Normal Pregnancy

- **Conception:** 2 weeks after 1\(^{st}\) day of LMP with a regular 28 day cycle
- **Naegele’s rule:** EDD is calculated by adding 7 days + 9 months to 1\(^{st}\) day of LMP. Therefore, pregnancy lasts 9mths, or 38wks from conception: 40 weeks from 1\(^{st}\) day of LMP
- **Trimesters:** 1\(^{st}\) is weeks 1-13, 2\(^{nd}\) is weeks 14-27, 3\(^{rd}\) is weeks 28-40
- **Puerperium:** delivery – 6wks. This is reversal of the changes which happened in weeks 1-40

**Maternal physiology**

**Cardiovascular and respiratory changes:**
- Plasma volume rises by 40\% up to 32 weeks. RBC’s increase by 20\% (30\% with Fe supplements)
- Cardiac output rises by 40\%. CO+BP falls when mother is supine due to vena caval compression.
- BP falls in early pregnancy (reduced PVR) but rises to pre-pregnancy levels in latter stages.
- 40\% increase in tidal volume, no increase in RR, PO2 or pH. PCO2 falls a little.
- Haemodilution: even though Hb rises, the concentration falls + O2 demand increases by 15\%.
- Placenta causes rise in fibrinogen, factors 7,8,10 + fibrinolysis inhibited. Prevents blood loss at delivery
- Increased red cell mass: protects against the 0.5L blood loss at delivery (1L if twins or CS)

**Renal changes:**
- Dilatation of renal pelvis + ureters (from pressure + progesterone): predisposes to acute pyelonephritis
- GFR increases by 50\%: reduces plasma urea, creatinine and osmolality
- Increased urinary protein loss. Levels above 500mg in 24hrs is abnormal however.

**Endocrine changes:**
- Insulin secretion doubles, blood glucose stays same. Glycosuria may occur in normal pregnancy
- Thyroid binding globulin doubles. T3 + T4 fall slightly. Goitre more common in iodine deficient areas
- Anterior pituitary increases in size x2 - causes increased risk of ischaemia in PPH (Sheehans syndrome)
- Total + free serum cortisol rise, as does urinary free cortisol

**Muskuloskeletal + skin changes:**
- Mainly softening of the joints in the lower back and pelvis
- More rashes, nose bleeds, gum bleeds, hyperpigmentation, spider naevi + palmar erythema

**Calcium and phosphate changes:**
- Ca demand rises (especially in 3\(^{rd}\) Trimester and in puerperium due to lactation) with a corresponding increase in gut absorption. Ca is actively transported across placenta
- Serum Ca + P levels falls in parallel with protein (bound to albumin). Ionised Ca remains stable

**Liver changes:**
- Hepatic blood flow does not increase
- Alk. Phos. levels rise by 50\% and albumin falls by 10g/L (causes a fall in total protein)

**Uterine physiology**
- Morula becomes blastocyst at the 32 cell stage
- Implantation occurs 7-14 days post conception: blastocyst attaches to endometrium + trophoblast cells invade. Organogenesis: 2-8 wks post conception. Fetal heart beats from 3wks after conception
- Inner cell mass is the precursor to the embryo. Trophectoderm becomes the placental trophoblast
- Fetus develops in amniotic cavity, attached to placenta by UC. Amnion is membrane lining of cavity – expands as P progresses. 2\(^{nd}\) layer of membrane is the chorion which is in apposition to the amnion
- Placenta is anchored to maternal decidua + the intervillous space is supplied by maternal spiral arteries
- Chord normally has 2 arteries (deoxygenated blood from fetus to placenta) + 1 vein (oxygenated blood from placenta to fetus). Vessels are cushioned by Whartons jelly
- Uterus holds 5L at term (500x pre-pregnancy): due to muscle hypertrophy. Blood supply from uterine + ovarian arteries. Cervical mucous plug protects during pregnancy – dilation results in the ‘show’
Normal Labour

• Prior to parturition, cervical ripening occurs. Fetal head descends as cervix ripens. **Bishops score** reflects proximity of labour. High score: labour is more imminent and induction would be easier.

<table>
<thead>
<tr>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
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</thead>
<tbody>
<tr>
<td>Position of cervix</td>
<td>Posterior</td>
<td>Mid-cavity</td>
<td>Anterior</td>
<td>-</td>
</tr>
<tr>
<td>Dilatation (cm)</td>
<td>0</td>
<td>1-2</td>
<td>3-4</td>
<td>5+</td>
</tr>
<tr>
<td>Station</td>
<td>-3</td>
<td>-2</td>
<td>-1-0</td>
<td>1-2</td>
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<tr>
<td>Consistency</td>
<td>Firm</td>
<td>Medium</td>
<td>Soft</td>
<td>-</td>
</tr>
<tr>
<td>Length</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

- **First stage**: onset (regular painful contractions w/ increasing frequency + intensity) to full dilatation
  - *Latent phase*: 0-3cm dilatation (ripening may happen prior to onset of actual labour)
  - *Active phase*: 3cm – just before full dilatation: progress should be at least 1cm / hour
  - *NB*: effacement prior to dilatation: primiparous labour (simultaneous in multiparous labour)
  - Midwife: pulse + BP every 30mins, temp 2 hourly, fetal heart auscultation / CTG every 15mins, monitoring of urine output + urinalysis, regular vaginal examinations (2-4 hourly)
- **Second stage**: full dilatation to delivery of the fetus
  - ‘passive 2nd stage’ – absence of pushing to allow descent of head, ‘active 2nd stage’ – pushing
  - 80-90% of women will deliver within 12hrs of admission. Prolonged labour is >24hrs
  - Head is at level of ischial spines or lower at the onset of S2
- **Third stage**: delivery of the P + M’s: active m’ment: oxytocin, early cord clamping, controlled traction

**Progress in normal labour**

- Progressive dilatation of the cervix
- Progressive descent of the presenting part
  - Assessed by abdo + vaginal exam. Should be engaged (max diameter of head has passed through pelvic inlet/brim) during the course of the latter stages of labour
  - The ‘station’ refers to the level within the pelvis to which the leading fetal part has descended
  - Descent of the head is the sole determinant of progress in S2
- Satisfactory fetal well-being
- Satisfactory maternal analgesia

**Mechanism of labour**

- If normal, starts spontaneously with no intervention + presentation is cephalic
- **The passages** (measurements are average gynaecoid pelvis):
  - Pelvic inlet: transverse plane: 13.6cm, anteroposterior: 11.5cm
  - Mid-cavity: circular 12.5cm
  - Pelvic outlet: transverse plane: 10.5cm, anteroposterior: 12.5cm
  - Sacroiliac joints + symphysis pubis stretch prior to and during labour
  - Head enters pelvis transversely + descends in this position through the mid-cavity
  - Internal rotation of head to occipito-anterior position
- **The passenger**:
  - At onset of labour the head is flexed + at level of inlet: vertex presentation. 9.5cm av. diameter
  - Occiput transverse at engagement, then descent, flexion and internal rotation. Recession between contractions. Crowning: biparietal diameter has passed pelvic outlet + head is at introitus (recession. ceases), extension and finally delivery of anterior then posterior shoulder.
- **The powers**:
  - Uterine activity alone in S1 + passive part of S2, assisted by maternal pushing in active S2
  - Dilates cervix but reduces intervillous blood-flow to fetus
  - Assessed by palpation or monitor (good for frequency + duration, less good for intensity)
Labour continued

- **Perineal tears**: 1st degree – perineal / vaginal skin, 2nd – inc deep muscle layer, 3rd – inc external AS
- **Episiotomy**: mediolateral incision normally used to prevent extension to sphincter
- **Uterine inversion**: can occur with too much traction + can lead to haemorrhage + shock. Replace immediately by instilling warm saline into the vagina under anaesthetic
- **Premature rupture of membranes**: prior to onset of labour – increases risk of intra-uterine infection – consider induction of labour with oxytocin infusion or prostaglandins followed by oxytocin

Failure to progress

- **Causes**: problem with PPP’s: malpresentation, large baby / small pelvis: CPD, poor quality contractions, not enough relaxation of ligaments / moulding of fetal head
- **Risks to mother**: endometritis, dehydration, uterine rupture if severe
- **Risks to fetus**: congenital pneumonitis / infection, hypoxia + acidosis
- **Management options**:
  - Continue to observe
  - Rupture membranes: amniotomy – local release of Pg’s may hasten labour
  - Oxytocin: IV to augment contract’s – increases pain + speeds labour (more common in premips)
  - Caesarean section: with FTP over 4hrs and other indications e.g. breech etc

Induction of labour

- **Indications**: hypertension / pre-eclampsia, post-maturity, antepartum haemorrhage, IUGR, diabetes
- **Assess cervix + Bishop’s score before deciding to proceed**
- **Method**: membrane sweep, vaginal prostaglandins (usually PgE2), amniotomy (only after BS is favourable i.e. >5), oxytocin: all thought to mimic natural actions
- **Problems**: maternal distress + pain, discomfort from pessaries, hypertonic uterus, failure: Caesarean

Prolonged pregnancy

- Induction at 41+ wks reduces meconium liquor and reduces perinatal mortality
- If mother does not want induction, monitor for fetal distress regularly (liquor volume + CTG)

Fetal hypoxia and acidosis in labour

- **Causes**: progressive hypoxia+acidosis 2ndary to uterine uterine activity, intrauterine infection, acute events: cord prolapse, placental abruption, scar rupture
- **Doppler flow studies**: flow to cord may be reversed in maternal diastole – bad sign
- **Assessment of liquor**:
  - Reduced volume – renal dx, swallowing probs, mental prob, prolonged + unrecognised ROM’s. increased volume: diabetes
  - Meconium staining – Grade1: dilute staining, normal volume. Grade2: moderate staining.
    Grade3: thick pea soup, low volume – bad sign. NB. Meconium can be normal esp with 40+wks
- **Assessment of fetal heart rate (drops when post-term)**:
  - Auscultate at 15min intervals – 1min after a contraction
  - Continuous monitoring only in high risk pts (low risk pts leads to increased rate of interventions)
  - Increases with sympathetic activity + decreases when head squeezed – like continually putting head under water (base line variability)
  - Acute hypoxia: reduced FHR. Chronic hypoxia: increased FHR
- **Assessment of Cardiotocography (CTG – chart goes at 1cm / minute)**:
  - DR C BRAVADO: Define Risk (e.g. pimip, normal preg = low risk), Contractions, Base line fetal heart rate, Variability, Accelerations, Decelerations, Overall impression
  - FHR should be 110-160bpm, variability of > 5bpm (pencil-line trace is bad – may be due to pethidine: do blood gas), at least 1 acceleration every 15mins, decelerations – normal if with onset of contraction, non-reassuring if variable, bad if late – do blood gas. ok pH > 7.2 in labour
Antenatal Disorders

- **Hyperemesis gravidum:** persistent nausea and vomiting in 1st Trimester requiring hospital admission. Not assoc w/ bad outcome for pregnancy – reassure mother.
  - Predisposing factors: multi pregnancy, hydatidiform mole (exclude GI eg appendix, UTI)
  - Rx: U+E’s, FBC, USS, IV hydration, vitamins, small freq meals, pysch support, antiemetics
- In 2nd Tri, UTI is most common cause of vomiting. Exclude GI e.g. pancreatitis, cholecystitis, appendix
- In 3rd Tri we always consider pre-eclampsia

**Pre-eclampsia and hypertension** (BP 140/90 is considered high in pregnancy):
- Hypertension: 5-10% of preg’s, commoner in premips. Definition: 2 or more readings of over 90mmHg diastolic or one reading > 110mmHg diastolic
- PET: pre-eclamptic toxaeemia: hyperT w/ proteinuria / abnormal bloods. Shows other organs involved
- Eclampsia: onset of convulsions in the presence of hypertension or proteinuria

- **Pre-eclampsia:** multi system disorder (not only HT), thought to be from placental origin
  - Predisposition: premips 6x more common, past hx of pre-eclampsia, family hx, pre-existing essential hypertension, multi-pregnancy, renal dx, diabetes, other medical conditions
  - Clinical features: **Direct vascular damage** causes hypertension, oedema, platelet aggregation + DIC, cerebral haemorrhage, more DVT’s. **Secondary effects** kidney – proteinuria, brain – eclampsia, liver – dysfunction + haemorrhage. **Fetal: IUGR, oligohydranmios**
  - Presentation: routine screening (BP + urine), in 2nd half of pregnancy: headache, visual disturbances, abdo pain. Signs: HT, proteinuria, hyperreflexia / clonus, abdo tenderness, IUGR
  - Treatment: cured only by the delivery of the fetus and placenta. Delivery is indicated when HT accompanied by symptoms, significant proteinuria or lab testing indicates severe disease. Give steroids is < 32wks. Other tx: labetalol, methyldopa, nifedipine, IV hydralazine (all anti-hypertensive’s), magnesium sulphate (for fits)

**Analgesia in Labour**
- **Non-drug:** relaxation techniques, acupuncture, TENS (no evidence of efficacy of any of these)
- **Entanox:** 50-50 mixture of nitrous oxide + O2, better than pethidine: faster onset, shorter duration
- **Pethidine:** effective analgesia but assoc w/ reduced APGAR’s, maternal nausea, loss of control
- **Epidural:** most effective analgesia but may delay 2nd stage with more oxytocin + C.S’s

**Post-Partum Haemorrhage**
- Blood loss > 500mL in 24hrs of delivery. Incidence 1-2%, increased risk w/ forceps, ventouse, twins
- Causes: uterine atonia, perineal tears are by far the most common
- Risks: multiple pregnancy, operative delivery, APH, PP, large fibroids
- Management: ABC, IV lines, cross-match 2-6 units, oxytocin / ergometrin, rub-up contraction, (releases Pg’s), topical Pg’s, FBC, U+E, clotting, EUA if still bleeding, may need emergency hysterectomy

**Ultrasound:** Gestational age, multiple pregnancy + chorionicity, assoc w/ fewer inductions for post maturity, detection of fetal malformations, diagnosis of placenta praevia. Umbilical artery Doppler.

**Fundal height:** 12-14wks suprapubic, 20-22wks umbilicus, 28wks midway between umbilicus + xiphoid

**IUGR:** (is gestational age correct?), **Fetus:** constitutionally small, chromosomal abnormalities, congenital malformations, congenital infection. **Placental:** placental dysfunction. **Maternal:** vascular disease – e.g. SLE, hypertension, pre-eclampsia

**Polyhydramnios:** 60% idiopathic, **Fetal** poor swallowing, obstructed GI tract, increased urine, CNS problem. **Maternal** diabetes, rhesus disease. **Oligohydranmios:** ruptured membranes, IUGR, fetal renal problem, maternal NSAID’s, ACEi’s, twin pregnancy
Antenatal Care

- First encounter is usually with the GP who confirms the pregnancy + writes to the hospital to request ‘booking.’ Also advice on folic acid etc.

- **Booking**: in hospital or at home with midwife – risk assessment, plan of care, start of screening, referral for medical / social needs. Includes addressing EDD, MH, any concerns
  - Examination: BP, heart, fundal height, consider speculum for smear + PV if indicated
  - Bloods: FBC (anaemia), Hb electrophoresis (haemoglobinopathies), VDRL/TPHA, blood group, antibody screen (haemolytic disease), Hep B screen, rubella screen, AFP for Downs, NTD’s, consider HIV and toxoplasma

- **General schedule**: booking visit at 12wks (hospital), GP every 4 wks to 28 wks, every 2wks from 28-36wks, every week from 36wks to delivery. High risk pts will need hospital, not GP-based care.

- **USS services**: usually 12wks for gestational age, multiple pregnancy, some fetal abnormalities (nuchal fold thickness, viability), 18-20wks anomaly scan, placental site and exclude IUGR. In 3rd trimester consider doing USS for fetal growth, liquor volume, presentation, Doppler if IUGR suspected

- **Maternal bloods**: triple test at 16 wks is serum AFP (low in Downs), hCG (high in Downs), unconjugated oestriol (low in Downs). USS at 12 (nuchal translucency) and 20 weeks.

- **Amniocentesis** can be performed at 10-14wks and **chorionic villus sampling** at 8-12wks

- Remember advice on stopping smoking and diet is given throughout

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Note
The original notes on these topics were written by S Forrington when a final 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. Disclaimer: no responsibility can be taken by either the author or publisher for any loss, damage or injury occasioned to any person acting or refraining from action as a result of this information