

# Obstetrics & Gynaecology Teaching

## Terminology

LMP – 1<sup>st</sup> day of bleeding of last period

1<sup>st</sup> trimester – LMP to 13/40

2<sup>nd</sup> trimester – 13/40 to 28/40 (includes viability – legally 24/40)

3<sup>rd</sup> trimester – 28/40 to delivery

Term – 37/40 to 42/40 considered normal term

Gravida – number of pregnancies, including the current one

Parity – X + Y

X: Number of pregnancies leading to delivery after 24 weeks

Y: Number of pregnancies which end before 24 weeks (TOP/miscarriage)

Naegle's Rule – EDD is LMP plus one year, minus three months, plus seven days.

## Menstrual Disorders

Notation: K=x/y where x is number of days of bleeding, y is total cycle length

Menorrhagia: Menstrual blood loss of 80ml/month – level likely to cause iron deficiency, against mean ~40ml, median ~30ml. Affects ~10% of population.

Subjective assessment by frequency of changing pads, need to change overnight, flooding, passage of clots, interference with life (take seriously, but may not correlate with volume lost). Problems are mainly social, unless iron deficiency occurs.

Causes: dysfunctional uterine bleeding (idiopathic – increased fibrinolysis and increased/disturbed prostaglandins), fibroids, polyps, endocrine (anovulation, obesity, thyroid), haemostatic disorders.

History, PBAC (Pictorial Blood loss Assessment Chart), objective measurement, menstrual calendar, FBC, US, endometrial assessment (sample if >40, persistent intermenstrual bleed, failed medical therapy, surgery considered) – exclude malignancy.

Treatment:

### Medical

- Non-hormonal
  - Tranexamic acid
  - NSAIDs
- Hormonal
  - cOCP
  - Cyclical gestagens
  - Mirena
  - Danazol
  - GnRHa

### Surgical

- Ablation/resection
  - Laser
  - Loop
  - Microwave
  - Hot water
- Hysterectomy (±BSO)
  - Abdominal
  - Vaginal
  - Laparoscopic
- Myomectomy

Significant surgical workload – menstrual problems commonest indication for hysterectomy in UK. 1:5 women at 55 have had hysterectomy.

Dysmenorrhoea – pain associated with menstruation, may precede onset.

*Primary*

No organic cause, presents from onset of ovulatory cycles  
50% women affected, 10% severely  
NSAIDs/cOCP

*Secondary*

Often endometriosis, adenomyosis, polyps  
Pain may precede menstruation  
Treatment depends on cause

Endometriosis – tissue histologically similar to endometrium at ectopic sites, usually pelvic (pouch of Douglas, ovary) but may be distant.

Symptoms – Dysmenorrhoea, deep dyspareunia, pelvic pain, rectal bleeding – always suspect, never dismiss.

May present with pain or infertility – treatment tailored to patients complaint.

Treatment – Medical (GnRHAs, Danazol, Progestagens, cOCP), Surgical (excision/ablation, radical)

Premenstrual Syndrome (PMS) – cyclical, commencing in luteal phase, relieved by menstruation. Combination of physical, psychological, behavioural symptoms.

Symptoms – Breast tenderness, bloating, GI upset, irritability/mood swings, aggression, depression.

May be no physical signs. Need to exclude other organic disease – psychiatric disease may present to gynaecologist!

Management – explanation/understanding. cOCP, Danazol, Oil of Evening primrose, high dose oestrogens, GnRHAs, SSRIs. Some fairly good, but none widely successful.

## **Gynaecological Endocrinology**

GnRH – produced in arcuate nucleus of hypothalamus, released in pulsatile fashion into pituitary portal system. Slow pulses cause anovulation, frequent/constant release lead to downregulation.

Gonadotrophins – produced in gonadotropes of anterior pituitary. Some cells produce both FSH and LH in response to GnRH. Common  $\alpha$  subunit,  $\beta$  varies. Only  $\alpha\beta$  heterodimer is biologically active. Surge in LH causes ovulation (FSH level usually higher than LH). Chronic anovulation leads to tonic LH elevation due to inappropriate feedback by oestrogen from peripherally converted androgens. LH elevation encourages androgen production.

LH causes cholesterol  $\rightarrow$  androstenedione in theca cells, FSH activates aromatase in granulosa cells to convert this to oestradiol. Regular cycle (21-35 days, less than 7 day variability) implies ovulation. Oligo- or amenorrhoea implies anovulation. Mid-luteal progesterone – measure 7 days before next period due.

Anovulation – history of oligo- or amenorrhoea (any bleeding suggests adequate oestrogen, galactorrhoea suggests pituitary problem, hirsutism elevated androgens). Take history, measure FSH, LH, prolactin, TSH, (testosterone if hirsute or virilised). US pelvis, pituitary imaging, assess adrenal androgens, karyotype (if primary or early onset), GTT/fasting lipids.

Causes:

- PCO – inappropriate feedback – tonic elevation androgens, follicular phase levels oestradiol. Associates with insulin resistance. May lead to NIDDM, endometrial carcinoma, iatrogenic complications. Presents with oligo- or amenorrhoea, infertility, hirsutism, menstrual problems. Tx depends on presentations. Diagnosis – US (enlarged ovaries, dense stroma, peripheral small follicles), normal FSH/LH, high normal/slightly raised testosterone, mild elevation prolactin possible).
- Kallman's syndrome – GnRH neurones fail to migrate to arcuate nucleus. Associated with anosmia.
- Pituitary adenoma – presents with amenorrhoea, galactorrhoea, symptoms of low oestrogen, visual disturbance, headache
- Premature Ovarian failure

Hyperprolactinaemia – confirm elevated level, CT/MRI imaging pituitary, TFT, visual field assessment. Tx with dopamine agonists (cabergoline, bromocriptine), rarely surgery, almost never radiotherapy.

Hirsutism – idiopathic, PCOS, adrenal pathology (Congenital Adrenal Hyperplasia, tumour), ovarian tumours. Tx – anti androgens (cyproterone acetate, spironolactone, Dianette), synthetic oestrogens (Dianette), ovarian suppression (GnRH), adrenal suppression (dexamethasone).

Menopause defined as the last period. Decline in oocyte numbers, increasing ovarian resistance to stimulation, no ovarian hormone production despite gonadotrophins, low oestrogen causing failure of endometrial proliferation. Vasomotor symptoms (hot flushes, night sweats), psychological (insomnia, poor concentration/memory, lethargy, reduced libido), loss of trophic effects (vaginal atrophy & dryness, prolapse, reduced breast size, urinary symptoms), osteoporosis, increased cardiovascular risk.

HRT types – unopposed (if no uterus), sequential (progesterone pulses), long cycle, continuous combined, gonadomimetic (latter two – no cycle). Others have withdrawal bleed/endometrial shedding – not a period. Administration – oral, transdermal, vaginal, implants. Relieves symptoms, protection against osteoporosis, reduced CV risk, reduced Alzheimer's risk, reduced colonic cancer risk. May cause resumption of menses, side effects, increased thromboembolic risk, increased risk of breast cancer (RR~4%, but low absolute risk still).

## **Multiple Pregnancy**

Incidence of twins 7/1000 (Japan) to 40/1000 (Nigeria). Europe ~1/80, triplets 1/80<sup>2</sup>.

Increased risks – N+V, miscarriage, PV bleed, anaemia, HT, backache, dyspnoea, antepartum haemorrhage, polyhydramnios, pre-term delivery (35/40 twins, 33/40 triplets), complicated delivery (55% UK twins LSCS), PPH, postnatal depression, financial burden, sleep disturbance

Foetal risks – stillbirth (still 3x higher at term), pre-term delivery, IUGR, congenital abnormalities, monochorionicity (twin-twin transfusion syndrome, conjoined twins, cord entanglement if monoamniotic)

Embryo cleavage:

4 days – dichorionic, diamniotic (can also result from dizygotic pregnancy)

4-8 days – monochorionic, diamniotic

8-13 days – monochorionic, monoamniotic

13+ days – conjoined

(DC – ‘lambda sign’ of placental tissue into intertwin membrane)

Prevention – caution with assisted fertility tx.

Monitor for maternal and foetal complications.

Deliver MCMA twins at 32-34/40 by CS, MCDA by 36-37/40 (usually CS), DCDA usually by 38/40, DCDA (CS if 1<sup>st</sup> twin breach, vaginal delivery if 1<sup>st</sup> cephalic, but with caution). Triplets CS by 33/40 or before.

### **Medical Disorders in Pregnancy**

Two patients to consider, but priority is usually to care for mother (e.g. if indicated, perform CXR). Better condition before pregnancy tends to lead to better condition during pregnancy.

Asthma is not directly affected by pregnancy (although breathing itself is).

Diabetes: 1-2% of women develop gestational diabetes as pregnancy ‘tests’ insulin system. Pre-existing diabetics get worsening glycaemic control – effect of hPL, emesis, increasing insulin requirements, strict control increases number of hypos. Proliferative retinopathy is accelerated, but no evidence of permanent effect on nephropathy.

Increased rate of miscarriage, infection (UTI, respiratory, vulvo-vaginal candida).

Congenital anomalies (3x background incidence – neural tube defects, cardiovascular). Macrosomia (polyhydramnios, increased risk of shoulder distocia) reduced with good control. Increased peri-natal mortality – metabolic anomaly, increased RDS risk.

Management of pregnant diabetic:

1. Management in combined clinic, visits two weekly as minimum
2. HbA1c monitored
3. All on insulin or diet controlled (no oral hypoglycaemics), regime altered to give good control – usually 3x short acting with meals, 1x long acting, but some have subcutaneous pumps
4. Assessment and monitoring of diabetic complications
5. Prompt treatment of infections

Management of pregnancy in diabetic:

1. Scan for viability and anomaly, mid-trimester for growth
2. Plan delivery ~38 weeks
3. Assess risks and mode of delivery
4. Carefully monitored labour (often caesarean), sliding scale
5. Neonatal input if required

Heart disease – aim to assess severity, avoid endocarditis, precipitants of failure, and foetal surveillance for IUGR.

Antenatal care involves patient education, prevention of anaemia, prompt and aggressive in-patient treatment of infection, consideration of altering anticoagulants (balance of risk), and foetal scanning for anomaly and growth. Intra-partum care involves good analgesia (epidural), careful fluid balance, continuous foetal monitoring, avoidance of prolonged 2<sup>nd</sup> stage, avoidance of ergometrine (↑BP).

Epilepsy affects ~1 in 350 pregnancies. Effects include neural tube defects, teratogenicity of anticonvulsants, depressed levels of clotting factors, physical injury, change in seizure frequency, altered drug metabolism.

Pre-pregnancy medication review – aim for low teratogenicity therapy. High dose folic acid, vitamin K in 3<sup>rd</sup> trimester, neonatal vitamin K, but no special requirements for delivery.

### **Hypertension in Pregnancy**

Measure BP with woman reclining, 30° lateral tilt, use Korotkoff I and V, unless V tends to 0, when IV is used (and recorded as such). Expect fall in BP, to 30/15mmHg below normal in second trimester, reverting in third. If BP >140/90, then significant increase in perinatal mortality. Chronic hypertension is recognised if it precedes pregnancy or is diagnosed before 12/40. However, pre-eclampsia can still occur and has a poorer prognosis in these patients.

Proteinuria up to 0.3g/l (0.5g over 24hr) is considered normal.

Usual definition of pre-eclampsia is two of:

- Hypertension >140/90 on two occasions over 4 hours apart
- Proteinuria
- Symptoms inc. headache, photophobia, visual disturbance, epigastric pain, altered LoC (this point is not in the classical definition)

Pregnancy induced hypertension – rise in BP without proteinuria, detected after mid-pregnancy. May be transient, first presentation of chronic HT, or go on to PET.

PET believed related to a defect in placentation. Genetic link, details not yet clear. Increased risk in primigravidae. Placental and circulating factors involved, lead to maternal HT, foetal hypoperfusion, reduced circulating platelets, DIC risk, hepatic dysfunction, renal dysfunction (ARF rare).

PET presentation – often frontal headache, visual disturbance, epigastric pain.

HELLP (Haemolysis, Elevated Liver enzymes, Lowered Platelets) syndrome – variant of PET. HT may not be present. DIC, placental abruption, foetal death common complications.

Treatment of PET – antihypertensives (hydralazine, labetalol, methyldopa, nifedipine) to keep MAP <125mmHg. Anticonvulsants (MgSO<sub>4</sub>) in severe PET/eclampsia. Corticosteroids prior to delivery.

## Problems of Early Pregnancy

Recognition of pregnancy:

- History
  - Amenorrhoea (defn: no period in 3 cycle lengths)/abnormally light periods
  - Breast tenderness
  - Nausea, may be any time of day
  - Urinary frequency
  - Tiredness
- Signs
  - Breasts enlarge, areolae darken
  - ↑vascularity of vagina, softening of cervix (Hegar's), uterine enlargement
  - Foetal heart sounds
  - Striae gravidarum, linea nigra
- Investigations
  - Urinary or serum  $\beta$ hCG
  - Ultrasound

Miscarriage (spontaneous), termination (intentional). Types:

- Threatened – PV bleed with closed cervix, often painless
- Inevitable – Open cervix, pain
- Incomplete – some products of conception lost
- Complete – all products of conception lost, pain and bleeding resolve
- Missed – Foetus dies with no bleeding, contents not expelled

Management – confirm pregnancy, scan for viability if cervix closed. Check blood group for need for anti-RhD. Monitor for shock. Conservative (await expulsion of contents), medical (Oxytocin and Misoprostil), or surgical (ERPoC).

Recurrent miscarriage – three or more consecutive miscarriages – distinct from sporadic miscarriage. Previous successful pregnancy best prognostic feature. Causes: Anti-phospholipid syndrome/other thrombophilic conditions, PCO syndrome, endocrine (diabetes, thyroid disease), uterine or cervical anomaly, genetic. Investigation: Thrombophilia screen, anti-cardiolipin antibodies, maternal/paternal karyotypes, hysteroscopy/HSG, US, LH, (GTT, T4, TSH as indicated) Rare to find a cause. If found, aim for aspirin+heparin in anti-phospholipid, surgery for uterine anomaly, weight loss for PCOS, good control of diabetes/thyroid.

## Ectopic Pregnancy

Must consider ectopic in a woman of child bearing age with lower abdominal pain.

Aetiology – PID, post-abortal, post-partum, tubal surgery, progesterone only contraception, (IUCD – increases risk of PID)

Presentation – Amenorrhoea, abnormal bleeding, pain (inc. shoulder tip), collapse/faint, desire to defecate.

Diagnosis by serial  $\beta$ hCG, US (empty uterus unless heterotopic pregnancy, esp. IFV, adnexal mass, free fluid)

Treat – resuscitation as required. Laparoscopy may be therapeutic (salpingectomy/salpingotomy), laparotomy in emergency. Single dose methotrexate is alternative treatment.

## Hyperemesis Gravidarum

Nausea and vomiting common any time of day in 1<sup>st</sup> trimester. Aim for hydration and nutrition by dietary adjustment (small meals, avoid greasy food). Minimal risk of miscarriage even in most severe cases. Assess level of hydration, HR, BP, uterine size (more common in multiple and molar pregnancies), consider non-obstetric causes (e.g. UTI).

If severe, admit. Assess hydration (ketones in urine, U&Es), fluids as indicated, US, LFTs and TFTs, anti-emetics.

## Obstetric Haemorrhage

### *Antepartum haemorrhage (APH)*

Bleeding from genital tract after 24/40. Similar causes earlier, but classed as threatened abortion.

Consider vaginal/cervical origin – may be unrelated to pregnancy

- Placenta Praevia
  - Placenta implants on lower uterine segment – distinct from low-lying placenta
  - Vasa praevia – isolated blood vessels on lower uterine segment
  - 0.5-1% of pregnancies
  - Risk factors: age, multiple pregnancy, high parity, uterine scarring, assisted contraception
    - I – just on LS
    - II – reaches os
    - III – covers os
    - IV – covers os, placenta central
  - Complications – haemorrhage, obstructs SVD, malpresentation, PPH
  - Management – Resuscitation, admit, delivery by LSCS, avoid vaginal examination until praevia excluded
  - Diagnosis – painless PV bleed, non-tender uterus, US, malpresentation/high head
- Abruptio placentae
  - Separation of all or part of placenta prior to delivery
  - 1% of pregnancies
  - Risk factors: social deprivation, smoking, IUGR, pre-eclampsia/hypertension, previous abruption, trauma
  - Types – concealed, revealed, mixed
  - Complications – Foetal death, DIC, renal cortical necrosis, hypertension/proteinuria
  - Recognition – hard, woody uterus, acute abdominal pain, shock
  - Management – Resuscitation, consider delivering, careful fluid management
- Bleeding of indeterminate origin
  - Many may be small abruptions
- Uterine trauma and rupture
- Cervical ectopy, polyps, carcinoma; vaginal trauma, varicosities; vaginitis

### *Postpartum haemorrhage (PPH)*

Primary – bleeding (>500ml) from completion of 2<sup>nd</sup> stage until 24hrs

Secondary – bleeding (>500ml) from 24hrs to end of puerperium (pregnancy hormones return to normal, ~6-8/52)

#### Primary

- Uterine atony – most common (80-90% of cases)
  - Prolonged labour, multiple pregnancy, augmented labour
  - Resuscitation, oxytocics, prostaglandin analogues, empty bladder
- Retained placental tissue
- Genital tract trauma (may be iatrogenic)

#### Secondary

- Retained products (os will be open), infection, infected retained products
- Treatment – ERPoC, antibiotics – colonisation from GI tract, little place for US, remember return of menses

#### RBC Isoimmunisation

- Foetal cells recognised by mother as non-self – risk of immunisation when foetal cells enter maternal circulation. Can result in foetal/neonatal haemolysis
- Major antibodies – anti D, anti c, anti-kell
- Sensitisation – APH, external cephalic version, amniocentesis/CVS, miscarriage/TOP, delivery
- Prevention
  - Screen for red cell antibodies – booking and 28/40
  - Give anti-D to cover sensitising events, consider in all Rh –ve women
- Kleihauer test – foetal Hb resists alkali denaturation and acid elution – appear against a background of ghost adult cells. Dose of anti-D can be tailored to size of foeto-maternal transfusion

### **CTG Monitoring**

Cardiotocograph – one measure of foetal wellbeing.

#### *DR C BRAVADO*

<i>Determine Risk</i>	(from obstetric history)
<i>Contractions</i>	(Clear trace, regular, no more than 5:10)
<i>Baseline RAte</i>	(110-160)
<i>Variability</i>	(>10)
<i>Accelerations</i>	(Present; up to 40 minutes at a time without ok)
<i>Decelerations</i>	(Early are fine, late more concerning)
<i>Overall impression</i>	(Decide if sinister/borderline/ok)

### **Delivery**

1-2% of births nationally are at home – safe for low-risk pregnancies.

Ventouse/forceps used in 7-10%. Caesarean rate 25% at Rosie, aim is <15%.

Start of labour can be indicated by waters breaking (but can be pre-labour rupture of membranes), loss of mucous plug, or contractions (but can be Braxton-Hicks – longer, irregular, painless).

Stage I – Labour begins, cervix alters tone/consistency

Stage II – Baby delivered

Stage III – Placenta delivered

(Stage IV – baby feeds)

Factors:

- Power
- Passageway
- Passenger

### *Stage I*

Cervix effaces (softer, thinner, flattens, but still closed) and dilates (10cm is fully dilated in typical woman). 0→3/4cm is often slow (can take days) – latent phase labour. Remainder is active phase labour, expect 1cm/2hrs. Expect 3-4 contractions in 10 minutes, each lasting ~45s. Foetal heart rate should be checked every 15 minutes for a routine birth. Abdo and vaginal exam. Stage I ends when cervix fully dilated.

### *Stage II*

Contractions change from expansive to expulsive.

Pelvis is widest laterally at inlet and widest AP at outlet, skull is longest AP – head rotates during delivery. Head should engage with occiput facing left or right, lateral or anterior (LOL, ROL, LOA, ROA). Occiput posterior (LOP/ROP) is more difficult. First structure to contact pelvic floor rotates anteriorly – normally head flexed, so this is occiput. Head delivered by extension. Shoulders contact pelvic floor and rotate, restitution of head, anterior then posterior shoulder delivers, remainder follows.

### *Stage III*

Physiological 3<sup>rd</sup> stage – cord intact, expelled (10-20mins, can be up to 1hr), then cut. Active 3<sup>rd</sup> stage – cord clamped and cut, oxytocic drugs (syntometrine) give, placenta pulled out using cord (usually <7minutes). Less bleeding.

LSCS – via Pfannenstiel incision (low bikini line). 99% of caesareans.

## **Preterm labour**

Preterm – 24-37/40, term – 37-42/40, post-term – >42/40.

Preterm labour may be spontaneous (~7% pregnancies, may be preceded by Preterm Premature Rupture Of Membranes – PPRM) or iatrogenic (induction/CS – 1/3 of premature deliveries).

Risks – low socioeconomic status, smoking, ?high parity, low maternal age/weight, previous preterm labour, present obstetric history (e.g. recurrent bleeding)

Aetiology – infection thought to have a role in many cases, multiple pregnancy (uterine stretch hypothesis), cervical weakness/'incompetence', Mullerian duct abnormalities, placental abruption/praevia, polyhydramnios, trauma, foetal abnormality, maternal illness, most are idiopathic.

Prediction – shorter cervical length on transvaginal US implies increased risk. Foetal fibronectin, bacteriuria, UTI also markers.

Management – history and examination. Investigations for cause. Decide if preterm

labour or not. Treat with tocolytics (adverse effects.  $\beta_2$  agonists, indomethacin,  $Ca^{2+}$  blockers, oxytocin antagonists, GTN), steroids (only drug with proven benefit – matures type II cells in lungs), antibiotics (used if PPRM >7 days, erythromycin), fluids as indicated, prepare for preterm baby. Cervical cerclage may be prophylactic or ‘rescue’.

### **Perinatal mortality**

Perinatal mortality rate – still births and deaths to age 1/52. 7.9 per 1000 births (UK)

Neonatal death rate – deaths age 7 to 28 days. 3.8 per 1000 total births (UK)

Stillbirth rate – 5 per 1000

Prenatal deaths – in utero, weight >500g

Causes of perinatal mortality – low birth weight/immaturity (48.5% neonatal deaths), infection, congenital abnormalities, APH, maternal disorders, unexplained (69.4% of stillbirths). Risk associated with maternal age and parity, social class, race, multiple births.

CESDI – Confidential Enquiry into Stillbirths & Deaths in Infancy (1992-). Various reports produced each year based on case reports.

CEMD – Confidential Enquiry into Maternal Death (1952-).

CEMACH – Confidential Enquiry into Maternal & Child Health (CESDI+CEMD) (2003-)

### **Contraception**

- Abstinence/non-penetrative sex
- Rhythm ( $\pm$ hormone levels (e.g. Persona)  $\pm$ temperature  $\pm$ cervical mucous)
- Barrier
  - Condom, femidom, diaphragm, cap
  - Limited effectiveness, but does reduce risk of STD
- OCP/implant/injection/patch
  - COC – prevents ovulation. POP – thick mucous.
  - Implant – 3 year duration, but removable. Best method available
- Sterilisation
  - Vasectomy/tubal ligation, vasectomy  $\sim$ 10x more successful
- IUD
  - Prevent implantation
  - Copper is spermicidal
  - Intrauterine System (IUS) – IUD + hormone, e.g. Marina
- Spermicides
- Coitus interruptus
  - Fairly effective if done properly

Post-coital contraception – pill or IUD. Levonelle prevents 95% of expected pregnancies if taken <24hrs, 85% 24-48hrs, 58% 48-72hrs.

'Ideal' contraceptive:

- 100% effective
- Not intercourse related
- Reversible
- No bad side effects
- Safe
- STI protection
- Acceptable
- Cheap
- Discreet
- Easy to use

## Infertility

Clomifene - induces ovulation, in particular useful in PCOS

Intra-uterine insemination (IUI) +/- clomifene – good for mild male factor

In-vitro fertilisation (IVF) – good for tubal obstruction

Intracytoplasmic sperm injection (ICSI) – important if significant male factor

## Gynaecological cancers

### Cervical

- 3400 cases pa, incidence similar in all age groups >30. Up to 40% detected by screening, 80% diagnosed on speculum exam
- Presents – post coital/intermenstrual bleeding, postmenopausal bleeding. Mass screening, ~84% of female population in UK screened
- Associated with HPV – RR 20-100. Present in >95% of CIN3 and cervix cancers
- Clinically staged. Surgery for early stages, RT for later.
- Fertility issues

### Ovarian

- 5400 cases pa, 95% age >40. Usually diagnosed late, CA125 and US. Epithelial tumours most common. High mortality in stage III/IV.
- Presents – non-specific abdo symptoms, anorexia, abdo distension, pelvic mass. Targeted screening for high-risk women.
- Surgically staged, treatment with surgery (stage and debulk) and chemo.
- BRCA H2 is risk factor
- CA125 follow up

### Endometrial

- 3900 cases pa, 95% after menopause. Presents early, most adenocarcinomas – histology affects prognosis
- Risk factors – post-menopause, unopposed oestrogens, Tamoxifen, obesity, diabetes, low parity/prolonged anovulation, pelvic irradiation, familial predisposition ('Lynch Syndrome')
- Presents – postmenopausal bleeding (10% risk of malignancy). Targeted screening
- Diagnosis with US, endometrial biopsy, hysteroscopy
- Surgical staging. Adjuvant RT treatment.

### Vulval

- 800 cases pa, usually age >60. Can be associated with VIN3. Local spread
- Presents – pruritus, lump, pain, 'burning'
- Surgery ± RT, may need plastic reconstruction