Clinical Problems in Obstetrics: Differential Diagnoses

**Abdo pain in pregnancy:** consider uterine, gynae and other – miscarriage, preterm labour, ectopic, abortion, fulminating pre-eclampsia, impaction of uterus, polyhydramnios, degeneration of fibroid, uterine rupture, ovarian cysts (more likely to tort in pregnancy), UTI/pyelonephritis, surgical (renal stones, gallstones, appendicitis, Meckel’s diverticulum etc). Early pregnancy usually threatened miscarriage (esp if precedes bleeding).

**Bleeding in pregnancy** – miscarriage, ectopic, placenta praevia, abortion, vasa praevia, show, cervical lesion, trauma.

**Small for dates** – wrong dates, transverse lie, IUGR, oligohydramnios, fetal anomaly (e.g. chromosomal).

**Large for dates** – wrong dates, multiple pregnancy, polyhydramnios, hydatidiform mole, large baby e.g. diabetes, fetal anomaly (e.g. hydrocephalus).

**Maternal collapse** – hypovolaemic e.g. haemorrhage, septicaemic, cardiogenic e.g. PE, anaphylactic, neurogenic e.g. uterine inversion.

**Seizures** – pre-eclampsia, epilepsy, cerebral vein thrombosis, amniotic fluid embolism, meningitis, hypoglycaemia, hyponatraemic, cocaine use (mimics pre-eclampsia with proteinuria, HT and seizures).

Care in Pregnancy

**Antenatal care in low risk pregnancies**
Visits at 10 weeks, 20, 24, 28, 30, 32, 34, 36, 38, 40, 41 (though often less frequent).

**Detailed history at first booking visit** – confirm pregnancy, risk factors, age, parity, planned, smoking, drugs, allergies, alcohol, previous obstetric and medical history, LMP (and how sure, regularity of periods), social factors.

**Investigations:** First visit – BMI, blood typing, antibody screening, rubella antibody status, hepatitis B, syphilis, HIV. 16 weeks – biochemical screening, maybe NT scan. 20 weeks – detailed anomaly scan. Every visit – BP, urinalysis, uterine size and in later pregnancy fetal lie, presentation and engagement.

**Give advice** – information about pregnancy, social welfare, diet, smoking, alcohol, not eating unpasteurised dairy produce, not handling cat litter.

**Examination**
Symphysiofundal height (in cm should = no. of weeks), lie (transverse, oblique, longitudinal), presentation (cephalic – vertex, face, brow -, breech, shoulder), position (occiput for vertex, sacrum for breech, chin for face), engagement, amniotic fluid volume, tenderness, fetal heart.

**Fetal imaging**
USS – simple, with Doppler for blood flow, or 3D for surface abnormalities e.g. facial clefts. Use to diagnose intrauterine pregnancy, fetal abnormality, NT scan, ectopic pregnancy, multiple pregnancy, trophoblastic disease, to assess gestational age (crown rump length, BPD at level of faux cerebra), placental site, fetal presentation, fetal growth and liquor volume and to guide invasive procedures. Blood flow – umbilical artery (absence of forward or reversed end diastolic flow shows increased placental resistance and is associated with increased risk of perinatal death or complications), middle cerebral artery (increased in anaemia). Transvaginally can see gestational sac at 5wks and cardiac activity at 6 wks.
Fetoscopy – invasive, risky, only specific procedures e.g. laser ablation in twin-twin transfusion.
MRI – CNS abnormalities.

**Antepartum fetal health assessment**

**Low risk pregnancies** – clinical assessment of fetal growth, routine auscultation of fetal heart (with sonicaid or Pinard stethoscope) and maternal perception of fetal movements.

**High risk pregnancies** (previous obstetric or medical history or event during pregnancy e.g. HT, IUGR) – serial ultrasound for biometric parameters, liquor volume, biophysical testing, umbilical artery Doppler.

**Cardiff fetal activity chart** – record time by which 10 movements have been felt, may help with high risk pregnancies.

**Biophysical profile scoring**: fetus with chronic hypoxia has depressed CNS (reduced HR variability) and renal activity (oligohydramnios). Measure and score FHR (accelerations), movements, tone, breathing, liquor volume. Problem is that this doesn’t weight relative importance. Amniotic fluid volume – either maximum pool diameter or amniotic fluid index (sum of 4 vertical pools, one in each quadrant).

**CTG** - Mature fetus is quiet about 30% of time and active 70% (baseline variations and accelerations). DR C BRAVADO (Date, Reason for CTG, Contractions, Baseline Rate, Accelerations, Variability, Decelerations, Overall impression). Reassuring CTG – baseline rate of 110-160bpm (higher in hypoxia, maternal pyrexia, pain or fear, lower in hypoxia), variable baseline about 15bpm (reduced with fetal sleep, hypoxia, pethidine; sinusoidal rhythm is serious – fetal asphyxia, fetal anaemia), accelerations = rise in FHR of >15bpm for >15sec. Decelerations – early (begins with contraction and finished by end of contraction, due to change in pressure on fetal head), variable (often due to cord compression, 25% risk of hypoxia), late (after peak of contraction, placental insufficiency, 50% risk of hypoxia). Classify CTG as reassuring, suspicious (one abnormal feature) or pathological (>1 abnormal feature, try left lateral position, stop syntocinon, give oxygen, correct hypotension, if persists need FBS or delivery). CTG also shows freq but not strength of contractions.

**Prenatal diagnosis**

**AFP** – decreased in Downs. Raised in open NTD (>2.5 multiples of median has 90% sensitivity), ant abdo wall, defects, IUFD, intrauterine bleeding. Also raised with multiple pregnancy and wrong dates.

**Karyotyping, DNA analysis, enzyme assay, FISH** (count chromosomes in interphase cells to confirm major defects in 24hrs).

**CVS** – transabdominal or more rarely transcervical under US guidance. Aspirate villi for karyotyping, DNA analysis or enzymology. Not before 10wks due to risk of limb defects. Risks - miscarriage, about 1% (higher if earlier), membrane rupture, infection, rhesus sensitisation, uterine trauma.

**Amniocentesis** – insert spinal needle transabdominally under US guidance. After 1st trimester, usually 16 wks. For chromosome analysis, fetal sexing, enzymology, DNA, analysis of rhesus disease. Risks – miscarriage (about 1%), rhesus sensitisation, early gestations: respiratory distress, postural deformities; later gestations: PROM, chorioamnionitis, preterm labour.

**Cordocentesis** – from umbilical cord after 18wks. To investigate Hb disorders, viral infections, rhesus disease, unexplained hydrops or anaemia. Risks – miscarriage, death, trauma, blood loss, PROM, labour, rhesus sensitisation.

**Kleihauer test** – for presence of fetal blood cells in maternal circulation, can be quantitative. Uses fact that fetal cells are more resistant to acid.
**Genetic counselling** – establish diagnosis, magnitude of risk of transmission, severity of disease and treatment, parental opinions. Options – not to have offspring, ignore risk, antenatal diagnosis and termination, pre-implantation diagnosis, artificial insemination or ovum donation.

**Intrapartum fetal monitoring**

Liquor – should be clear. Meconium stained - may indicate hypoxia (also post dates or breech) so continuous FHR monitoring is indicated, aspiration at birth is a risk. Mild blood staining - may be a show, heavier may be placental abruption or praevia.

FHR monitoring – detect intrapartum hypoxia (which causes acidosis and FHR changes). Low risk pregnancies – listen every 15min in 1st stage and after every contraction in 2nd stage. High risk (IUGR, prematurity, breech, multiple, epidural, augmented labour, diabetes, hypertension etc.) or those with meconium or auscultated abnormalities need continuous monitoring with CTG. Can also use fetal scalp electrode.

FBS – to confirm hypoxia. Amnioscope is introduced through cervix to fetal scalp. Scalp stabbed with guarded blade. Normal pH >7.25, base excess <10. Pathological <7.20, needs delivery, serious long term morbidity is usually <7.1.

**Analgesia in labour**

Pain due to uterine ischaemia, cervical dilatation (sacral), perineal stretching (pudendal nerve).

Non-pharmacological – birthing ball, ambulation, supportive partner, water, relaxation, breathing, TENS (gate control theory), aromatherapy.

Pharmacological – entonox (nitrous oxide and oxygen), opiates e.g. pethidine (problems are vomiting and neonatal depression, but rapid).

Epidural – effective esp if breech or instrumental, L3/4/5. Use loss of resistance technique, infusion of e.g. bupivicaine and fentanyl. Need to monitor BP, respiration, FHR and level of block and empty bladder. Complications – dural puncture, total spinal block, hypotension, paralysis.

Pudendal nerve block – nerve runs under ischial spine. Use pudendal needle (like spinal needle but with guard so can put up vagina). Aspirate as near vascular bundle and if no blood inject lignocaine. Should block lower 1/3 of vagina and skin.

**Interventions and procedures**

External cephalic version – for breech presentation. Manipulate through ant abd wall to cephalic under ultrasound with tocolysis at about 38 wks (later so good prognosis if have to do emergency section for complications, less chance of reverting and chance to revert spontaneously). Success about 50% (lower in primips), very safe, if successful high chance of successful vaginal delivery. Complications – cord entanglement (don’t do if cord round neck), abruption – all are very rare and checked by long CTG after. Contra-indications – cord round neck, previous C section scar, fibroids, oligohydramnios, other complications in pregnancy.

24 hour urine collection – empty bladder. From then on collect all urine until 24 hrs have passed. At end of 24 hrs empty bladder again and add to collection.

Steroids e.g. betamethasone – give if likely to be preterm delivery or if likely to need to induce early for maternal or fetal compromise. Help to promote fetal lung maturity so reduce neonatal morbidity. Take 48 hrs to work, need 2 doses 24 hours apart. Current evidence suggests only giving one course.
**Tocolysis** – usually just to allow time for steroids to work. Atosiban (oxytocin antagonist), ritodrine or salbutamol (beta sympathomimetics), nifedipine. Contraindicated in thyroid disease, cardiac disease, chorioamnionitis, intrauterine death, relatively in advanced labour and APH.

**Episiotomy** – to avoid inevitable severe perineal tear, to expedite delivery if fetal distress in 2nd stage, for most forceps and breech deliveries. Cut posterolaterally from fourchette (to minimise risk of extending into anal sphincter) during a contraction. Complications – discomfort, haemorrhage, haematoma, infection, dyspareunia (if tight repair).

**Induction of labour** – when delivery will be safer for mother or fetus e.g. maternal disease (e.g. pre-eclampsia, diabetes), fetal disease (e.g. rhesus disease), placental insufficiency, post-maturity, fetal death or anomaly.

Absolute contraindications – non-longitudinal lie, obstruction to vaginal delivery, history of USCS or uterine rupture. Relative – grand multiparity, LSCS, breech, prematurity.

Methods: (membrane sweep – releases PGs, 70% of woman at term will go into labour within 48hrs), prostaglandin E2 if cervix not favourable (usually vaginal pessaries or gel), amnihook for AROM (may be all needed if cervix favourable), oxytocin infusion (if PG and ARM fail to cause uterine activity and dilatation of cervix).

Bishop score – indicates favourability of cervix, up to 13 based on: dilation, consistency, length, position, engagement of head. High score is favourable – dilated, soft, short, anterior, low head. With a favourable cervix (>6), failed induction rate is <1%, about 5% if unfavourable.

Complications – iatrogenic prematurity, uterine hyperstimulation (>5 contractions/10min), infection, failure, increased risk of uterine rupture after LSCS (1.5% with ARM and synto, 3% with prostin compared to 0.4% with spontaneous labour). For dead fetus then extra-amniotic E2 and antiprogesterone.

**Forceps** – need full dilatation of cervix, adequate analgesia (epidural, spinal, pudendal block), empty bladder, known position of fetal head, no fetal head palpable per abdomen, no cephalopelvic disproportion.

Indications: maternal conditions that make pushing undesirable (e.g. cardiac disease, hypertension), fetal compromise, cord prolapse, poor progress (e.g. due to maternal exhaustion or OP position), delivery of head in breech.

Either non-rotational (e.g. Neville-Barnes, cephalic and pelvic curves, for OA position), little (Wrigley forceps – now mainly used at C section) or rotational (e.g. Kielland’s, only have cephalic curve, so can rotate to convert transverse or OP to OA, handles slide to allow for asynclitic head). Generally need episiotomy. Fetal trauma mainly marks or facial nerve palsy, various others if applied incorrectly.

**Ventouse delivery** – conditions as for forceps, also need maternal effort (i.e. ability to push and contractions). Various different kinds of suction cups, can get additional part to allow rotation of OP or use Kiwi ventouse (as small so will fit posteriorly in vagina so can be applied to vertex). Apply to fetal head on vertex in front of post fontanelle (so flexes head), create vacuum (checking no vagina or cervix caught) and apply traction with maternal pushing and contractions (usually only 3 times). Complications – scalp abrasions, chignon, cephalhaematomas, rarely IC haemorrhage, tentorial tears and scalp necrosis, vaginal trauma if trapped.

Compared to forceps – less analgesia, less maternal trauma (as don’t increase area), more fetal trauma, may not work as well with malrotation, higher failure rates, not for face presentations or preterm (as increased risk of haematoma).

**Caesarean section** – rate about 20% in UK, rates increased due to epidural anaesthesia reducing anaesthetic risks, increased use of fetal monitoring, deskilling, evidence of lower PNM for breech babies, increasing demand from women.

Indications: Elective – recurrent cause (e.g. CP disproportion), >2 previous CS, breech, placenta praevia, maternal disease, maternal choice (a first elective section still has higher mortality than vaginal delivery), multiple pregnancies.
Emergency LSCS—fetal distress, cord prolapse, obstructed labour (e.g. fibroid), prolonged labour (e.g. dysfunctional uterine activity, malposition, CP disproportion), bleeding from placenta praevia, maternal risk. Lower segment C section (incision into part of uterus that doesn’t contract in labour and is covered by a full bladder) – most common, lower risk of rupture (0.4%), wound is extraperitoneal so less risk of peritonitis. Upper segment – vertical incision in upper uterus, greater blood loss, higher risk of rupture in subsequent labour (6%). Used for transverse lie, uterine abnormalities, placenta praevia, some low birthweight babies with oligohydramnios (poorly formed LS). Repair uterus in 2 layers. Complications – haemorrhage, infection, thromboembolic disease, paralytic ileus, damage to bladder increased risk of CS and placenta praevia in future pregnancy, complicate later gynae conditions e.g. adhesion of uterus to bladder, mortality 1:3000.

Anaesthetics in pregnancy – main risk is aspiration of stomach contents. Prevent by empty stomach, giving sodium citrate or H2 blocker, cricoid pressure and head up, cuffed endotracheal tube.

Therapeutic abortion – certification of 2 doctors. 1967 abortion act. Indications – pregnancy <24 weeks and risk of injury to physical or mental health of woman (usually this one – 94%, clause C) or existing children, risk to life of pregnant woman, to prevent grave permanent injury to physical or mental health of pregnant woman, substantial risk that child would be seriously handicapped. Before need to determine gestation, FBC, rhesus status and give counselling. Long term sequelae – psychological morbidity, infertility (mainly related to infertility so give prophylactic antibiotics against chlamydia, gonococcus and BV), ectopic pregnancy and cervical incompetence. Early medical TOP - <9 wks, mifepristone (anti progesterone, sensitises myometrium and softens cervix) followed by PV PG pessary (e.g. gemeprost, misoprostol). Surgical - <14 weeks, dilatation and suction aspiration. Late medical - >14 weeks oral mifepristone with repeated PG pessaries, consider feticide if >20 weeks, intracardiac injection of KCl. Risks are infection, trauma and haemorrhage.

Normal Pregnancy, Delivery and Pueperium

Fertilisation in outer third fallopian tube. 4 cell stage at 36-48 hr. Blastocyst arrives at uterus at 72hrs with 16 cells (outer trophoblast forms chorion, inner cell mass forms fetus). Implants at 6 days. Gestation sac on vaginal USS at 5 weeks after 1st day of LMP. Organogenesis complete by 8 wks. EDD is 280 days after 1st day LMP (assumes conception at 14 days), date pregnancy from 1st day of LMP. Can estimate by crown rump length (+/- 5 days) or biparietal diameter on USS, more precise if early.

Symptoms of pregnancy – amenorrhoea, fetal movements. Physiological changes in pregnancy cause various others – GI – nausea (80% of primips, usually only for 1st trimester), vomiting, oesophagitis, constipation, haemorrhoids, gum hypertrophy; urinary frequency (due to increase in RBF and pressure); breast tingling, enlargement and pigmentation, increased skin pigmentation (choasma, linea nigra, umbilical pigmentation), umbilicus eversion, varicose veins and striae; cardiorespiratory – palpitations, breathlessness.

Signs – cervical softening, nipple pigmentation, enlarged uterus (palpable above pubis at 12 weeks, umbilicus at 22, xiphisternum at 36), fetal heart heard (14 weeks).

Investigations – urine test for bHCG (by 1st day of missed period), vaginal ultrasound (sac at 6 wks, heart at 7).
**Fetal development**

3rd week - heart begins to beat.
4th week – neurulation, neuropores close, limb buds, eye and ears start to form. Development of lungs, liver, GI tract and kidneys starts.
2nd month – face and neck start to form, limb buds elongate, muscles and cartilage develop, sex organs begin to form.

**Labour**

*Partogram* – fetal condition (fetal heart rate – every 15min, colour of liquor), progress of labour (cervix dilatation, descent of head – abd in fifths, or vaginally relative to ischial spines, strength and freq of contractions, drugs given to augment labour) and maternal condition (well being, pulse, BP, temp, drugs).

**Contraction**s – should be fundally dominant passing down to lower segment, if they are not then activity is dysfunctional. Muscle fibres contract but don’t return to original length. Upper segment thickens and heaps up, lower segment becomes thinner and stretched.

**First stage** – onset of labour (retrospective diagnosis – progressive cervical effacement, shortening and softening to merge with lower segment, and dilatation and regular painful contractions) to full dilatation of cervix. Often have a *show* = passage of blood stained mucus plug. Latent phase = until about 4 cm dilated and fully effaced, about 9h in primips and 5 in multips. Active phase = to full dilatation, 5h in primips, 2 in multips – should dilate at least 1cm/h and pp descend. Contractions should be about 3 in 10. Generally assess dilatation and descent of pp by VE every 4 hours.

**Second stage** – from full dilatation to delivery, 40 min in primips, 20 in multips, assess if no delivery within an hour, probably need assisted delivery. Woman feels urge to push, see anus dilatation and perineum bulges. Pushing is by abdominal wall muscles and fixed diaphragm to raise intra-abdominal pressure, this is not essential for delivery. If epidural often give a passive hour before starting pushing. Crowning is when introitus is maximally stretched. Prevent rapid decompression of fetal head and uncontrolled tearing of perineum, check for cord around baby’s neck. *Apgar score* records condition of baby at birth (colour, cry, tone, breathing, pulse).

**Third stage** – until delivery of placenta. Active management (less blood loss) – im injection of oxytocin with delivery of ant shoulder, clamping and cutting cord and assisted delivery of placenta when signs of separation (contraction of uterus, gush of blood, descent of cord) by controlled cord traction (need to support uterus to avoid uterine inversion). Alternative is physiological management – more primary postpartum haemorrhage. Check placenta – 3 vessels (2 arteries), 2 membranes, complete. Uterus contracts and constricts blood vessels to prevent bleeding.

**Control of labour** – not totally understood. Oestrogen increases uterine muscle activity, progesterone suppresses. Fetal adrenals produce DHEAs in late pregnancy which placenta converts to oestrogen. Decidua releases PGs causing contractions. Oxytocin is release when pp presses on pelvic floor.

**Mechanics of delivery**

Flexion of fetal head. Head engages (usually ROT) into wider transverse diameter of inlet (transverse is 13cm, AP is 11cm), descend to pelvic floor then turns (internal rotation) to OA as wider diameter of outlet is AP (13cm compared to 11). Head is born by extension. External rotation (restitution) so shoulders are AP. Lateral flexion of head for delivery of anterior shoulder. Caput and moulding.

**Puerperium**

= time taken to return to the normal non-pregnancy state. Typically taken as 6 weeks. Need to be seen daily for 2 wks to check BP, temp, scars, breasts, signs of thrombosis, lochia (loss from vagina) and involution of uterus.
Complications – puerperal sepsis, perineal pain (bruising, oedema, infection) – analgesia and bathing help, vaginal haematomas – need surgical evacuation, haemorrhoids (prolapse at delivery), sub conj haemorrhage (asymptomatic and resolve), wound infection, endometritis, thromboembolic disease, mastitis. After pains – variable, may need analgesia.

Contraception after pregnancy
Non-lactating women can ovulate within 4 wks of delivery, on average return of menstruation at 2 months. Rare for breastfeeding women to ovulate, but not a recommended method alone. Can commence COCP after 3 wks (not before due to risk of thrombosis), avoid COCP in breastfeeding as oestrogen may suppress lactation. POP can be started immediately and doesn’t affect lactation but can cause annoying bleeding. IUD – usually at 6 weeks, due to risk of expulsion or perforation if earlier. Can perform sterilisation at C section (though maybe higher failure rates).

Breastfeeding
Milk is synthesised by alveolar epithelium. contains lactose, casein and triglycerides. Colostrum has more salt, proteins, Ig and lactoferrin.
Breast feeding helps mother lose weight, protects baby against disease, promotes maturation of GI tract and speeds involution via oxytocin.

Anatomy and Physiology

Bony pelvis diameters:
Inlet – AP 11, oblique 12, transverse 13.
Middle – 12, 12, 12.
Outlet – AP 13, oblique 12, transverse 13.
Vertex presentation (suboccipitobregmatic) – 10cm
Flexed cephalic presentation (suboccipitofronal) – 11cm
Face presentation (submentobregmatic) – 10cm
Brow presentation (mentovertical) – 13cm.

Coagulation cascade
Intrinsic (slow, APTT, starts at XII), extrinsic (quick, PT, starts at VII), final common pathway (TT, V and X to II and fibrinogen). In pregnancy factors VII, VIII, X and fibrinogen increase, so APTT, PT and TT fall and increase risk of DVT (x6). In pre-eclampsia may be DIC.

Normal physiology of pregnancy
CV – CO increases by 30-40% due to increase in HR and SV. Blood volume increase, mainly due to plasma so some haemodilution (10-11g/dl Hb is OK). Blood pressure drops in 1st half then begins to rise to pre-pregnancy levels. PVR falls. Inverted T waves. Increased flow in the uterine a and also to kidneys and beast tissue. This can lead to oedema (also due to compression of venous return and low albumin), fainting and postural hypotension, flushing, palpitations and breathlessness (mechanical due to diaphragm not moving, less reserve, should not be at rest or orthopnoea).

GI – constipation (prog stops smooth muscle contracting), reflux (stomach pushed up and sphincters relaxed, advise antacids and small meals), haemorrhoids (increased venous pressure and constipation), sickness (may be related to ketosis).

Renal – increased RBF and GFR (by up to 50%). Increased creatinine clearance (decreased creatinine and urea levels), decreased serum urate (pre-eclampsia if >10x no. of weeks), lose some protein (accept up to 0.5g/24hr) and glucose. Dilatation of pelvis and ureters (obstruction of outflow and relaxation of smooth muscle) increases risk of ascending infection (major cause of preterm labour) so take UTIs seriously.

LFTs – increased alkphos (isoenzymes from bones and placenta), ALT and AST in low normal range, albumin falls (haemodilution and loss in urine).
Blood clotting – all major clotting factors increase, so increased risk DVT and PE (exacerbated by venous stasis). Decreased clotting times.

Hormonal – trophoblast secretes HCG (prolongs corpus luteum), corpus luteum produces progesterone, placenta produces HCG, human placental lactogen, prolactin, progesterone, oestriol, prostaglandins. Progesterone – glandular development, decreased myometrial activity, decreased smooth muscle tone (also effects GI system, ureters and veins). Oestriol causes uterine growth, softening of ligaments. HPL – alters glucose and insulin metabolism, may initiate lactation.

Glucose metabolism – lower fasting blood glucose as fetus uses glucose. Peripheral resistance to insulin due to HPL, cortisol, sex steroids.

Weight gain – about 10kg due to fat and fluid (half), fetus, placenta and amniotic fluid (normal range at term 500-1500ml). Rapid gains in late pregnancy may indicate fluid retention due to pre-eclampsia.

Placenta: umbilical cord insertion - battledore, succenturiate lobe (placental vessels in membranes), velamentous insertion (cord vessels in membranes), bipartite (2 areas).

Abnormalities of Pregnancy and Childbirth

Miscarriage
Spontaneous loss of pregnancy before 24 weeks, where fetus shows no sign of life after delivery. 20% of detected pregnancies, mostly first trimester.

Classification: Threatened – painless bleeding, viable fetus, os closed (75% settle spontaneously). Inevitable – os open. Incomplete – some products of conception expelled. Complete – all expelled. Delayed or missed – fetus dead but not expelled (need ERPC or PG). Blighted ovum – anembryonic pregnancy, only gestation sac can be seen.

Aetiology – mostly due to chromosomal defects. If in 1st trimester only investigate if 3 consecutive. Other causes – immunological, uterine abnormality e.g. fibroids, cervical incompetence (2nd trimester losses), endocrine, acute maternal illness esp febrile, maternal disease (esp SLE, thrombophilia – give low dose aspirin), hypothyroidism, infection, toxins, trauma.

Management – present with amenorrhoea, vaginal bleeding and maybe pain, shock (if products in cervical canal) or passage of products or by scan. If os closed then USS to determine if viable fetus. No proven treatment if threatened. Management of others – conservative, medical (antiprogestogens) or surgical evacuation of uterus.

Recurrent miscarriage (=3 or more consecutive) – most common causes are antiphospholipid syndrome (anticardiolipin and antiphospholipid antibodies), PCOS, cervical incompetence, abnormality of uterus, genetic (e.g. balanced translocation). Treat APL syndrome with heparin and low dose aspirin and cervical incompetence with cervical suture at 16 weeks. If coming for advice – establish when miscarriages occurred, family history, general health, medications.

Chances of future successful pregnancies – after 1 miscarriage – 85%, after 2 – 75%, after 3 – 60%, if APS with treatment – 40%.

Ectopic pregnancy
Implantation outside of uterine cavity, usually ampulla of fallopian tube, also abdominal, ovarian, cervical (more serious as hard to operate and more prone to haemorrhage). About 1% of pregnancies. Leads to intraperitoneal or intratubal bleeding.

Aetiology: caused by factors which decrease embryo transport – PID, salpingitis, tubal scarring, use of coil, assisted reproduction techniques, POP (decreases tubal transport).
Outcome – can be absorbed (at early stages), tubal abortion (expelled to peritoneal cavity), tubal rupture (acute intraperitoneal haemorrhage). Presents with amenorrhoea followed by unilateral pain and bleeding. Usually have cervical excitation, unilateral tenderness and mass. If tubal rupture, also shock and rigid abdomen.

Management – USS (may show sac in tube, otherwise empty uterus, blood in pouch of Douglas), hCG assay, cross-matching. With hCG over 1500IU/l should see intra-uterine gestation sac on vaginal USS and should rise by 66% in 48hr. If hCG not falling need laparoscopy – see distended tube filled with blood. Normally laparoscopic salpingectomy (esp if other tube normal as reduces risk of future ectopics and persistent trophoblast), otherwise salpingotomy. If in shock then laparotomy. After surgery, repeat ectopic rate is 20% and intrauterine pregnancy is 50%. Medical treatment with methotrexate for compliant women with minimal symptoms and small ectopics.

Trophoblastic disease

Symptoms – irregular bleeding in 1st trimester with vesicles, LGA soft uterus, no fetal heart and exaggerated symptoms of pregnancy e.g. hyperemesis, high HCG. Possibly early pre-eclampsia. HCG has TRH like activity and can cause hyperthyroidism. Snowstorm appearance on USS and high bHCG. Tissue has hydropic vesicles. Often diagnose at curettage for presumed incomplete miscarriage.

Management – evacuation of vesicles with gentle suction or hysterectomy, CXR to check for lung mets and follow up to ensure bHCG disappears (6 months if go within 6 weeks, don’t conceive during this), if persists may be incomplete evacuation or malignant change.

Choriocarcinoma – 10% of moles are malignant, treat with metrotrexate (very effective), hysterectomy rarely indicated.

Congenital infections
Problem is that many sequelae are not detectable by USS (e.g. deafness, blindness, mental retardation). Can detect fetal infection by cordocentesis but not severity.

Rubella – rash, fever, malaise, conjunctivitis in mother. 90% risk of damage in survivors of 1st trimester infection – microphthalmia, microcephaly, cardiac defects, growth restriction, congenital cataracts. 20% sensorineural deafness in 2nd trimester. If born with active infection have encephalitis, jaundice, hepatosplenomegaly. Most mother should be vaccinated, screen for antibodies, those who are negative get vaccine after delivery.


Syphilis – highest risk of fetal infection with early disease (90% with primary and secondary). 25% fetal loss, 25% growth restriction, 50% congenitally infected neonate. On USS – hydrops, polyhydramnios, hepatomegaly. Newborns may be asymptomatic or have early syphilis (rash, lymphadenopathy, hepatosplenomegaly).

CMV – asymptomatic mother. Reactivation has minimal risk. 30% of fetuses affected of which 10% are symptomatic (thrombocytopenic purpura, chorioretinitis, microphthalmia, microcephaly, deafness, mental retardation). Cells have inclusions.


**Listeriosis** – risk factors are unpasteurised dairy products and raw vegetables. Congenital infection only with heavy colonisation – miscarriage and stillbirth, diffuse septicaemia (90% mortality, early onset disease) or meningitis and mental retardation (late onset).

**HIV** – all pregnant women offered testing. Without interventions vertical transmission is about 20%, with interventions (antiretroviral drugs, elective C section, avoid breast feeding) <2%. If no AIDS then doesn’t affect pregnancy.

**Herpes** – primary infections need acyclovir for 5 days and in late 3rd trimester to prevent recurrence, deliver by C section to prevent neonatal infection. Risk negligible with recurrences due to passive immunity.

**Group B strep** – main cause early onset neonatal sepsis. 20% women have vaginal colonisation.

**Chlamydia and gonorrhoea** – can cause conjunctivitis, arthritis, meningitis and septicaemia. Treat with appropriate antibiotics.

**Drugs in pregnancy**
Teratogens have most damaging effects between 4 and 8 wks (late embryonic period). Before implantation, insults cause embryo to die or survive intact as cells are totipotent. After this (foetal phase), teratogens affect overall growth or the size and function of a specific organ rather than causing structural abnormalities.

Teratogens may be drugs, diseases or physical factors e.g. hyperthermia.

**Prescribed drugs** – particularly teratogenic: antiepileptics (NTD and facial clefts), warfarin (damage to cartilage and heart about wk 5), methotrexate, lithium (Ebstein’s anomaly of tricuspid valve). Avoid any drugs in 1st trimester if possible. Later development – tetracyclines cause staining, antithyroid drugs cause goitre, warfarin causes fetal bleeding, indomethacin closes ductus arteriosus (NSAID, can be used therapeutically), beta blockers may affect placental function. Neonatal depression with 3rd trimester drugs e.g. narcotic analgesia.

**Smoking** – mean birth weight reduced by 250g. Fetal growth restriction, preterm labour, PROM, abruption, sudden infant death, increased thrombotic risk.

**Alcohol** - >20u/wk is a problem – fetal alcohol syndrome (intellectual impairment, growth retardation, characteristic face – micrognathia, hypoplastic filtrum, saddle shaped nose, maxillary hypoplasia).

**Substance abuse** – complications are commoner and increased risk of hep B and HIV. Neonatal withdrawal symptoms. Cocaine mimics pre-eclampsia.

**Vit A** – excess levels cause teratogenesis so avoid eating liver.

**Rhesus disease**
Principle group determined by D locus. Rh disease when Rh- mother (15%) has Rh antibodies, which act against Rh+ baby causing haemolysis (anaemia, jaundice, cardiac failure, hydrops fetalis, hepatosplenomegaly). Less severe disease with other antigens.

**Sensitisation** – exposure to Rh antigens leads to production of antibodies. Prevent by giving anti-D immunoglobulin at potentially sensitising events (delivery, abortion, ectopics, APH, amniocentesis, ECV) with positive Kleihauer test. No. of fetal cells guides amount (500iu for 4ml).
Management – screen Rh- mothers for antibodies. If no antibodies – screen for antibodies at 28 and 34 weeks, at delivery take cord blood for group, Coombs test (to see if cells coated with antibodies) and maternal blood (Kleihauer test for fetal cells). Can assess rhesus status of baby by maternal blood test. If mother has antibodies – assess disease severity by amniotic fluid optical density difference (amount of bilirubin), fetal Hb, middle cerebral a blood flow and antibody titre. Mild disease – deliver at term and phototherapy. Moderate – induce early. Severe - intrauterine blood transfusion. Postnatally take cord blood for Hb, Coombs test and bilirubin. At birth baby may have respiratory distress due to prematurity, pulmonary oedema or hypoplastic lungs (secondary to pleural effusions). Baby may need exchange transfusions after birth for anaemia or hyperbilirubinaemia.

Hypertension and pre-eclampsia

Hypertension → 140/90 or by >30/15 above booking values. If pregnancy-induced usually occurs after 20wks, if presents at booking is not pregnancy induced (investigate for secondary causes). For pre-existing hypertension, change to labetalol, methyldopa or nifedipine (ACE inhibitors and diuretics should be avoided), check for placental disease and super-imposed pre-eclampsia (increased risk) and induce at 38wks if severe. Don’t use ergometrine at 3rd stage as this can exacerbate hypertension (syntocinon should be used instead). Only tend to treat pregnancy-induced hypertension if >160 systolic or >120 MAP (provided no proteinuria, fetal or maternal compromise etc.) due to risk of cerebral haemorrhage and accelerated placental degradation and abruption (30% fetal loss). Emergency BP control is with IV hydralazine or labetolol. As long as controlled and no pre-eclampsia does not pose risk to mother or fetus in itself.

Pre-eclampsia = pregnancy induced hypertension with proteinuria (>0.3g/24hr without UTI, + represents about 0.5g/l, ++ 1g/l and +++3g/l). 5% of pregnancies.

Pathology – multisystem endothelial dysfunction and excessive inflammatory response to pregnancy: peripheral (oedema and hypertension), cerebral (vasoconstriction causes eclamptic seizures), renal (proteinuria and decreased urine output, also due to decreased intravascular volume), liver (abnormal LFTs, epigastric pain), lungs (ARDS), placenta (hypoperfusion and IUGR).

Risk factors – primigravidity, previous pre-eclampsia, FH, pre-existing HT, renal disease, migraine, diabetes, extremes of age, any pregnancy with large placenta (diabetes, multiple, mole, hydrops).

Aetiology – caused by pregnancy and cured by delivery of placenta. Thought to be due to abnormal placentation (trophoblast cells fail to invade and convert spiral arteries to vascular sinuses so placenta is poorly perfused). Change in PG:thromboxane ratio to favour thromboxane causes vasoconstriction and platelet agglutination leading to increased peripheral resistance and decreased fetal perfusion. This causes poor fetal growth and signals from the placenta (e.g. fatty acids, lipoproteins, TNF, syncytiotrophoblast fragments) cause maternal endothelial cell dysfunction. This leads to DIC, organ hypoperfusion (vasospasm) and plasma volume loss (increased permeability). Affected by maternal (inflammatory response and placentation) and fetal (placentation) genes.

Symptoms and signs – hypertension (may be labile due to vessels spasming), proteinuria, renal impairment, headache, visual disturbances, epigastric pain (indicates severe pre-eclampsia, prob liver capsule distension), oedema (facial is particularly significant), brisk reflexes, clonus, fetal growth restriction. Progress is very unpredictable, usually develops after 28wks.

Risks to mother – can develop to eclampsia (cerebral oedema and vasospasm cause hypoxia and fits), DIC, liver capsule distension, pulmonary oedema, abruption, cerebral haemorrhage, renal or liver failure, HELLP (haemolysis, elevated liver enzymes, low platelets). Most of deaths were from intracranial haemorrhage suggesting inadequate BP control.

Risks to fetus – IUGR, IUFD, abruption, iatrogenic prematurity.
Notes on Obstetrics.  Author: Liz Tatman

Management – admit and monitor BP (if >160 need to give drugs to prevent cerebral haemorrhage, generally IV hydralazine, but will not treat pre-eclampsia itself), CTG, fetal growth and wellbeing, bloods (FBC, U&Es, urate - >10xno. weeks is high - , LFTs, platelets – fall, if low need clotting screen), urine protein. Pre-eclampsia is only resolved with delivery. If >34wks and compromised induce. Often worse or unstable in first 48 hours post delivery then resolves. If <34wks – close inpatient monitoring, give steroids (will prob need to deliver baby early), consider antihypertensives and anticonvulsants (mag sulphate, also antihypertensive, main risk is respiratory suppression). Prob deliver by C section – generally use slow epidural (risk of spinal causing severe hypotension) and clotting screen first. Fluid balance – have low urine output and low CVP but should fluid restrict as fluid is extravasated to lungs and brain. Do not give diuretics, even though oedema, as worsens intravascular fluid depletion. Fitting – manage airway, turn on side, give oxygen, give diazepam if fit is not self-limiting, give mag sulphate, stabilise and deliver.

Diabetes mellitus
Maternal risks – increased insulin requirements, hypoglycaemia (and often lose warning signs, esp if good control as less far to fall, ensure family know how to treat), DKA (50% neonatal mortality), worsening of retinopathy (associated with good control), pre-eclampsia (esp if already have renal complications, renal disease can mimic this with hypertension and proteinuria), UTIs, miscarriage, traumatic delivery and high C section rate (50%).
Fetal risks – increased risk of malformations (x3, esp cardiac and NTD, sacral agenesis is almost exclusively found in diabetics), intrauterine death, IUGR, prematurity (iatrogenic), macrosomia (insulin has GF like action), polyhydramnios, abruptio, prolonged labour, traumatic delivery e.g. shoulder dystocia, hypoglycaemia, jaundice (produce lots of RBCs which are then broken down), RDS, PNM.

All reduced by good control. High maternal blood glucose crosses placenta and is laid down as fat (macrosomia). Neonatally, hyperinsulinaemia causes reactive hypoglycaemia.

Management: Prepregnancy – high dose folic acid and improved glucose control to reduce risk of congenital abnormalities. There is concern about teratogenicity of oral hypoglycaemics, but hyperglycaemia is also teratogenic so this may be confounded and probably should still take until confirmed pregnancy. Need to know duration of diabetes, glycaemic control. Treatment and secondary complications. Antenatally – close control of blood glucose (regular meals including late supper, high fibre carbs, multiple injections of insulin) and regular monitoring (7 point profiles twice weekly, regular HbA1c), switch to insulin (avoid oral hypoglycaemics as cross placenta) and review other medication (e.g. ACE inhibitors), regular BP and ophthalmoscopy, fetal screening (for wellbeing, use NT scan not serum screening for Downs), regular scans for growth, close monitoring for complications (e.g. UTI, pre-eclampsia). Delivery – induce at 39 weeks due to late unexplained IUFD, IV insulin and glucose infusion, elective C section for macrosomic babies, use steroids with caution in hospital (hyperglycaemia).

Gestational diabetes – usually after 24wks, more like type 2 diabetes (increased insulin resistance – increases anyway in pregnancy with gestation due to HPL, cortisol and sex steroids, if susceptible crosses threshold and become diabetic).
Risk factors – glycosuria, FH of diabetes, previous macrosomia, obesity, polyhydramnios.
Management – as DM but don’t have risk of early complications (abnormalities, microvascular disease), 50% risk of developing type 2 diabetes in later life. Test for glycosuria at every visit, random blouse if glycosuria noted and at booking visit, GTT in high risk groups or if random glucose raised.

Maternal disease
Effect of disease and treatments on pregnancy, effect of pregnancy on disease (e.g. worsen heart failure, improve RA due to higher steroid levels), alteration in presenting symptoms and signs (e.g. some signs that would be abnormal in the non-pregnant woman are normal in pregnancy), effects on the neonate (e.g. metabolic problems, effect of autoimmune antibodies e.g. thyroid stimulating, antiplatelet, antiRo – heart block).
Anaemia – lower limit of normal is 11g/dL in first trimester and 10.5g/dL after 28wks due to haemodilution. Anaemia may be due to haemodilution, iron deficiency, or more rarely folate deficiency, B12 deficiency, infection or haemoglobinopathies. Investigate type by FBC. Prevention – supplementation with 100mg elemental iron (and vit C to enhance absorption) and 300mg folate per day is controversial. No need to treat mild physiological anaemia, treat deficiencies with supplements (GI disturbance, anaphylaxis if parenteral, continue 3 months pp), very rarely with transfusion. Importance is doubled perinatal mortality and haemorrhage becomes life-threatening.

Venous thromboembolic disease – leading cause of maternal mortality in UK. Risk factors – pregnancy (5x more common - increased coagulation factors, reduced fibrinolysis and endogenous anticoagulants, venous stasis), immobility, obesity, age, dehydration, hyperemesis (via dehydration and immobility), pre-eclampsia, operative delivery, family history. Can present as DVT (swollen, warm, painful lower leg), PE (chest pain, haemoptysis, SOB, tachycardia, collapse), rarely venous sinus thrombosis (headache, vomiting, reduced LOC). Diagnose by imaging (venous Doppler of lower leg, VQ scan, angiography, MRI). Compared to non-pregnancy DVTs, more occur on left (90%, due to compression of L iliac vein) and more likely to embolise. Subcut injections of LMW heparin to treat and prevent e.g. after C section (but increases risk of haemorrhage), rarely inf vena cava filter. Screen for thrombophilia, esp factor V Leiden.

Thyroid disease – can get a small goitre, tachycardia, heat intolerance in normal pregnancy. Thyroid binding capacity is increased so although total levels of T3 and T4 increase, free levels are usually within normal range. Hypothyroidism (usually already diagnosed as anovulatory infertility) – if untreated increase in stillbirths. Hyperthyroidism – if untreated 50% fetal mortality, can treat with carbimazole but at lowest dose possible (but may cause fetal hypothyroidism and goitre, not teratogenic but can cause small defects in skin), can get neonatal hyperthyroidism if thyroid stimulating antibodies cross placenta (monitor fetal heart rate and scan for goitre), risk of ‘thyroid storm’ if uncontrolled (hyperpyrexia and tachycardia, AF).

Epilepsy – all anticonvulsants have a small risk of teratogenicity (7%) in first trimester (NTD, congenital heart disease, cleft lip and palate and various dysmorphic features), however, risk of fit to pregnancy > medication, especially after 7 weeks as neural tube is closed by then. Take higher dose folic acid, attempt to reduce to monotherapy, offer fetal cardiology scan, be aware of enzyme inducing effects (e.g. prescription of steroids, neonate may need vitK), monitor plasma anticonvulsant levels (pregnancy may affect and compliance). Pregnancy does not affect seizure rate, though labour is a risk (stress and sleep deprivation). Breast feeding is not contraindicated, though care should be taken to avoid harm if a seizure occurs.

Fibroids – miscarriage, increased pressure symptoms, malpresentation, obstructed or dysfunctional labour, red degeneration (pain, premature labour), primary PPH.

Haemophilia carriers – risk of bleeding problems (monitor factor VIII, usually rises in pregnancy), possibility of child being affected (male) or carrier (female), avoid traumatic procedures to mother and fetus.

Thrombophilia – can cause recurrent miscarriages and IUGR or IUFD. Clexane can be given safely as molecules are too big to cross the placenta.

ITP – risk of bleeding in mother (check platelet count) and thrombocytopenia in fetus (50%) as antibodies cross placenta.

SLE – increased rate of miscarriage, IUFD, pre-eclampsia esp if renal function affected an vascular problems (if anticardiolipin antibodies) e.g. thrombosis, placental infarction, IUGR. If anti-Ro antibodies, fetus is at risk of neonatal lupus syndrome – lupus dermatitis and haematological abnormalities, esp congenital heart block. Conceive when disease is quiescent, give low dose aspirin, stay on steroids, consider thromboprophylaxis.
Obesity – increased risk of DVT, gestational diabetes, bigger babies, don’t labour as well, anaesthetic complications.

Cardiac disease – most of the changes in CV function happen by 12 wks so if no problem by then good prognosis. Other problem is 3rd stage of labour – sudden preload increase as uterus contracts and empties blood into circulation. A soft ejection murmur is normal in pregnancy.

Obstetric cholestasis – itching (esp on palms and soles), generally in 3rd trimester. Only maternal problem is symptoms, ursodeoxycholic acid and sedative anti-histamines at night gives relief. Can cause unexpected IUFD (cardiac deaths), usually after 37wks, so monitor fetal condition 2/3 times per wk and induce at 37 wks. Get increased transaminases and bile acids (>12). About 50% recurrence in subsequent pregnancies.

Acute fatty liver of pregnancy – very rare, present in late pregnancy with abdo pain, vomiting and jaundice, then DIC and renal failure. 80% maternal mortality. Need immediate delivery, blood transfusion, FFP and blood sugar control.

Hyperemesis gravidarum – nausea is common up to 16wks, due to HCG and ketosis (vicious circle). Advise small meals and a night time snack to prevent ketosis. If serious can lead to hypovolaemia, electrolyte disturbance and thiamine deficiency. Need to admit, exclude cause (e.g. UTI, CNS lesion, multiple pregnancy, mole, acute fatty liver), give IV fluids, anti-emetics and thiamine.

UTI – more common due to stasis (dilated and partially obstructed ureters), increased vesico-ureteric reflux and glycosuria. Need to treat bacteriuria (even if asymptomatic) as 30% chance of developing acute pyelonephritis (complications are fetal demise, premature labour, permanent renal damage). Pyelonephritis – fever, loin tenderness, dysuria, pyuria, send MSU for culture and start IV augmentin, renal US after delivery to exclude structural anomaly.

Symphysis pubis dysfunction – pain due to softening of ligaments. May need wheelchair in bad cases.

Amniotic fluid
Produced from placental amnion and fetal urine.

Polyhydramnios – failure of fetal swallowing e.g. tracheooesophageal fistula, excess urine production e.g. diabetic mother, twin-twin transfusion. Have abdo discomfort and preterm uterine tightenings, uterus is large for dates with fetal parts difficult to feel. Problems are pressure symptoms, premature labour, malpresentation and cord prolapse. Need to exclude diabetes, twins and anencephaly, repeat amniocentesis is possible but risk of infection and preterm delivery so rarely done

Oligohydramnios – ruptured membranes, fetal renal abnormality, fetal illness (IUGR).

Antepartum haemorrhage
= bleeding from 28wks or when fetus is viable.
General management – admit and avoid VE until placental site established by USS. Serious bleeding is indicated by blood on feet and haemodynamic compromise.

Placenta praevia – placenta encroaching on lower segment, generally picked up on scan (but small ones can be missed), if in doubt examination in theatre. Major = crosses internal os (grades 3 and 4 – completely covers), minor = in lower segment but not across os (grades 1 and 2 – to edge of os). At 20 wks a lot of placentas are low lying but the majority move up by term as the uterus expands, those covering the os are likely to persist.
Presentation - typically painless vaginal bleeding, recurrent small bleeds or can get very serious bleeds, often around 32 wks as lower segment forms and creates shearing forces.
Management - usually admit and keep in hospital even if bleeding settles, need to check for IUGR. Often have to deliver early, by section if major (difficult as abnormal lie, bleeding on entry to the uterus; generally GA as with spinal get poor vascular reflexes in limbs to compensate for haemorrhage), if minor can induce with fetal monitoring. Main risk is now PPH, as lower segment contracts poorly after delivery.

Risk factors – multiple pregnancy, previous C section, uterine abnormality, multiparity. Associated with malpositions and malpresentations.

Abruption – bleeding from normally sited placenta as it separates before birth. As uterus is still distended, can't contract on uterine vessels so bleeds. Usually painful with hard tender uterus (if in labour doesn’t relax between contractions), Couvelaire uterus (purple as blood in myometrium), possibly absent fetal heart.

Management – resuscitate, opiate analgesia and deliver, usually by section. Blood passed PV may only represent small amount of bleed. Often CTG abnormalities and intra-uterine fetal death, if multiple small abruptions can get IUGR. Can’t see on scan unless very big, may have retroplacental clot and positive Kleihauer test (fetomaternal transfusion). Risk of PPH as DIC and uterus doesn’t contract well due to blood in myometrium. Associated with pre-eclampsia (need to exclude this).

Risk factors – grand multiparity, external trauma, overstretched uterus (e.g. polyhydramnios), past history of abruption, increased maternal age, smoking.

Blood products: blood (red cells), platelets, fresh frozen plasma (clotting factors), cryoprecipitate (fibrinogen).

Vasa praevia – bleeding from abnormally placed fetal vessel (velamentous insertion to placenta or succenturiate lobe) going through membranes at time of ROM. Blood lost from fetal circulation so high mortality rate. Get sinusoidal pattern on CTG.

APH can also be due to a show or cervical lesions (ectropion, cancer, cervicitis).

Prematurity and preterm labour
= delivery at less than 37 completed wks. 5% of deliveries.

Risk factors – multiple gestation, antepartum haemorrhage, fetal abnormality or death, uterine abnormality, incompetent cervix, infection, PROM, short cervix (<2.5cm) esp if funnelling, polyhydramnios, maternal pyrexia. May be iatrogenic because of pre-eclampsia, severe IUGR or deteriorating maternal or fetal health.

Consequences – death, RDS, intraventricular haemorrhage, pulmonary haemorrhage, necrotizing enterocolitis, sepsis, hypothermia, jaundice, hypoglycaemia, retinopathy, chronic lung disease, neurodevelopmental delay.

Prognosis - >95% of babies born at 32 weeks now survive, about 50% at 26 wks. Prognosis depends on availability of NICU, gestational age, birth weight, condition at birth, antenatal steroids. Tends to recur.

Management – admit, give steroids, tocolytics for 48hrs to allow steroids to work, check for causes e.g. UTI. C section esp if before 32 weeks has better outcome, ventouse delivery is contraindicated as increased risk of cephalohaematoma, forceps can be used. Preventing preterm labour - antibiotics, cervical suture if cervical incompetence, funnelling or shortening.

Post maturity
Post-term = after 42 weeks. Perinatal mortality rises due to placental insufficiency and as head becomes larger and moulds less easily. Most hospitals induce at term +12.
PROM  
= rupture before uterine contractions. If at less than 37wks, then preterm PROM. Diagnose by presence of liquor e.g. biochemical swab tests to differentiate from urine. Can lead to preterm labour, chorioamnionitis (3%), cord prolapse, rarely sepsis or endometritis and if prolonged pulmonary hypoplasia, umbilical cord compression and postural deformities.  
**Management** – admit, look for infection (amniotic fluid sample, pain, discharge, pyrexia, tachycardia), limit vaginal examinations, empirical treatment with erythromycin, steroids, monitor fetal wellbeing. Management is controversial – most now advocate watch and wait as more deaths due to prematurity than infection. 90% will establish in labour within 48hrs. If >34 wks – amnio to exclude infection, some advocate immediate delivery, others induction within 2 weeks. If <34 weeks – transfer to hospital with NICU, amnio, if no infection the conservative management. Only stop labour if occurs within 48hrs (before steroids can work). Deliver if infection, fetal compromise or reach 34-37 wks.

Chorioamnionitis  
Maternal pyrexia, tender uterus, foul smelling vaginal discharge, fetal tachycardia, increased WCC, organisms in amniotic fluid. Induce labour, generally avoid C section to reduce risk of maternal infection, but indicated if fetal distress, breech, failed induction. Broad spectrum IV antibiotics for mother and baby.

Prolonged progress in labour  
**Prolonged latent phase** – rare, almost always in primips. May be due to wrong diagnosis of labour, high pp, PROM, cervical dystocia. VE, short CTG, mobilise woman, pain relief, ARM, syntocinon. If after 8 hrs no progress then C section.

**Secondary arrest of cervical dilatation** - cervix reaches 5-7cm then stops. Uterine contractions become less frequent. More common in primips, usually due to inefficient uterine activity and fetal malposition (often OP), CPD. Rupture membranes if still intact and consider augmentation with oxytocin. Very rarely causes fetal distress. If after 8 hours of synto (4 for multips) no progress then C section for relative CPD, to avoid risk of uterine rupture and PPH.

**Primary dysfunctional labour** – slow progress after onset of established labour. Can lead to fetal distress, incoordinate uterine activity, maternal pain, anxiety and dehydration. Catecholamines stimulate uterine activity from lower segment so becomes incoordinate. Dehydration and acidosis cause H+ ions to compete with calcium so further dysfunctional uterine activity. May be due malpresentation, OP position, relative CPD, macrosomia. Treat with synto – 50% will need section for fetal distress.

Primary postpartum haemorrhage  
= loss of >500ml of blood within 24h of delivery. 1% of births.  
**Aetiology** – TTTT (trauma, tone, tissue, thrombin). Hypotonic uterus (reduced by active management of 3rd stage): uterus overdistension e.g. multiple pregnancy, large baby, polyhydramnios; uterine muscle fatigue e.g. high parity, prolonged labour; uterine infection; uterine abnormality; uterus relaxing drugs e.g. anaesthetics, nifedipine, MgS04; placenta praevia, abruption). Trauma – cervical, vaginal, perineal, uterine rupture. Retained tissues – e.g. incomplete placenta, succenturiate lobe. Clotting disorders – pre-existing, pre-eclampsia, DIC, anticoagulants.  
**Management** – oxygen, IV oxytocics, take blood for crossmatching, put up IV drip (blood given generally if loss >1l), determine cause. If uterine atony – massage uterus, syntocinon drip. If retained placenta – controlled cord traction, empty bladder, manual removal after 20mins. Manage uncontrollable bleeding with bimanual pressure (fist in post fornix and hand on ant abd wall), injection of PGE2alpha to uterus, pressure balloon in uterus, uterine sutures (need laparotomy), ligating int iliac or hysterectomy. Hemabate is intramyometrial PGF2alpha (carbaprost) to stop bleeding.  
**Placenta increta** – morbidly adherent placenta. Enbeds to myometrium so can’t separate.  
**Placenta percreata** – goes through myometrium and embeds to surrounding structures. High mortality as can’t control bleeding. Best treatment is hysterectomy – if want more children then removal is very dangerous, leave to atrophy with control of infection.
Secondary post partum haemorrhage – up to 6 weeks. Usually due to retained placenta and/or infection. Uterus fails to involute and cervix stays open. May need ERCP and antibiotics.

Perineal tears – 1st degree (skin only), 2nd degree (skin and perineal body), 3rd degree (anal sphincter), 4th degree (rectum). 3rd and 4th are repaired in theatre.

Multiple pregnancy
Rates – spontaneously occurring twins 1 in 80 pregnancies, higher order 1 in 80^n-1. Numbers increasing due to use of clomiphene and IVF. Twin conceptions > twin births as early spontaneous miscarriage of one fetus (vanishing twin).

Zygosity - 1/3 monozygotic (identical, one ovum which splits), 2/3 dizygotic (2 ova fertilised separately, familial and racial predisposition, increased with maternal age, parity, height and obesity).

Chorionicity and amniocity - most importantly thing to determine clinically. Dizygotic – always dichorionic, monozygotic - depends when splits (if early DCDA – 1/3, then MCDA – 2/3, if day 10-12 MCMA – 1%, if after 12 days conjoined). Dichorionic – lambda sign (=thicker insertion of membrane to placenta), always if different sex. Monochorionic – T sign on USS, higher morbidity, shared placenta so at risk of twin-twin transfusion syndrome, TRAP, can’t do selective termination, more problems if one twin dies (1 in 4 risk of serious harm, particularly neurological due to haemorrhage into dead twin causing hypotension). Monoamniotic twins are at risk of cord entanglement, high morbidity.

Presentation – usually present at USS, also as large for dates, hyperemesis, raised AFP, 3 poles felt.

Complications – every complication is increased (except postmaturity). PNM is 6x higher, mainly due to prematurity (10% before 32 wks, >40% preterm), esp if MC and for 2nd twin. Also IUGR, miscarriage, pre-eclampsia, anaemia, polyhyramnios, APH.

Management – supplement with iron and folate, monitor more closely antenatally, screen with NT not serum screening. Usually induce at 38-39 weeks. Generally can have vaginal delivery with vertex/vertex presentation, epidural advised to allow interventions for second twin (most at risk as harder to monitor and more likely to be malpresentation), must clamp cord in case fetal circulations connected, usually start syntocinon for 2nd twin as contractions tail off. If first twin is any other than cephalic need C section. Continuous monitoring of both fetuses in labour.

Twin-twin transfusion syndrome – only MC twins, affects 1/5 of MC twins. Due to unequal distribution of blood – have connection between circulations in placnta, if lots of these are AVA in one direction there is a net movement of blood between twins, AAA protect. Donor twin - anaemic, growth restricted, oligohydranmios (extreme is stuck twin, pushed right up to side with membrane tightly around), no bladder; recipient twin is plethoric, large for dates with polyhydranmios, big bladder. Treat by laser to placenta (or repeated amniodrainage), 90% mortality if not treated (due to hypoperfusion in donor and heart failure in recipient), survivors may have developmental conditions.

TRAP (twin reversed arterial perfusion) – only MC twins, when very close cord insertions. Acardiac acephalus twin receives arterial supply from pump twin (so retrograde flow – enters via umbilical a). Untreated 50% of pump twins die from heart failure (esp if acardiac twin is >50% of pump twins weight). High rate of chromosomal abnormalities.
Breech presentation

**Aetiology** – mostly idiopathic but also uterine abnormalities (esp bicornuate uterus), fetal abnormalities (e.g. hydrocephalus or anencephaly), multiple pregnancy, placenta praevia. 25% at 30 wks but only 3% at term.

**Types** – extended (most common, both legs extended), flexed, incomplete (one leg flexed, one extended), footling (both hips extended).

**Management** – if at 36wks clinically breech need scan to confirm position and investigate any reason. Explain it is a controversial area. Try external cephalic version – very safe and good chance of vaginal delivery if successful. Otherwise elective C section or vaginal delivery.

**C section** – least fetal risk, Canada term breech trial showed vaginal delivery was associated with increased PNM. However, implications of C section and controversy over whether there is a difference in outcome at 2 yrs, C section is no different to that for cephalic presentation.

**Vaginal delivery** – continuous monitoring, epidural, episiotomy. Risks are body being delivered then head getting stuck (head is biggest part and delivered last so doesn’t act as pelvimeter) and cord spasm before baby can oxygenate itself (cord is delivered early and exposed before head is out), as well as intracranial injury, damage to internal organs, umbilical cord prolapse. Deliver in lithotomy position, avoid touching baby and stimulating breathing, don’t handle abdomen as risk of damaging liver etc., body delivers self, may need to bring legs or arms down (Loveset’s manoeuvre - rotate one way to bring back to top then other way to bring arms across chest and bring down), when nape of neck exposed, head delivered by forceps (flex and protect against rapid decompression) or Mariceau-Smellie-Veit manoeuvre (head flexion and traction). Can’t use forceps of ventouse to expedite delivery.

**Other problems in labour**

- **Transverse lie** – risk of cord prolapse with ROM. If near term admit (common at earlier gestation) and section, try external version, or stabilising induction (start syntocinon, hold head in pelvis and rupture membranes). Can lead to shoulder presentation, obstructed labour and uterine rupture.

- **OP** – suspect with early urge to push, backache. Usually due to deflexed head (which may be due to inefficient early contractions. Encourage to change position to rotate head, most will rotate in labour so leave alone. Labour may be longer. If poor progress, rotate manually or with Kielland’s forceps. Increased risk of PPH.

- **Brow presentation** – mentovertical diameter is 13.5 cm. Needs section.

- **Face presentation** – due to lax uterus, polyhydramnios, deflexed head, abnormally shaped head. Mentoanterior will deliver, mentoposterior obstructs and needs section.

- **Absolute CPD** – no possibility of vaginal delivery. Rare, may be due to hydrocephalus, abnormal pelvis.

- **Relative CPD** – large baby but would pass if mechanisms of labour function correctly, arrest if head is deflexed or fails to rotate. CPD can really only be diagnosed by trial of labour. Suspect in short woman, from scan evidence, high head at term. If estimated weight >4.5kg usually offer C section.

- **Shoulder dystocia** – obstetric emergency. Shoulders do not spontaneously deliver as ant shoulder is trapped behind symphysis pubis, may get turtle necking (head delivers then retracts). HELPERS - call for Help, consider Episiotomy, raise Legs for McRoberts manoeuvre (to increase diameter of pelvic outlet – push legs up and back), suprapubic Pressure, Enter vagina for various internal manoeuvres e.g. screw manoeuvres (to decrease diameter of fetus by internal rotation), Roll woman onto all 4s or Replace head (Zafenelli manoeuvre), Symphysiotomy.

Risks to fetus – Erb’s palsy, cerebral palsy, fractured humerus or clavicle, neonatal death.
Risk factors – diabetics (as fat baby not necessarily bigger head), previous shoulder dystocia, long 2nd phase, instrumental delivery of head.

**Cord prolapse** – due to prematurity, malpresentations, polyhydramnios, multiple gestations, PROM or ARM with high head. Risk of loss of fetal blood supply due to mechanical compression of cord or spasm of arteries due to cooling, drying or handling. Immediate delivery needed – emergency C section or ventouse/forceps if cervix fully dilated. If not in hospital - hold cord in vagina with sterile gauze (minimal handling to avoid spasm), keep warm and moist and press on PP to relieve pressure on cord. Woman should adopt knees-to-chest position, fill bladder.

**Impacted uterus** – if retroversion persists to mid trimester uterus may become incarcerated in pelvis, mainly in multips. Causes pain, urinary retention, constipation, vaginal bleeding. Insert catheter, can usually resolve by patient positioning (knees to chest or sleeping prone) or manipulating. Risk of pregnancy loss.

**Uterine eversion** – usually due to cord traction with fundal placenta and inadequate uterine support. Causes acute vasovagal bradycardic shock. Replace uterus immediately, give atropine, fill with warm saline, give antibiotics and syntocinon.

**Uterine rupture** – due to obstructed labour (longitudinal lie, CPD, failure to progress – should prevent by C section), ruptured scar (pain continues). Risk factors are multips, syntocinon, prologned labour, previous C section. Can cause PPH.

**Amniotic fluid embolism** – very rare but can be fatal. Sudden shock and cyanosis at height of contraction. Raised CVP and opacities on CXR.

**IUFD** – confirm with US. If chorioamnionitis, deteriorating coagulopathy or placenta praevia then C section, If not then induction and vaginal delivery is safer and voids complications. Try to establish cause – infection (esp toxoplasmosis, rubella, CMV, parvovirus), abruption, diabetes, lubus, thrombophilia. It is useful to allow parents to hold baby and spend time together, take photographs, handprints and lock of hair. Parents should register baby. PM may give useful information for future pregnancies.

**Puerperal problems**

**Cracked nipples** – avoid prolonged suckling, ensure taking whole nipple, rest breast and express milk, usually heal rapidly.

**Breast engorgement** – common on days 3-4 (oestrogen falls and prolactin stimulates). Express milk, give analgesia, support breasts. If severe and don’t want breastfeeding can give bromocriptine.

**Mastitis** – usually staph or strep. Get pain and fever. Keep expressing milk, give antibiotics and analgesia.

**Puerperal fever** – usually genital tract, UTI, mastitis, superficial phlebitis, DVT, chest infection, wound infection. Can be endometritis – foul lochia, tender uterus.
The Fetus and Neonate

Chromosome abnormalities

Risk of chromosomal abnormalities – depends on age, gestation (higher if earlier as more likely to miscarry), previously affected pregnancies, known parental chromosomal abnormalities.

Downs = trisomy 21 (97% cases trisomy, rest translocations, 1/3 of these have recurrence risk if balanced translocation, or mosaics). 1 in 700 live births (1 in 2000 aged 25, 1 in 100 aged 40), no known environmental risk factors, vast majority not inherited and low recurrence risk.

Consequences – increased risk of fetal loss, growth restriction, hypotonic facies, characteristic facial appearance (but still family resemblance - epicantic folds, round face, small ears, Brushfield spots on iris), brachycephaly, single palmar creases, cardiac defects (40%, half need surgery), duodenal atresia (10%), LDs (most moderate but very variable, avg IQ about 50, usually learn walking language and self-care but rarely fully independent), Alzheimers, increased infections and risk of leukaemia, decreased life expectancy.

Antenatal screening – not diagnostic, only give indication of raised or lowered risk, be aware that some will be missed by screening and the majority of high risk by screening will not have Downs.

Double test (16 wks) – low AFP and high hCG is high risk, risk calculated based on this and maternal age. Add estriol for triple test and inhibit for quadruple test. NT scan – 12 wks, improves sensitivity, offered to multiple pregnancies and diabetics (double test less good), >35yr olds, IVF pregnancies. If risk greater than 1 in 250 then diagnostic test – karyotyping by amniocentesis or CVS. For 5% false negative, sensitivities of screening for high risk then diagnostic test - double = 60%, NT = 72%, combined test (NT and 1st trimester serum screening) = 85%. Soft markers – cardiac defects, duodenal atresia, echogenic bowel, short femur, choroid plexus cysts, renal pelvis or cerebral ventricular dilatation.

Edward syndrome = trisomy 18. 1 in 3000 live births, risk increased with maternal age. Miscarriage, growth restriction, severe LD, cardiac defects, hypoplastic lungs, flexion deformities, overlapping fingers, exomphalos. <10% survive a year.

Patau syndrome = trisomy 13. Rare, risk increases with maternal age. Miscarriage, midline defects, holoprosencephaly, severe LD, cleft lip and palate, polydactyly, cardiac defects. <20% survive a year.

Turner syndrome = 45X0, or deletion of short arm. 1 in 5000, usually sporadic. Miscarriage, cystic hygroma, hydrops, growth restriction, broad chest (widely spaced nipples, wide carrying angle), neck webbing, coarctation of the aorta (10%), streak ovaries, horseshoe kidneys, mild impairment of intellect. Short stature and failure of secondary sexual development, Need oestrogen replacement and growth hormone.

Congenital abnormalities

Neural tube defects – 1 in 1000, fallen due to screening. Due to genetic factors and folic acid deficiency, no increased risk with maternal age. Prevent by 400 micrograms folic acid preconceptually and 1st trimester, if previous or family history then 5 mg. Screening – raised AFP (other causes are abdo wall defects, multiple pregnancy, incorrect GA, bleeding), detailed scan. Risk of recurrence is about 5%.

Spina bifida – fluid filled sac with neural tissue and underlying defect of spinal arch, mostly lumbosacral, varying degree of handicap – paralysis, incontinence, deformities, hydrocephalus. Either myelomeningocele (cord and meninges, worse prognosis) or meningocoele (just dura and arachnoid mater, usually have normal lower limb neurology). Anencephaly – absence of forebrain and skull vault, incompatible with life.

Encephalocele – herniation of meninges and brain through skull, usually occipital. Recurrence risk is 2-3%, reduced to <1 by folic acid.
Ventriculomegaly = enlarged ventricles with excessive CSF.

Hydrocephalus = when this causes enlargement of skull. Isolation or due to aqueduct stenosis, intraventricular haemorrhage, viral infection, NTDs. Variable prognosis – worse if cortical thinning, may need ventricular shunts, mild cases can have normal outcome.

Microcephaly = head circumference >3SD below mean. Familial, congenital viral infections, genetic syndromes.

Abdo wall defects – failure of rotation and re-entry of gut during fetal development. Gastrochisis = separate from insertion of umbilical cord, peritoneal covering lost (so no membrane on USS), may be linked to environmental toxins.

Exomphalos = herniation of viscera through defect at umbilicus, peritoneal covering usually preserved. Associated with other congenital abnormalities and aneuploidy (esp 13, 18) so karyotyping advisable. Termination if other anomalies or surgical closure soon after birth (2 stage, risk of hypothermia and dehydration).

Prune belly syndrome – rare, probably in utero decompression of enlarged bladder due to urethral obstruction. Deficient musculature, undescended testes, renal anomalies (main morbidity).

Renal tract anomalies
Potter sequence: 1 in 3000, due to oligohydramnios (renal agenesis, dysplastic or polycystic kidneys, urinary obstruction, leakage of amniotic fluid). Fetal growth restriction and deformation, low set ears, limb flexion abnormalities, hypoplastic lungs (causing death soon after birth).

Urinary obstruction (usually posterior urethral valves) – cause hydronephrosis or renal dysplasia, can cause prune belly syndrome if bladder massively distended. Monitor by liquor volume.

Ectopia vesicae – ventral herniation of bladder between pubic symphysis, surgically correct but often incontinent.

Skeletal tract abnormalities
Osteogenesis imperfecta – type 1 collagen abnormalities. Excessive tendency to fractures, antenatally and postnatally. Deformed shortened poorly mineralised long bones.

Short-limbed dwarfism – mutation in fibroblast GF receptor. Achondroplasia most common, thanatophoric dwarfism more severe. Feature depend on type.

Limb reduction deformities – genetic syndromes, trauma e.g. CVS, toxins e.g. thalidomide, amniotic bands.

GI anomalies
Diaphragmatic hernia – see on USS, outcome depends on pulmonary hypoplasia and other associated abnormalities (if none 70%).

Tracheo-oesophageal fistula – blind ending oesophagus with varying communication with trachea, most associated with other abnormalities, polyhydramnios. Pass gastric tube before feeding.

Intestinal obstruction – double bubble with duodenal atresia (associated with Downs), often polyhydramnios.

CCAM (congenital cystoid adenomatous malformation) – affects lungs, type 1 = a few big cysts, better prognosis, type 2 = intermediate, type 3 = many small cysts, white appearance on USS, worse prognosis. 50% regress spontaneously in utero.

Beckwith-Wiedemann syndrome – macroglossia, macrosomia, visceromegaly, abdo wall defect (75% exomphacocele), neonatal hypoglycaemia, polyhydramnios, preterm delivery. Association with embryonal tumours esp Wilms tumour. Sporadic (most), C11 mutation, paternal uniparental disomy. 20% mortality due to complications.

VATER association – vertebral, anal, tracheal or oesophageal and renal lesions.
**Hydrops fetalis**

Fluid accumulation – oedema, ascites, pleural and pericardial effusions.

**Aetiology** – anaemia (haemolysis, haemorrhage e.g. twin-twin transfusion syndrome or fetomaternal, marrow infiltration, parvovirus disease), cardiac failure (arrhythmias, cardiac anomalies, AV shunts), hypoproteinaemia (nephritic syndrome, hepatic enzyme defects), obstructed venous return, congenital infection (esp parvovirus, CMV, syphilis), chromosomal anomalies.

**Management** – identify cause (success in 80%) by USS, maternal blood tests and fetal invasive test. Treatable causes – anaemia with intrauterine transfusion, arrhythmia with maternal digoxin, pleural effusions with insertion of shunts. Overall survival is 25%, better if corrected antenatally.

**Fetal growth restriction**

Failure of fetus to achieve genetic growth potential. Either symmetrical (intrinsic, early onset) or asymmetrical (head circ increases but not abdo ‘brain sparing’, extrinsic and late onset). 10% of all live births. Identify by USS (abdo or head circumference – progression along centile is more important than absolute value) or serial symphysiofundal height measurements. Detection is poor (up to 50% not recognised).

**Aetiology** – intrinsic cause e.g. chromosomal abnormalities, viral infections or extrinsic e.g. uteroplacental vascular insufficiency, smoking, maternal malnutrition. Associated with pre-eclampsia and abruption. Risk factors – previous history, smoking, APH, multiple pregnancy, maternal disease.

**Prognosis** – greater perinatal mortality and morbidity and developmental problems. Perinatal asphyxia, meconium aspiration, hypothermia, hypoglycaemia, polycythaemia. It is thought that this may cause hypertension and IHD in later life (Barker hypothesis).

**Traumatic delivery**

**Facial nerve palsy** – forceps delivery, general resolve spontaneously.

**Erb’s palsy** – stretching of C4,5,6,7. Often after shoulder dystocia. 15% have persisting deficit.

**Cephalohaematoma** – ventouse delivery, trapped RBCs can lead to jaundice.

**Intracranial haemorrhage** – tear falx if delivery to fast. May have fits.

**Intraventricular haemorrhage** – esp premature babies.

**Neonatal conditions**

**Hypoxic ischaemic encephalopathy** – get seizures, >50% risk of neurodevelopmental damage. Neonatal encephalopathy grading – 1: hyperalert, floppy, jittery, dilated pupils; 2: lethargic, fits, 3: flacid, no suck, no Moro reflex, prolonged fits. Prognosis – good if grade 1, or grade 2 for <5 days, poor if 3. Investigate further by EEG.

**Necrotising enterocolitis** – risk reduced by antenatal steroids. Give iv antibiotics and fluids.
Epidemiology

Birth = live births + still births.
Live births = complete expulsion from the birth canal of a fetus showing signs of life (e.g. HR, breathing, movement). In practice, this means that gets over 24 wks, as miscarried fetus may transiently have a HR e.g. at 12 wks.
Still births = any baby born after 24 wks that shows no sign of life (regardless of when baby died). 5/1000.

Low birth weight = <2500g.
Very low birth weight = <1500g.
Extremely low birth weight = <1000g.

Perinatal mortality – still births + deaths in 1st week. Mainly congenital anomalies (reduce by screening and prevention), prematurity (prevention e.g. cervical suture, steroids and better care), asphyxia. Risk factors – extremes of age, primips or grand multips, low socioeconomic class, extremes of maternal weights, short stature, smoking and drugs, maternal disease, complicated past obstetric history (CS, anomalies, prematurity, pregnancy failure), complications of this pregnancy. 8/1000 in UK, about 75/1000 in LEDCs.

Neonatal mortality – live births that die within 1st month.

Maternal mortality – death of a woman while pregnant or within 42 days of termination of pregnancy i.e. delivery, TOP, miscarriage (late includes up to 1 year). Either direct (obstetric complications), indirect (pre-existing disease) or fortuitous (unconnected). Use ratio of maternal deaths:births. UK – 10:100 000. Causes of direct deaths in decreasing order – (suicide and accident), thromboembolism, hypertensive disorders, amniotic fluid embolism, early pregnancy deaths (e.g. ectopic, miscarriage, TOP), sepsis, haemorrhage. Indirect are mainly cardiac and psychiatric. LEDCs – 1000:100 000, life time risk even higher as have more children.

Confidential enquiry into maternal deaths – 3 yearly report reviewing maternal deaths.

Levels of evidence: I = Strong evidence from at least one systematic review, II = Strong evidence from at least one RCT, III = Evidence from well designed other experimental studies, IV = Evidence from well-designed non-experimental studies, V = Expert opinion.

Important Note
These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources.
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