pregnancy-induced hypertension maternal fetal factors maternal fetal factors This immunization PROM choricamininitis post-term pregnancy fetal factors suspected fetal jeopardy as evidenced by biochemical or biophysical indications fetal demise Contraindications maternal maternal prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus prior classical incision or complete transection of the uterus incision active Note maternal genital herpes maternal classical incision or complete transection of the uterus classical incision or comp	 maternal short anterior cervix with open os ("inducible" or "ripe") if cervix is not ripe, use prostaglandin (PG) gel (see below)
□ maternal factors	 adequate fetal monitoring available cephalic presentation
fetal factors suspected fetal jeopardy as evidenced by biochemical or biophysical indications maternal prior classical incision or complete transection of the uterus unstable maternal condition gross CPD active maternal genital herpes maternal-fetal placenta or vasa previa tetal distress malpresentation PG synthesized by cervical cells and in amniotic fluid to facilitate abour onset and progression PG gel used to augment slow or arrested cervical dilatation or effacement intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM vaginal when cervix favorable, may use with ROM use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction risks include hyperstimulation and fetal heart rate abnormalities botain reactive NST prior to administration Foley catheter may be used to mechanically dilate the cervix Medical oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute potential complications hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) PGF-2 alpha used for intrauterine fetal demise (IUFD) Surgical artificial rupture of membranes (amniotomy) - may try this as initial	 □ maternal factors • pregnancy-induced hypertension • maternal medical problems, e.g. diabetes, renal or lung disease □ maternal-fetal factors • Rh isoimmunization • PROM • chorioamnionitis
 maternal prior classical incision or complete transection of the uterus unstable maternal condition gross CPD active maternal genital herpes maternal-fetal placenta or vasa previa fetal distress malpresentation Cervical Ripening Principles PG synthesized by cervical cells and in amniotic fluid to facilitate labour onset and progression PG gel used to augment slow or arrested cervical dilatation or effacement intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM vaginal when cervix favorable, may use with ROM use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction risks include hyperstimulation and fetal heart rate abnormalities obtain reactive NST prior to administration Foley catheter may be used to mechanically dilate the cervix Medical oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute potential complications hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) uterine muscle fatigue, uterine atony (may result in PPH) vasopressin-like action causing anti-diuresis PGF-2 alpha used for intrauterine fetal demise (IUFD) Surgical artificial rupture of membranes (amniotomy) - may try this as initial	 ↓ fetal factors • suspected fetal jeopardy as evidenced by biochemical or biophysical indications
e distress • malpresentation Cervical Ripening Principles □ PG synthesized by cervical cells and in amniotic fluid to facilitate labour onset and progression □ PG gel used to augment slow or arrested cervical dilatation or effacement • intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM • vaginal when cervix favorable, may use with ROM • use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction • risks include hyperstimulation and fetal heart rate abnormalities • obtain reactive NST prior to administration □ Foley catheter may be used to mechanically dilate the cervix Medical □ oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute □ potential complications • hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) • uterine muscle fatigue, uterine atony (may result in PPH) • vasopressin-like action causing anti-diuresis □ PGF-2 alpha used for intrauterine fetal demise (IUFD) Surgical □ artificial rupture of membranes (amniotomy) - may try this as initial	 □ maternal prior classical incision or complete transection of the uterus unstable maternal condition gross CPD active maternal genital herpes □ maternal-fetal placenta or vasa previa
 □ PG synthesized by cervical cells and in amniotic fluid to facilitate labour onset and progression □ PG gel used to augment slow or arrested cervical dilatation or effacement intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM vaginal when cervix favorable, may use with ROM use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction risks include hyperstimulation and fetal heart rate abnormalities obtain reactive NST prior to administration □ Foley catheter may be used to mechanically dilate the cervix Medical □ oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute □ potential complications hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) uterine muscle fatigue, uterine atony (may result in PPH) vasopressin-like action causing anti-diuresis □ PGF-2 alpha used for intrauterine fetal demise (IUFD) Surgical □ artificial rupture of membranes (amniotomy) - may try this as initial 	• distress
 □ oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute □ potential complications hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) uterine muscle fatigue, uterine atony (may result in PPH) vasopressin-like action causing anti-diuresis □ PGF-2 alpha used for intrauterine fetal demise (IUFD) Surgical □ artificial rupture of membranes (amniotomy) - may try this as initial 	 □ PG synthesized by cervical cells and in amniotic fluid to facilitate labour onset and progression □ PG gel used to augment slow or arrested cervical dilatation or effacement intracervical dinoprostone (Prepidil) when cervix long and closed and no ROM vaginal when cervix favorable, may use with ROM use associated with reduced rate of C/S, instrumental vaginal delivery, and failed induction risks include hyperstimulation and fetal heart rate abnormalities obtain reactive NST prior to administration
☐ artificial rupture of membranes (amniotomy) - may try this as initial	Medical □ oxytocin 2 mU/minute IV, increasing by 1-2 mU/minute every 20-30 minutes to a maximum of 36-48 mU/minute □ potential complications • hyperstimulation/tetanic contraction (may cause fetal distress or rupture of uterus) • uterine muscle fatigue, uterine atony (may result in PPH) • vasopressin-like action causing anti-diuresis
	☐ artificial rupture of membranes (amniotomy) - may try this as initial

AUGMENTATION OF LABOUR

- augmentation of labour is used to promote adequate contractions when spontaneous contractions are inadequate and cervical dilatation or descent of fetus fails to occur
- oxytocin @ 2 mU/minute IV, increased by 1-2 mU/minute q 20-30 minutes to a maximum of 36-48 mU/minute
- ☐ half-life of oxytocin is ~2 minutes (thus need continuous drip because effects wear off fast)

- ABNORMAL PROGRESS OF LABOUR

 □ expected patterns of descent of the presenting part and cervical dilatation fail to occur in the appropriate time frame (see Figure 6) can occur in all stages of labour
- ☐ traditionally three causes of abnormal labour have been recognized
 - Power: poor, inadequate or uncoordinated uterine contractions

 - Passenger: fetus too large in size or unusual presentation
 Passage: cephalopelvic disproportion (CPD) = pelvis of inadequate size, shape or consistency, or maternal soft tissue resistance relative to fetus
- \square initial diagnosis of CPD requires progression into the active phase and the presence of adequate uterine contractions

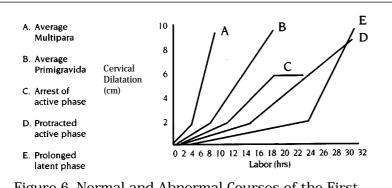


Figure 6. Normal and Abnormal Courses of the First Stage of Labour (Friedman Curve)

Prolonged Latent Phase (Curve E)

- a period of 20 hours or more in the primigravida or 14 hours or more in the multigravida during which labour has not progressed to the active phase
- most often patient not really in labour (avoid amniotomy for fear of false labour and increased risk of intrauterine infection)
- utoo early and too heavy sedation is present in 30-40% of these patients
- careful search for factors of CPD should be made
- ☐ treatment: oxytocin augmentation if diagnosis of labour is certain otherwise rest +/- sedation

Protraction Disorders (Curve D)

- of dilatation: when slope of cervical dilatation is less than
- 1.2 cm/hour in the primigravida or 1.5 cm/hour in the multigravida
- ☐ of descent: a rate of descent of less than 1.0 cm/hour in the
- primigravida or 2.0 cm/hour in the multigravida in about 1/3 CPD will be present so that secondary arrest of
- dilatation usually develops

 □ 2/3 will progress steadily through labour with ultimate uneventful vaginal delivery
- ☐ treatment: oxytocin augmentation if contractions are inadequate (see Augmentation of Labour Section) and/or amniotomy

Arrest Disorder (Curve C)

- of dilatation: progress in dilatation does not occur for a period of 2 hours or more in a patient who has entered the active phase
- ☐ arrest usually occurs at a cervical dilatation of 5 to 8 cm

 □ of descent: no progress in station for > 1 hour during second stage □ should search for factors causing CPD (nearly 50%; requires C-section) □ CPD if adequate contractions measured by intrauterine pressure
catheter (IUPC) and no descent/dilatation for > 2 hours if CPD ruled out, IV oxytocin and amniotomy can be attempted
SHOULDER DYSTOCIA
Definition, Incidence and Complications impaction of anterior shoulder of fetus against symphysis pubis after fetal head has been delivered (life threatening emergency) occurs when breadth of shoulders is greater than biparietal diameter of the head incidence is 0.15-1.4% of deliveries watch for "turtle sign" (head advances during contraction but returns to previous position at end of contraction) chest compression by vagina or cord compression by pelvis can lead to hypoxia danger of brachial plexus injury (Erb palsy) fetal fracture (clavicle, humerus, cervical spine) maternal perineal injury, may result in PPH
Associated Conditions maternal maternal obesity diabetes multiparity fetal prolonged gestation
 macrosomia labour prolonged 1st and 2nd stages prolonged deceleration phase (8-10 cm) instrumental midpelvic delivery
Management goal: to displace anterior shoulder from behind symphysis pubis initial gentle traction with maternal pushing adequate analgesia apply suprapubic pressure (to dislodge shoulder) with downward traction ask for help legs into hyperflexion on maternal abdomen (McRobert maneuver) anterior shoulder disimpaction release posterior shoulder (deliver posterior arm and shoulder) maneuver of Wood's corkscrew (insert hand beyond occiput into vagina and push anterior shoulder forward to the oblique or push the posterior shoulder through a 180 degree arc to reduce the biacromial diameter presented to the pelvic inlet) episiotomy (midline) cleidotomy: deliberate fracture of the clavicle (last resort) Zavanelli maneuver (involves flexion of the fetal head, replacement of the fetus within the uterine cavity and emergent C-section; reported success in a small series)
BREECH PRESENTATION
Definition ☐ fetal buttocks is the presenting part
Incidence ☐ occurs in 3-4% of pregnancies at term (25% before 28 weeks)
Classification (see Figure 7) ☐ complete: flexion at hips and knees

☐ frank: flexion at hips, extension at knees most common type of breech presentation only breech presentation delivered vaginally ☐ footling: may be single or double with extension at hip(s) and knee(s) so that foot is the presenting part Etiology maternal pelvis (contracted)uterus (shape abnormalities, intrauterine tumours, fibroids extrauterine tumours causing compression) • grand multiparity

maternal-fetal placenta (previa) amniotic fluid (poly/oligohydramnios) ☐ fetal prematurity multiple géstation • congenital malformations (found in 6% of breeches; 2-3 x the incidence in vertex presentations) Diagnosis ☐ Leopold maneuvers and U/S Management extenal breech version • criteria: > 37 weeks, singleton, unengaged presenting part, reactive NST • contraindications: previous T3 bleed, prior classical C-section, previous myomectomy, oligohydramnios, PROM, placenta previa, abnormal U/S, suspected IUGR risks: abruption, cord compression
method: tocolysis, followed by transabdominal manipulation of fetus, guided by ultrasound • if patient Rh negative Rhogam given prior to procedure ☐ good prognostic factors (for a successful version) • multiparous good fluid volume • small baby skilled obstetrician skilled obstetrican
 criteria for vaginal delivery
 frank breech, GA > 36 weeks
 EBW 2200-3800 g based on clinical and U/S assessment
 fetal head flexed
 continuous fetal monitoring
 maternal pelvis adequately large (clinically, or "proven" by previous delivery) no other indication for C/S experienced obstetrician ☐ C-section for all other presentations (except mentoanterior face presentation) A. Complete Breech C. Footling Breech B. Frank Breech

Illustration by Jennifer Bosy

Figure 7. Types of Breech Presentation

VAGINAL BIRTH AFTER CESAREAN (VBAC) □ recommended after previous low transverse incision □ success rate varies with indication for previous C-section (generally 60-80%) □ risk of uterine rupture (< 1% with low transverse incision)
Contraindications (i.e. need to do a repeat C/S before onset of labour) □ previous classical, inverted T, or unknown uterine incision, or complete transection of uterus (6% risk of rupture) □ history of hysterotomy or previous uterine rupture □ multiple gestation □ estimated fetal weight > 4000 g □ non-vertex presentation or placenta previa □ inadequate facilities or personnel for emergency C-section
UTERINE RUPTURE □ associated with previous uterine scar (in 40% of cases), hyperstimulation with oxytocin, grand multiparity and previous intrauterine manipulation □ generally occurs during labour, but can occur prior with a classical incision
Complications maternal mortality 1-10% maternal hemorrhage and shock DIC maniotic fluid embolus hysterectomy fetal distress —> 50% mortality
Management ☐ immediate delivery for fetal survival ☐ maternal stabilization (may require hysterectomy)
AMNIOTIC FLUID EMBOLUS
Definition ☐ amniotic fluid debris in maternal circulation ☐ rare intrapartum or immediate postpartum complication ☐ 80% mortality
Presentation □ sudden onset of respiratory distress, cardiovascular collapse and coagulopathy
Risk Factors placental abruption rapid labour multiparity uterine rupture
Treatment ☐ supportive measures, coagulopathy correction

INDICATIONS FOR OPERATIVE VAGINAL DELIVERY ☐ operative vaginal delivery is with forceps or vacuum extraction ☐ fetal
 non-reassuring fetal status consider if second stage is prolonged as this may be due to poor contractions or failure of fetal head to rotate maternal
 need to avoid voluntary expulsive effort (cardiac/cerebrovascular disease) exhaustion, lack of cooperation and excessive analgesia may
impair pushing effort TFORCEPS
Low Forceps ☐ head visible between labia in between contractions ☐ often called outlet forceps ☐ sagittal suture in or close to A-P diameter ☐ rotation cannot exceed 45 degrees
Mid Forceps ☐ presenting part below spines but not yet visible at introitus ☐ not below 2+ spines
Types of Forceps ☐ Simpson forceps for OA presentations ☐ rotational forceps (Kjelland) when must rotate head to OA ☐ Piper forceps for breech
Absolute Prerequisites vertex, face or breech presentations fully dilated cervix empty bladder (risk of tear if full) adequate analgesia ruptured membranes position and station are known presenting part below ischial spines experienced obstetrician pelvis of adequate size and shape uterine contractions present facilities to perform emergency C-section if needed
VACUUM EXTRACTION □ traction instrument used as alternative to forcep delivery, aids maternal pushing □ same indications as forceps □ advantages • easier to apply • less force on fetal head, less anesthesia required • less maternal and fetal injury • will dislodge if unrecognized CPD present □ disadvantages • suitable only for vertex presentations • maternal pushing required • contraindicated in preterm delivery
LACERATIONS ☐ first degree • involves skin and vaginal mucosa but not underlying fascia and muscle ☐ second degree • involves fascia and muscles of the perineal body but not the anal sphincter ☐ third degree
 involves the anal sphincter but does not extend through it fourth degree extends through the anal sphincter into the rectal lumen

EPISIOTOMY

☐ making an incision in the perineal body at the time of delivery ☐ midline (better healing, increased risk of deep tear) vs. mediolateral (less risk of extensive tear, poorer healing/more pain)
Indications ☐ to prevent a tear (episiotomy easier to repair) ☐ to relieve obstruction of the unyielding perineum ☐ instrumental delivery ☐ controversy over whether it is preferable to make a cut, or let the perineum tear as needed
CESAREAN DELIVERY
Indications □ maternal • obstruction, active herpetic lesion on vulva, invasive cervical cancer, previous uterine surgery □ maternal-fetal • failure to progress, placental abruption or previa □ fetal • fetal distress, malpresentation, cord prolapse, certain congenital anomalies
Risks anesthesia hemorrhage infection (UTI, wound, endometritis) increased recovery time/hospital stay

DBSTETRICAL ANESTHESIA

1	ח	Λ	. 1	ГЛ	T	\mathbf{D}	Λ	т	L	N	1 7	Λ	. 7	ZC	י י	П	IΤ	D	т	NI		י י	r /	١ ١	D	$\boldsymbol{\Gamma}$	١ı	Т	D)
	Г	r	M	Ш	N	Г	н	1	п	LV	V	Н	M	ZS		D	U	П	ш	LN	U	Г.	L A	١.	D	Ų.	N	J.	П	٠

- ☐ early first stage: pain via visceral afferents enter the spinal cord at T10-L1

 • dilatation of the cervix

 - lower uterine distension
- lower uterine distension
 contraction of the uterus
 □ late first stage and second stage pain via visceral and somatic afferents (pudendal nerve) enter the spinal cord at S1-S5
 contraction of the uterus
 distention and stretching of pelvic structures (pelvic peritoneum, fascia, ligaments, and muscles)
 pressure on lumbar nerves
 □ third stage of labour is usually well tolerated with spontaneous placents
- third stage of labour is usually well tolerated with spontaneous placental delivery
 analgesia may be necessary for manual extraction of placenta

ANALGESIA	
Psychoprophylaxis and Physical Analgesia "natural childbirth" (e.g. Lamaze prenatal classes) we informed mother utilizes relaxation techniques to state the state of the stat	imulate the
descending inhibitory pathways whirlpool baths, transcutaneous nerve stimulation (acupuncture inhibit nociceptive impulses and redu propagating muscle tension	TNS), and ce pain
especially effective in early stages of labour	
Intravenous Analgesia ☐ meperidine (Demerol)	
 best used in early stages of labour, less effect is well established 	ive once labour
 rapidly cleared by fetus if IV (prolonged if IM) peak fetal level 2-4 hours after maternal injection 	

- can suppress respiration in the newbom (treatment with naloxone)
- side effects of orthostatic hypotension, nausea, and vomiting

- Inhalational Analgesia
 ☐ nitrous oxide

 50% nitrous oxide in O2

 self-administered during contractions

 does not prolong labour or interfere with uterine contractions but administration > 20 minutes may result in neonatal depression

 provides partial pain relief during labour as well as at delivery

ANESTHESIA
Local Perineal Anesthesia ☐ local blocks ☐ lidocaine for episiotomy ☐ pudendal blocks
epidural • most commonly used technique for both labour and delivery • does not prolong first stage, but may reduce maternal expulsive efforts • 0.25% bupivacaine (Marcaine) usually used for labour (longer acting compared to lidocaine and less motor block) • 2% lidocaine (Xylocaine) usually used for vaginal deliveries and C-section in varying doses • 19 gauge indwelling catheter inserted into lumbar epidural space • preload mother with 500-1000 mL IV fluid to prevent maternal hypotension associated with epidural (fetal depression rare if maternal hypotension avoided) • test dose given first to check for spinal block followed by another dose to rule out intravascular injection - if no dizziness or tinnitus, rest of dose is given • complications: inadvertent total spinal with cardiovascular collapse and respiratory arrest, intravascular injection with seizures, post-ictal depression and possible cardiac arrest walking epidural • goal is effective analgesia with no motor blockade • 0.125% bupivacaine plus low dose fentanyl spinal block • for C-section need anesthesia of T4-T8 • injection of local anesthetic into subarachnoid space • fastest onset • least drug exposure for fetus because small dose required • not appropriate for labour due to intense motor blockade • beware of rapid hypotension and preload mother with 1000 mL IV fluid
General Anesthesia I not used for vaginal deliveries I rapid sequence induction to prevent aspiration I pre-oxygenate mother with 100% O ₂ as she is prone to hypoxia during intubation secondary to decreased FRC and increased O ₂ consumption

DEFINITION □ period of adjustment after pregnancy when anatomic and physiologic changes are reversed □ immediate - first 24 hours after delivery □ early - first week □ traditionally, puerperium lasts 6 weeks
POST-DELIVERY EXAMINATION ☐ The 8 B's: Blues (post-partum), Breathing (DVT/PE), Breast, Belly, Bowels, Bladder, Bleeding, Baby
BREAST 2 events stimulate lactation • sudden drop in placental hormones (especially estrogen) • suckling stimulates release of prolactin and oxytocin □ colostrum secreted for ~ 2 days (contains protein, fat, minerals, IgA and IgG) • replaced by milk after ~ 3-6 days (contains protein, lactose, water, fat) □ breast-feeding encouraged (see Pediatrics Notes)
UTERUS ☐ through process of catabolism, uterus weight rapidly diminishes ☐ cervix loses its elasticity and regains firmness ☐ start oxytocin drip or give oxytocin 10 U IM after 3rd stage (i.e. after delivery of placenta; some give IM dose after delivery of head) ☐ generally should involute ~ 1 cm (1 finger breadth) below umbilicus per day in first 4-5 days ☐ involution then slows down; reaches non-pregnant state in 4-6 weeks postpartum
LOCHIA ☐ normal vaginal discharge postpartum ☐ monitored for signs of infection or bleeding ☐ normally decreases and changes colour from red (lochia rubra; due to presence of erythrocytes) to yellow (lochia serosa) to white (lochia alba; residual leukorrhea) over 3-6 weeks ☐ foul smelling lochia suggests endometritis
PUERPERAL COMPLICATIONS
RETAINED PLACENTA placenta undelivered after 30 minutes risk factors: placenta previa, prior C/S, post-pregnancy curettage, prior manual placental removal, uterine infection placenta separated but not delivered, or abnormal placental implantation • placenta accreta: placenta adherent to myometrium • placenta increta: invasion of myometrium • placenta percreta: invasion of myometrium beyond serosa increased risk of infection or bleeding management • 2 large bore IVs, type and screen • perform Brant maneuver (firm traction on umbilical cord with one hand applying pressure suprapubically to hold uterus in place) • oxytocin 10 IU in 20 mL NS into umbilical vein • manual removal if above fails • D&C if required
UTERINE INVERSION ☐ uterus prolapses through the cervix and passes out of the vaginal introitus ☐ often iatrogenic (excess cord traction) ☐ more common in grand multiparous (lax uterine ligaments)

 urgent management essential (may require general anesthetic if unsuccessful) replace uterus without removing placenta remove placenta manually and withdraw slowly IV oxytocin infusion re-explore uterus
POSTPARTUM PYREXIA
Definition ☐ any fever of > 38.0 °C on any 2 of the first 10 days postpartum, except the first day
Causes ☐ Wind
 atelectasis (especially after general anesthesia) pneumonia Water (UTI) Wound (gram +/-, aerobes, and anaerobes)
 C-section incision site episiotomy site empiric treatment: clindamycin + gentamicin prophylaxis against post-C/S endometritis begin antibiotic immediately after cord clamping
and administer only 1-3 doses • cefazolin is most common
 Walking pelvic thrombophlebitis (diagnosis of exclusion) DVT
 Breast engorgement may cause slight physiologic temperature rise on
first day • mastitis (Staphylococcus aureus most common) □ Endometritis • blood and genital cultures
POSTPARTUM HEMORRHAGE (PPH)
Definition □ loss of > 500 mL of blood after delivery (most women probably lose > 500 mL, often underestimated)
Etiology (4 T's) tone: uterine atony (most common cause of PPH) occurs within first 24 hours labour (prolonged, precipitous) uterus (infection, over-distension) placenta (abruption, previa) maternal factors (grand multiparity, GA) halothane anesthesia tissue: retained placenta (see above) trauma: laceration (vagina, cervix, uterus), episiotomy, hematoma,
uterine rupture, uterine inversion (see above) thrombin: coagulopathy most identified prior to delivery (low platelets increases risk) includes hemophilia, DIC, aspirin use, ITP, TTP, VWD (most common) maintain fibrinogen > 1000 mg/mL, platelets > 50 000
Management determine cause, call for help
 □ supportive ABC's, fluid, +/- transfusions, +/- other blood products □ examination reexamine patient, ensure complete delivery of placenta, check for uterine atony and drain bladder check for convical and various lacorations
check for cervical and vaginal lacerationselevate the uterus and massage through patient's abdomen

PUERPERAL COMPLICATIONS ... CONT.

☐ investigations
 pelvic U/S if indicated to look for cause medical oxytocin (5 U IV push then 40 u/L NS drip) methylergonavine maleate (ergotamine; 0.2 mg PO or 0.25 mg IM) (normotensive patients only; must explore uterus before giving ergotamine) prostaglandins (PGF-2 alpha intrauterine or IM) hemabate (prostaglandin; 0.25-1.00 mg intramyometrium every 15 minutes) uterine packing (3-4 five yard Kerlex rolls tied together and soaked in betadine and removed in 12-24 hours; controversial) surgical seek and suture lower genital gract lacerataions D&C (beware of vigorous scraping which may cause Asherman)
 but (beware of vigorous straping which may cause Asherman) hypogastric, ovarian artery or uterine artery ligation arterial embolization hysterectomy (last option) complications: Sheehan syndrome (pituitary necrosis)
POSTPARTUM MOOD ALTERATIONS □ postpartum blues • very common, 85% of new mothers • onset day 3-10 • considered an extension of the "normal" hormonal changes and adjustment to a new baby • self-limited, does not last more than 2 weeks □ postpartum depression • signs and symptoms of major depression occurring in a woman within 6 months of childbirth (see Psychiatry Notes) • incidence of 10-20% • suspect if the "blues" last beyond 2 weeks, or if the symptoms in the first two weeks are severe (e.g. extreme disinterest in the baby, suicidal or homicidal ideation) • treatment with antidepressants is often necessary • interferes with bonding and attachment between mother and baby so it can have long term effects □ postpartum psychosis • rare (0.2%) • presents as an acute psychotic episode, or can occur in the context of a depression
DRUGS CONTRAINDICATED IN PREGNANCY
 ☐ most drugs cross the placenta to some extent ☐ use any drug with caution and only if necessary ☐ Motherisk at the Hospital for Sick Children in Toronto is a valuable resource (416-813-6780)
ANTIBIOTICS safest = ampicillin, cephalosporins erythromycin • maternal liver damage (acute fatty liver) • used only if contraindication to penicillin use tetracyclines • staining of child's teeth sulpha drugs • antifolates therefore theoretical risk in first trimester • risk of kernicterus in third trimester metronidazole • antimetabolite therefore theoretical risk in first trimester

 chloramphenicol grey baby syndrome (fetal circulatory collapse secondary to accumulation since fetus cannot metabolize this drug)
OTHER DRUGS □ alcohol
 increased incidence of abortion and stillbirth, congenital anomalies, fetal alcohol syndrome (growth retardation, CNS involvement and facial anomalies)
 cigarettes decreased birth weight, placenta previa/abruption, increased spontaneous abortion, preterm labour and stillbirth
 □ anticoagulants • warfarin crosses placenta, heparin does not • fetal warfarin syndrome: nasal hypoplasia, epiphyseal stippling, optic atrophy, MR, intracranial hemorrhage • also spontaneous abortion, stillbirth, prematurity, IUGR □ ACE inhibitors
☐ anticonvulsants • facial dysmorphogenesis, IUGR, mild MR, NTD's, congenital anomalies
□ lithium • Ebstein's cardiac anomaly, goitre, hyponatremia □ cocaine
 microcephaly, growth retardation, prematurity, MR DES (and other estrogenic or androgenic compounds) vaginal adenosis, adenocarcinoma, uterine malformation in daughters exposed to DES in utero retinoids (e.g. Accutane)
IMMUNIZATIONS □ administration is dependent on the risk of infection vs. risk of immunization complications □ safe
• tetanus toxoid, typhoid fever (killed bacterial), diphtheria, influenza, hepatitis B
 avoid live vaccines —> risk of placental and fetal infection polio and mumps
 contraindicated rubella (see Antenatal Complications Section)
BREASTFEEDING AND DRUGS □ safe
 penicillins, aminoglycosides, cephalosporins oral contraceptive use (low dose) is now believed to be safe
 avoid chloramphenicol (bone marrow suppression) metronidazole (mutagenic in vitro) sulphonamides (hemolysis with G6PD deficiency) nitrofurantoin (hemolysis with G6PD deficiency) tetracycline (stains teeth and bones)
lithiumantineoplastics and immunosuppressantspsychotropic drugs (relative)

