Anatomy

Embryology - Fallopian tubes, uterus and upper 2/3 of vaginal develop from Mullerian ducts. Lower 1/3 develops from urogenital sinus.

Upper genital tract – uterus, fallopian tubes and ovary.
Lower genital tract – vulva, vagina and ectocervix.

These are subject to different diseases as have different epithelium (squamous in lower and columnar in upper) so LGT commensal bacteria can penetrate UGT and become pathogens.

Cervix: Ectocervix – stratified squamous epithelium. Endocervix – columnar epithelium. Squamo-columnar junction is far out before puberty so mostly see columnar, but moves up during puberty as hormonal changes cause pH changes so columnar cells metaplace to squamous. This creates transformation zone (TZ) with squamous-columnar junction (SCJ) at top end of this. TZ is unstable so more vulnerable to oncogenes, especially human papilloma virus (HPV), so is where cervical neoplasia arises. Junction migrates down with puberty, pregnancy and OCP displaying columnar epithelium = ectropion. Migrates up with age so need to ensure all of TZ is visualised.

Uterus – muscular body supported by uterosacral and cardinal ligaments. Usually anteverted (cervix points backwards) and anteflexed. 20% retroverted and retroflexed. Non-pregnancy size – 7x5x3cm, at full term – 30x25x20 (mostly due to hypertrophy). 3 layers of muscle – outer longitudinal, middle thick spiral muscle fibres with blood vessels, inner oblique. Supplied by uterine (and ovarian) artery, from int iliac.

Broad ligament – peritoneum draped over uterus containing fallopian tubes. This also covers sup post vagina as pouch of Douglas.

Round ligaments - remnants of gubernaculum. From fundus of uterus to deep inguinal ring to labia majora. Contain muscle fibres.

Infundibulo-pelvic ligament – contains ovarian vessels.

Fallopian tubes – fimbriae pick up ovum and cilia of endosalpinx transport it. Four parts – cornual, isthmus, ampulla, infundibulum (lateral end, with fimbriae).

Ovaries – steroid production and gametogenesis. Blood supply is ovarian arteries from aorta. Outer cortex of germinal epithelium, then tunica albuginea and inner medulla.

Vulva – Mons, labia major, clitoris, labia minor, vestibule, external urethral meatus, Bartholins gland, hymen.

Perineum – area between vaginal opening and anus. Perineal body is fibromuscular tissue into which levator ani and deep transverse perineal muscles insert.
History

Always ask about:

- Parity and complications.
- Last menstrual period and length of menstrual cycle.
- Contraceptive use and sexual activity.
- Last cervical smear.
- Other common symptoms – pain, heavy bleeding, irregular bleeding, discharge, urinary or bowel symptoms, dyspareunia.

Urogynae history - symptoms, obstetric history, menopause, risk factors (increased IAP, cough, obesity, heavy lifting), does person still want intercourse (type of pessary and surgery), fitness for surgery.

Menorrhagia – markers of severity (anaemia, clots, flooding), thyroid symptoms, medications e.g. anticoagulants, coagulation problems, coil.

Oligo/a menorrhoea – how long, ever been regular, contraception, menarche? Disease specific questions – PCOS (acne, virilisation, hirsuitism), hyperprolactinaemia (drugs, headache, visual field), thyroid, weight change, stress, ovarian failure (hot flushes), recent pregnancy.


Sexual history – current symptoms, sexual contact history (last 3 months usually, gender, regular or non-regular, sexual orientation, type of intercourse, contraception, still in contact), past STIs, recent drug history (resistance) and allergies, HIV risk assessment (no. of partners with unprotected sex, men, foreign, prostitution, drug abuse, blood transfusion).

Pruritis vulvae – precipitating factors, change in routine, washing, soap products, medications, discharge, other skin conditions, atopy.

Post-menstrual Bleeding (PMB) – is woman definitely post menopausal when, how heavy, type of blood, how long, precipitating factors, HRT, other drugs (e.g. warfarin, tamoxifen), cervical screening, parity, other conditions (e.g. diabetes, PCO).
Differential Diagnosis

Menorrhagia - PID, cancer or hyperplasia, bleeding disorders, dysfunctional uterine bleeding, thyroid disease, intramural or submucous fibroids, maybe endometriosis, adenomyosis.

Primary amenorrhoea – hypothalamic (idiopathic, radiotherapy, surgery, craniopharyngomas, anorexia), chromosomal (Turner’s, XY androgen insensitivity), endocrine (CAH), congenital (vaginal septum, absence of a uterus).

Secondary amenorrhoea – physiological (pregnancy, lactation, menopause), hypothalamic, hyperprolactinaemia, PCOS, thyroid disease, adrenal disease, premature ovarian failure, Sheehan syndrome, Asherman syndrome.

Dysmenorrhoea – physiological, endometriosis, adenomyosis, PID, fibroids.

Chronic pelvic pain – endometriosis (related to periods, dyspareunia), adenomyosis, ovarian cyst (unilateral), adhesions (surgical history), fibroids, pelvic venous congestion (controversial, hormone related dilated veins). USS and laparoscopy – will show endometriosis, PID, cysts or adhesions.

Acute pelvic pain – adnexal torsion, ectopic pregnancy, ruptured ovarian cyst, PID.

Superficial dyspareunia – infection, dermatological disease, postmenopausal atrophy, scar from episiotomy, undilated hymen, psychosexual causes.

Deep dyspareunia – PID, endometriosis, pelvic tumours, fixed uterine retroversion, pelvic congestion, bladder or bowel disease (esp IBS), psychosexual causes.

Pelvic mass – ovarian cysts, ovarian cancers, fibroids, abscesses, endometriosis, haematocolpos (pre-pubertal girls), bowel related, exclude pregnancy. USS is best investigation.


Intermenstrual bleeding – endometrial cancer (USS, hysteroscopy), cervical cancer (smears, colposcopy), cervical polyps, ectropion, fibroids, PID, dysfunctional uterine bleeding, coil, progestogens.

Post-menopausal bleeding – endometrial carcinoma (5%), endometrial hyperplasia, atrophic vaginitis, cervical, vulval and ovarian carcinomas, urethral caruncles, vaginal foreign bodies esp pessaries, polyps.
Examination, Investigation and Procedures

Generally in dorsal position or left lateral position (Sims).

**Pelvic examination** – for pelvic organs. Use gloves and lubrication. Elevate and steady uterus and adnexae. Determine uterus size (enlarged with pregnancy, fibroids), position (anteverted, axial, retroverted) and mobility of uterus, excitation tenderness (=tenderness that arises in adnexae when broad ligament is stretched by movement of the cervix) and presence of masses.

**Cuscoe speculum** – to visualise cervix and vaginal walls. For cervical smears and swabs. Dorsal position, insert and rotate to visual cervix (circular in nulliparous, slit in multip).

**Sims speculum** – to assess uterovaginal prolapse and vesicovaginal fistulae. Use in left lateral position with knees drawn up (L leg straighter supporting other leg). Insert one end into vagina and gently pull backwards to visualise anterior wall. Ask pt to cough to see cystocele, stress incontinence, cervical descent. To view posterior wall, sponge forceps are inserted to retract anterior wall and speculum is slowly withdrawn. Ask to cough for enterocoele.

**Smears** - Clearly visualize cervix using bivalve speculum. Insert narrow point of wooden Aylesbury spatula into the endocervical canal. Rotate through 360 degrees in transformation zone (TZ). Spread material evenly on a microscope slide and immerse in fixative. Liquid based cytology is being introduced.

**Cervical screening programme** – All women between 25 and 50 should have smears taken at 3 yearly intervals, 5 yearly after age of 50 until 65. Screening under 25s is not recommended in guidelines as not cost effective, lots of false positives, very few cancers, high morbidity of loops as likely to want children in future and may need further loops, avoid overtreatment as most cases will resolve. Don’t screen over 65s unless never screened before as unlikely to develop new lesions. Screening at 3 yearly intervals picks up 91% of pre-malignant cases, screening yearly would only increase this to 93%. Smears can show mild, moderate or severe dyskaryosis (cytological diagnoses). If mild repeat, if moderate or severe refer to colposcopy. Technically unsuitable smears – about 8% (should be 1% with liquid cytology), due to blood, pus, inflammation, not cells from SCJ or not enough cells.

**Swabs** – High vaginal swabs are taken from the posterior fornix using a bivalve speculum. These are used to detect lower genital tract pathogens e.g. candida or TV. Endocervical swabs are used to detect pathogens that infect columnar epithelium and may cause PID e.g. Chlamydia and gonorrhoea.

**Colposcopy** – used for all women who have had a smear showing borderline changes or mild dyskaryosis that do not resolve spontaneously, moderate or severe dyskaryosis, suspected neoplasia. It is a binocular microscope to enable TZ to be visualised. Clean mucus of the cervix and apply acetic acid to stain abnormal areas acetowhite or iodine to stain normal areas brown. Density of whiteness, punctuation, mosaicism and abnormal vessel formation suggest degree of abnormality. Forceps may be needed to expose transformation zone if it lies in the endocervical canal (more common if older). Punch biopsy colposcopically abnormal areas. Can also use diathermy loop excision of TZ which treats and gives sample for histology.

**Ultrasound** – high freq sound waves reflected from anatomical structures, fluid looks black. Can use abdominal probe or vaginal probe (don’t need full bladder). Useful for almost all pelvic abnormalities. Can use Doppler to demonstrate blood flow. Can detect gestational sac of intrauterine pregnancy from 5th week and embryonic structures from 7th.
Notes on Gynaecology. Author: Liz Tatman

Hysterosalpingo contrast sonography (HyCoSy) – technique to assess tubal patency. Insert echogenic suspension into uterine cavity and use ultrasound. Indirect so shows tubal patency but not that oocyte can enter tube and does not give any information about pelvis.


Hysteroscopy – allow visual examination of uterine cavity. Insert telescope under local or general anaesthesia and pass fluid to dilate. Can see polyps, fibroids, adhesions and abnormal epithelium e.g. hyperplastic, atrophic, malignant. Can also carry out treatment e.g. take biopsies, divide adhesions and remove polyps or misplaced IUCDs and laser lining in menorrhagia. Complications include perforation, infection, bleeding, cervical shock or fluid overload. Can do as day case or in outpatients, minimal recovery time.

Laparascopy – general anaesthesia with patient paralysed and ventilated. Inflate with CO2. Modified Lloyd-Davies position. Used to investigate and/or treat pelvic pain, ectopic pregnancy, infertility, sterilisation, trauma, lost IUCD, gynaecological cancer (lymph node sampling etc. for staging), tubal patency (lap and dye). Complications – bleeding, infection, shoulder tip pain, misplaced gas, perforation of organs, hernia. Morbidity – 20/100 000 cases of major vessel damage, recognised bowel damage and unrecognised bowel damage.

LLETZ – either under GA or in colposcopy with local. With diathermy loop remove transformation zone in one go. Send for histology. May get bleeding afterwards, should refrain from intercourse. Complications – pain, bleeding, infection, incomplete removal of cells.

Abdominal hysterectomy – open abdomen with laparotomy, remove pedicles of uterus and take out uterus under GA. Hospital stay 6 days. Risk of damage to ureters, bladder or bowel.

Vaginal hysterectomy – need a mobile adhesion-free non-enlarged uterus. Good if uterine prolapse. Remove pedicles of the uterus from the bottom up through the vagina – uterosacral and cardinal ligaments, uterine arteries, fallopian tubes, round ligaments, broad ligaments, ovarian ligaments. Tie ligaments together and stitch vault of vagina to them to prevent later prolapse. Complications are bleeding, infection, vault prolapse, damage to bladder or bowel. Need 3-5 days in hospital but then have to take it easy for at least 6 weeks to allow structures to heal. Advantages over TAH - less postoperative bowel dysfunction, shorter anaesthetic time, no abdo scars,

Endometrial ablation – GA, using hysteroscope remove all of endometrium with electrocoagulating loop (induces iatrogenic Ascherman’s syndrome with adhesions). Need 1 day in hospital and keeps uterus. Main risks are uterine perforation, bleeding, bowel damage, endometrium returning. 70% success, 50% amenorrhoea.

Post operative care – encourage to move about, expect some bleeding for up to 2wks, abstain from intercourse for 6wks.

Radiotherapy – good for neoplasia confined to the pelvis. Side effects: short term - skin erythema and hair loss, urinary urgency and frequency, nausea and vomiting, abdo pain, diarrhoea, bone marrow suppression. Long term in pelvis – fistulae, stricture formation, sterility. Can be external or brachytherapy.

Management – conservative (behavioural advice, reassurance, alternative therapies, weight loss), medical (divided into hormonal and non-hormonal), surgical (conservative or radical, laparoscopic or open), other (e.g. IVF).
In general for malignancy – if localised then surgical excision, if confined to pelvis radiotherapy and if more widespread chemotherapy.
Complications – anaesthetic, general (pain, bleeding, infection, DVT, scarring), specific.

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Endocrine Disorders

Oestradiol
synthesised from cholesterol, metabolised to oestrone and oestriol (which are excreted in urine).
Released by maturing follicle.
**Action** – oestrogens cause pituitary inhibition, secondary sexual development, fusion of epithyses, myometrial hypertrophy, endometrial hyperplasia, favourable cervical mucus, tendency to thrombosis.
**Pharmacology** – synthetic preparation is ethinyloestradiol. Used in combined pill, HRT (after menopause, Turner’s syndrome, if have uterus need to oppose with cyclical progesterones to prevent hyperplasia). Metabolised by liver, can give orally, vaginally, transdermally or sub cut implant. Don’t give in oestrogen dependent tumours, liver disease, thromboembolic history or hypertension.
Antagonists to oestrogen are clomiphene and tamoxifen.

Progestogens
Produced by corpus luteum and placenta.
**Action** – causes pituitary inhibition of FSH and LH, raised body temp, secretory changes, viscous cervical mucus, maintenance of pregnancy.
**Pharmacology** – various synthetic progestogens (natural, testosterone derivatives, less androgenic progestogens). 2nd generation e.g. norethisterone compared to 3rd generation e.g. desogestrel have less thromboembolic risk, worse lipid profile, more androgenic. Used in COP, POP and high dose palliatively for uterine cancer. Antagonist is mifepristone (used for termination of pregnancy – detachment of trophoblast, lutoelysis, reduced HCG).

FSH – released from ant pituitary, stimulated by hypothalamic GnRH. Production reduced by increasing oestrogen levels.
LH – released from pituitary by GnRH. Bolus released midcycle causes ovulation.
Androgens – produced by ovary and adrenals.

PCOS
= syndrome of oligomenorrhoea, hirsutism, obesity and infertility. Spectrum of disease – normally cycling women may have polycystic ovaries.
**Diagnosis** – 2 out of oligo/amenorrhoea, clinical or biochemical hyperandrogenism, scan findings (>12 follicles between 2-8mm or >10cm3). Incidence varies from 5-30% depending if clinical or ultrasound diagnosis.
**Pathology** – disordered pituitary gonadotrophin secretion and ovarian steroid production. May be due to ovarian enzyme abnormality or disordered feedback on pituitary. Leads to increased LH, anovulation and increased androgen production. Obesity reduces levels of sex hormone binding globulin so increases circulating concentrations of free androgens and therefore hirsuitism etc.
**Management** – investigate by serum LH, FSH (LH is 3x higher than FSH, but no longer diagnostic), prolactin and testosterone and USS of ovaries. Hirsuitism – cosmetic e.g. waxing, cream, OCP, anti-androgens (cyproterone acetate). Obesity – diet and metformin. Oligo/a- menorrhoea – OCP, clomiphene (also induces ovulation). Increased risk of endometrial hyperplasia and adenocarcinoma, probably reduced by use of OCP to cause regular bleeds.

Hyperprolactinaemia
Leads to secondary amenorrhoea, infertility, galactorrhoea. Can be due to pituitary adenoma, drugs e.g. phenothiazines, primary hypothyroidism, pregnancy. Treat by cabergoline or bromocriptine, hypophysectomy, Rx.
Fertility Control

**Subfertility**
Failure to conceive after 12 months of regular (2/3 times per week) unprotected intercourse. Primary or secondary (previously been pregnant). Affects 1 in 6 couples.

**Fecundability** – the rate of conception in a population in a given time – about 20% per month. 90% of couples conceive within 18 months. Falls with age after about 31 for females.

**Causes of primary infertility** – 30% idiopathic, 25% male, 20% ovulation (hypogonadotrophic – low FSH and LH e.g. weight disorder, hypothyroidism – increased TRH downregulates pituitary GnRH, pituitary disease e.g. Kallman syndrome; hypergonadotrophic – ovarian failure, PCOS; hyperprolactinaemia – drugs, idiopathic, pituitary disease), 15% tubal, 10% endometriosis. (Remember in backwards order ETOMIC.)

**Causes of secondary infertility** – 40% tubal, 20% idiopathic, 20% male, 15% ovulation, 5% endometriosis.

**History and examination** – mainly to identify cause. Regular periods (if regular 95% are ovulating), history of infection, pelvic surgery or coil use causing tubal scaring, endocrine disorders (PCOS, atrophy of premature menopause, hyperprolactinaemia, extremes of weight, stress, thyroid disease), pelvic pathology (scarring, fibroids, cysts). Good prognosis – age <30, duration of infertility <3 years and previous pregnancy. In male – operations and infections, stress, testes (reduced size and increased firmness if fibrosis and spermatogenic failure, swollen epididymis if blockage), drugs (anabolic steroids, cannabis).

Generally investigate after 12 months, earlier if known disease or older. Investigations centred around main causes i.e. tests of ovulation, tubal patency and semen quality.

**Male investigations:** Semen analysis – 2 occasions, abstain from ejaculation for 3 days. spermatogenesis takes 70 days but usually repeat immediately. Normal - density of >20mil sperm/ml, >50% forward progression and >30% normal morphology. High gonadotrophin levels with testicular failure, normal with reduced testosterone in hypogonadotrophic hypogonadism.

**Female investigations:** Tests for ovulation (mid luteal day 21 serum progesterone – should be >20, basal body temp, ultrasound follicle monitoring, laparoscopy for corpus luteum, LH surge, mucus changes, mittelschmerz, endometrial biopsy – this is best but invasive), FSH and LH levels, testosterone, prolactin, thyroid function, tests for ovarian reserve (FSH - >10 days 2-5 is abnormal, inhibin B – falls if abnormal, scan). Scan ovaries and uterus. Laparoscopy for visualisation of pelvis (endometriosis, anatomical abnormalities). Tubal patency – lap and dye (risk factors), Hysterosalpinography (now replaced by HyCoSy). Post-coital test – aspirate cervical mucus at time of ovulation within 6 hours of intercourse, tests capacity of sperm to remain motile, effective coitus. Tests for antisperm antibodies.

**General management** – folic acid, stop smoking, limit alcohol, avoid polypharmacy, lose weight.


**Female management** – hormonal therapy for ovulatory disorders (clomiphene, human menopausal gonadotrophins – risk of ovarian hyperstimulation). Monitor to avoid ovarian hyperstimulation and multiple order pregnancy, risk of ovarian cancer. Tubal damage - surgery (not very effective - adhesiolysis, salpinostomy, reimplantation to myometrium, making tubes patent does not necessarily restore function - also need nutrients and transport for embryo.), IVF techniques.
PCOS – weight loss, metformin, clomiphene (induces ovulation by increasing production of FSH) or HMG (hormone) with LHRH analogues (can cause severe ovarian hyperstimulation), ovarian drilling (can get 85% to ovulate but only 50% pregnant as abnormal eggs). Hyperprolactinaemia – bromocriptine, cabergoline. Endometriosis – conservative endoscopic surgery e.g. diathermy and adhesiolysis (EndoCan study showed this increases fertility), IVF.

IVF - gonadotrophins or clomiphene to develop multiple ovarian follicles (give GnRH analogues before to desensitise pituitairy and prevent premature LH surges), measure oestradiol to confirm stimulation and prevent overstimulation, hCG to cause ovulation, recover oocytes surgically before release from follicle (usually under US guidance with needle through vagina), mix with semen, transfer embryo to uterus after 48 hrs (2-8 cell stage).

GIFT - gamete intrafallopian transfer for mucus hostility or unexplained.
ICSI – intracytoplasmic sperm injection.
IUI – intrauterine insemination.

Prognosis – only 25% will conceive if infertile for >3 years. IVF – nationally 25% per cycle.
Contraception

Counselling – establish why wants contraception, risk status for pregnancy and STI, sexual history, previous contraception used, PMH, DH, motivation to use contraception and likely compliance, type preferred.

Pearl index – no. of women/100 who become pregnant in 1 year using contraceptive method.

Natural methods – avoid intercourse 1 week before ovulation and 2 days after. Predict ovulation by rhythm, temperature or assessing cervical mucus.

Barrier methods – condoms, used correctly with spermicide 2-3 failures/100women years, in practice <4. Also prevent STIs. Caps, e.g. diaphragm, with spermicide is as effective as a condom. Covers cervix. Insert prior to intercourse and leave for at least 6 hours.

Hormonal: Combined oral contraceptive – oestrogen and progesterone. 21 days with 7 day break (withdrawal bleed). Causes inhibition of GnRH, FSH and LH by negative feedback, prevents follicular maturation and make endometrium unsuitable for implantation. Cervical mucus made impenetrable by progesterone. Don’t use with hormone dependent tumours, thromboembolism, arterial disease, focal migraine, clotting abnormalities or liver disease. Smoking, hypertension and diabetes are relative contraindications. Protects against ovarian and endometrial cancer. Good as most effective reversible method, <1 pregnancy per 100 women years. Forgotten pills – if within 12 hours take pill (contraception probably ok), if later then additional contraception for 7 days, if last 7 days of pack go straight to next pack. Progesterone only pills – act on endometrium, cervical mucus and fallopian tubes. Must be taken at same time every day without breaks. 1 per 100 women years. Used during lactation or with contraindications for COCP. Increased risk of ectopics and erratic bleeding are main problems. Depot or progestogen implants – inhibit ovulation, make endometrium atrophic and thicken cervical mucus. Problems are weight gain, delayed return of fertility and bleeding irregularities. Implants – every 3 years.

Post-coital contraception – levonorgestrel 2 x 750mg 12 hr apart. First needs to be within 72 hours. 86% prevention.

IUCD – most contain copper. Inhibits sperm migration and ovum transport and sterile inflammatory response prevents implantation. Safe and effective but needs to be fitted and follow-up care. Failure rate 1.4 per 100 women years. Works if fitted within 5 days of unprotected intercourse. Contraindications – undiagnosed genital tract bleeding, pregnancy, pelvic infection, Wilson’s disease (copper containing), previous ectopic, uterine abnormality. Last 5 years. Complications – pain, vasovagal shock on insertion, uterine perforation, infection (inadvisable in people at high risk of STI), menorrhagia, expulsion, male dyspareunia, ectopic pregnancy. Good as requires minimal input from user.

IUS (Mirena coil) – progesterone releasing coil. Very effective, lasts up to 6 years. Also reduces menstrual blood loss. Erratic bleeding for 6 months often followed by amenorrhoea.

Sterilisation – female – block fallopian tubes by excision or clips (e.g. Filschie). 0.1% failure rate. Consider irreversible. Male – more effective, remove vas deferens, must wait till azoospermia confirmed. No longer need consent of spouse.
Menstrual Problems

**Menstrual cycle**
Pulsatile production of GnRH by hypothalamus stimulates pituitary to produce gonadotrophins. LH acts on thecal cells to stimulate conversion of cholesterol to androstendione and testosterone. FSH acts on granulosa cells to stimulate aromatisation of androstendione to oestrone and testosterone to oestriol.

**Follicular phase** – FSH rises and stimulates follicle development, usually only one follicle matures. Follicles secrete oestrogen causing duct proliferation in breast, favourable cervical mucus and glandular changes in endometrium (proliferative phase). Rising oestrogen causes LH surge and ovulation and expression of progesterone receptors.

**Luteal phase** – corpus luteum secretes progesterone, 0.5 deg elevation in temp, changes in breast and endometrium (secretory phase). Bleeding occurs because corpus luteum function decreases so O and P levels fall. Usually lose about 60ml blood, superficial layer is lost but basal layer remains and regenerates. If implantation occurs, HCG release supports corpus luteum, maintaining endometrium until placenta takes over at 12 weeks. Normal cycle is 21-36 days, with 4-8 days bleeding.

**Follicle development** – recruit many primordial follicles, only one matures. Granulosa cells secrete fluid. Stromal cells form theca interna (androgens) and theca externa. LH causes meiosis to be completed. Granulosa cells become luteal cells.

**Puberty**
Androgen production starts at about 9 leading to increased sebaceous gland activity and hair growth. Secondary sexual characteristics then develop e.g. breast growth (oestrogen), pubic and axillary hair (androgens). Score development of both by Tanner score (0 is not developed, 5 is fully). GnRH starts to be released pulsatilely from hypothalamus so stimulate release of FSH and LH from ant pituitary. First cycles are usually anovular so heavy dysfunctional bleeding. Avg age of menarche is 13.

**Primary amenorrhoea**
= failure to start menstruation, investigate at 16 or 14 if no breast development.

**Aetiology** – usually due to endocrine or anatomical causes (e.g. absence of uterus, imperforate hymen). Divide by whether other secondary sexual characteristics present e.g. breasts, pubic hair, can assess internal organs by ultrasound.

**If secondary sexual characteristics present:** Imperforate hymen – cyclical pain but no bleeding, haematocolpos, bulging bluish membrane vaginally, incise membrane then give COP for a few months. Absent vagina. Testicular feminisation – XY, with androgen insensitivity, female phenotype but no internal organs, breast development due to hepatic oestrogens, need orchidectomy and oestrogen replacement.

**If not present:** Hypothalamic failure – anorexia, surgery, Kallman’s syndrome, failure of FSH and LH secretion. Pituitary tumour. Ovarian dysgenesis – tall, no epiphyseal fusion. Turner’s syndrome – XO, ovarian dysgenesis (streak ovaries), primary amenorrhoea, short stature, sexual infantilism. 5alpha recutase deficiency – as testicular feminisation but at puberty get testosterone production from internalised testes and androgenisation of presumed female, remove testes and give oestrogen replacement to get phenotypic female. General ill health e.g. thyroid, coelia, anorexia.

**If signs of virilisation:** (e.g. hirsuitism, clitoromegaly) Congenital adrenal hyperplasia – XX, ambiguous genitalia, vagina and uterus present, may be defective production of cortisol. Adrenal or ovarian tumour.

**Secondary amenorrhoea**
= absence of periods for more than 6 months.

**Aetiology** – most common pregnancy, lactation or menopause. PCOS. Hyperprolactinaemia – inhibits FSH and LH, due to pituitary tumour, drugs e.g. phenothiaazines, haloperidol, hypothyroidism; treat with bromocryptine.
Hypothalamic – weight loss, stress. Premature ovarian failure – high FSH, menopausal symptoms. Thyroid disease. Rarely Sheehan syndrome – hypopituitarism due to postpartum ischaemic necrosis of pituitary often due to haemorrhage, Ascherman syndrome – normal hormonal profiles and ovulation but amenorrhoea due to uterine scarring, mostly iatrogenic due to severe curettage or ablation.

**Progesterone withdrawal test** – give progesterone for 5 days. If woman bleeds afterwards she has oestrogen in her circulation and a uterus (+ for PCO, - for Turner’s, androgen insensitivity, hypogonadotrophic hypogonadism and premature ovarian failure). If has oestrogen should induce regular withdrawal bleeds to prevent risk of endometrial hyperplasia and adenocarcinoma.

**Menorrhagia**
= >80ml but difficult to measure, so in practice if interferes with life. Severity suggested by – clots, flooding, unable to go out, iron-deficiency anaemia.
**Aetiology** – fibroids or polyps (increase uterine mucosa area), adenomyosis (endometrium in muscle wall), endometritis, IUD, pelvic infection, clotting disorder, hypothyroidism, stopping contraceptive pill (e.g. following sterilisation), functional ovarian tumours.

**Dysfunctional uterine bleeding** – abnormal uterine bleeding after exclusion of pelvic pathology (by smear, ultrasound, biopsy and maybe hysteroscopy, laparoscopy), represents about 50% of women with menorrhagia, usually associated with anovulatory cycles.

**Management** – examination (anaemia, thyroid disease, pelvic masses), blood tests (FBC, clotting screen, TFTs), USS (endometrial pathology, polyps, fibroids, ovarian pathology), endometrial biopsy (if >40yrs).
Having excluded serious pathology - if around menopause or puberty or irregular (i.e. anovulatory) use OCP/HRT, progestogens (prob best if perimenopausal as give regular bleeds and protect endometrium from hyperplastic change) or mirena coil.
If ovulatory then tranexamic acid (antifibrinolytic – during bleeding, not if history of clotting disorder, GI side effects), mefenamic acid (NSAID anti PG – during bleeding, slightly less effective than tranexamic acid but free from side effects), OCP, POP, danazol (esp around menopause, androgenic side effects so only for severe cases where surgery is inappropriate), IUS, GnRH analogues (hypoestrogenic side effects so only severe cases, shrinks fibroids, shows what effect of hysterectomy would be). If fail endometrial ablation or hysterectomy.

**Endometriosis**
Proliferation of endometrial tissues (benign) outside uterine cavity (usually in pelvis), which cause chronic scarring and adhesions.
10% of premenopausal women, 30% of those with infertility. Reduced by taking pill and pregnancy.
**Aetiology** – retrograde menstruation, vascular or lymphatic spread, metaplasia.
**Pathology** – inflammation, fibrosis and adhesions. Deposits typically on ovaries, ligaments and pouch of Douglas.
**Symptoms and signs** – pain (cyclical, worse before menstruation, dyspareunia, backache, dysmenorrhoea), infertility, tender fixed retroverted uterus, ovarian masses (chocolate cysts and adhesions) haematuria (if bladder involved), rectal bleeding (if bowel involved). Can be assymtomatic.

**Management** – diagnose definitively by laparascopy. May need cystoscopy, sigmoidoscopy.
**Medical treatment** - improves in pregnancy so, OCP without breaks, progestogens, danazol (twice daily) or gestrinone (twice weekly) (testosterone derivative, suppresses growth of endometrium), GnRH analogues. Problems – relapse, breakthrough bleeding, PMS like symptoms, androgenic side-effects with danazol, hypoestrogenic symptoms (bone loss, flushes) with GnRH analogues.
**Surgical** – diathermy and division of adhesions laparoscopically. Laparotomy to restore anatomy or pelvic clearance.
Infertility – causes infertility due to direct damage to pelvic organs or production of local cytokines which interfere with processes. Most effective treatment is surgical or IVF.

Dysmenorrhoea

Primary dysmenorrhoea – without organ pathology, usually starting at menarche, spasmodic, present to some extent in most women, generally improves during period. Reassurance and explanation, analgesics, PG inhibitors e.g. mefenamic acid (ponstan), combined pill to prevent ovulation.

Secondary dysmenorrhoea – congestive, begins before menstruation and may be relieved by bleeding, often associated with dyspareunia, usually has a physical cause – endometriosis, adenomyosis or chronic salpingitis.

Other conditions

Cancer risk – high risk with increasing age, post-menopausal, sporadic symptoms. Need more radical investigations and management.

PCB – mostly cervical pathology.

PMB – need to investigate, always USS and pipelle biopsy. More likely to be serious if recurs or is heavy, fresh and prolonged. If atrophic endometrium on scan and no endometrial sample obtained then reassure but stress reporting further episodes. If recurrent or continuous bleeding (25% will have cancer or atypical hyperplasia), or increased thickness on scan - hysteroscopy and biopsy, or hysterectomy.

IMB – consider pregnancy, consider carcinoma (esp if over 40, obesity, oestrogen exposure), USS for endometrial thickness, pipelle biopsy, hysteroscopy.

Ascherman’s syndrome – very rare, presents with secondary amenorrhoea due to adhesions in the uterus and loss of basal layer. Iatrogenically induced therapeutically in endometrial resection or by over-vigorous curettage.

Adenomyosis – endometrial glands found in muscle. Cause dysmenorrhoea, menorrhagia, bulky tender uterus, dyspareunia, reduced fertility, thick enlarged uterus. Treat with progesterones (or danazol or gonadotrophin analogues) or hysterectomy.

Cyclical mastalgia – avoid sat fats, evening primrose oil, bromocriptine, danazol.

PMS – progesterone suppositories, COCP, danazol, fluoxetine (for psychological symptoms).
Cervical Conditions

Cervicitis – chlamydia and gonorrhoea, herpes, can mask malignancy.

Ectropion – bleeding (esp post coital), increased mucus production or discharge and infection. Related to hormones e.g. pregnancy, OCP. Treat by stopping pill or cautery.

Nabothian follicles – trapped mucus, very common, may lead to reporting abnormal looking cervix. No treatment needed.

Polyps – from endocervix, bright red vascular growths. Can cause PCB and IMB. Treat by avulsion, in older women usually need to exclude uterine pathology.

Premalignant conditions
Classification – mild, moderate or severe dyskaryosis (enlarged nucleus, increased chromatin, irregular nuclear borders - cytological opinion). Grading (histologically) as CIN I, II or III depending on thickness of epithelium affected.

Aetiology – associated with HPV types 16, 18 and 32. Generally strains which don’t cause warts, interfere with p53 function so immortalised and Rb gene so fixed in S phase, found in >30% of women in 20s, mostly cleared after a few years. Risk factors – factors which increase likelihood of HPV infection (e.g. early age of intercourse, multiple partners) and smoking.

Management – identify by smears. Emphasise to women that they do not have cancer but it may become cancer if left alone. Refer for colposcopy after 3 borderline smears, 2 mildly dyskaryotic or one moderately or severely dyskaryotic smears. On colposcopy recognise malignant change by acetowhite change, punctuation, mosaic vessels and increased intercapillary distance. CIN I may undergo spontaneous resolution. 20% become carcinoma in 20 years if left. Manage conservatively with regular follow up for CIN I, or with loop excision (LLETZ) for II or III (can do this at colposcopy). This is generally successful at treating. May get burn artefact so higher incidence of reporting incomplete excision. Need cone biopsy if can’t visualise all of TZ or not sure of getting all with LLETZ. Follow up with smear at 6 and 12 months then yearly smears for 5 years (as long as all normal). Complications are infection, haemorrhage, cervical incompetence, cervical stenosis. HPV virus vaccines may be a better way of preventing cancer in the future.

Carcinoma of the cervix
High incidence in developing countries, low in developed due to screening, peaks at age 35 and 64. 95% are squamous cell carcinomas in TZ (5% adenocarcinomas, not detected by colposcopy). Present with vaginal bleeding especially post-coitally or by screening. Look exophytic, friable and bleed easily. Generally direct invasion.

Staging: 1a = stromal invasion, microinvasive – penetration through BM but <3mm deep and lateral extension < 7mm. 1b = clinically visible limited to cervix. II – not extended as far as pelvic side wall or lower 1/3 of vagina. III – confined to true pelvis. IV – outside true pelvis or involving mucosa of bladder or rectum.

Management – generally hysterectomy for microinvasive disease, maybe cone biopsy if want children. For stage 1b and II then radiotherapy or radical (Wertheim’s) hysterectomy. Radiotherapy – ovarian function affected, vaginal stenosis and GI side effects, brachytherapy is locally implanted radiotherapy. Radical hysterectomy – also fallopian tubes, upper 1/3 of vagina, parametrium and pelvic lymph nodes; leaves ovaries and functional vagina, fewer bladder and bowel disturbances, only younger women, with stage I or IIa in reasonable health. Maybe trachelectomy (cervix but not body removed) to preserve fertility. 5 yr survival >80%, 40% if in lymph nodes.
Uterine Conditions

Congenital abnormalities – bicornuate to complete duplication of uterus and cervix.

Fibroids – leiomyomata. >20% of Caucasian women over 30yrs, more in Afro-Caribbeans. Subserous (project from peritoneal surface), intramural, submucous (encroach on cavity) or pedunculated (from subserous or submucous). Increase in size in response to oestrogen, shrink after menopause. Generally symptomless but can cause menorrhagia (submucous or intramural), abdo distension, pressure e.g. urinary frequency, infertility. Pain is rare unless degeneration or torsion. Complications – degeneration (as poor vascularity – hyaline, cystic, red, sarcomatous), calcification, necrosis, infection, torsion. Treat by hysteroscopic resection, GnRH analogues (e.g. zoladex) or danazol to reduce before surgery, myomectomy, uterine artery embolectomy, hysterectomy.

Polyps – can cause bleeding, dysmenorrhoea. Should be removed using polyp forceps or hysteroscope.

Retroverted uterus – 20% of women. Most normal, fully mobile. Sometimes fixed due to inflammation in pelvis (infection, endometriosis), in which case dysmenorrhoea, dyspareunia, infertility.

Endometritis – rare unless factors allowing ascending infection e.g. abortion, childbirth, IUCD insertion, surgery. Causes lower abdominal pain, fever, uterine tendency. Often chlamydia.

Endometrial hyperplasia: Simple – cystic, if not ovulating have continuous high oestrogen, metropathica haemorrhagia (7+wk cycles then heavy bleeding, can mistake for miscarriage), treat by giving progestogens. Complex – normal cells, glandular crowding, treat with progestogens and resample at 3 months. Atypical – abnormal cells, treat as endometrial cancer. May be related to HRT or tamoxifen (competitive oestrogen antagonist).

Uterine cancer
Mostly adenocarcinoma, preceded by hyperplasia. Other rarer cancers with worse prognosis e.g. squamous differentiation, clear cell, sarcoma. Typically older post-menopausal women, rare under 40. Generally presents with PMB – this should always be considered to be due to endometrial cancer until proven otherwise.

Staging – Stage I = body only, II = also cervix, III = confined to true pelvis, IV = outside true pelvis or involving bladder or rectum.

Risk factors – prolonged exposure to oestrogen (nulliparous, late menopause, obese, PCOS), ovulation inducing drugs, blood group A, diabetes, family history, BRCA 1 or 2. Rate increasing – due to increased exposure to oestrogen? as people getting fatter (adipose tissue produces oestrogen), fewer children and HRT.

Management – investigate with transvaginal USS (<5mm is reassuring, only relevant in post-menopausal women), endometrial sampling (pipelle biopsy, not in pregnancy or PID, can cause uterine spasm or perforation, will detect 95% of cases), hysteroscopy. Total abdominal hysterectomy and bilateral salpingooophorectomy and peritoneal washings for staging (lymph node sampling not advocated – ASTEC trial). Maybe adjuvant Rx if high grade or involving outer endometrium. High dose progestogens can delay progress and improve symptoms but is not curative. 70% 5 year survival rate as presents early.
Ovarian and Tubal Conditions

**Tubal infection**
Usually ascending infection through cervix associated with intercourse, transcervical surgery, intrauterine foreign bodies e.g. coil, retained products of conception.

**Acute** – tubes become swollen and torted forming pyosalpinx then hydrosalpinx, acutely unwell with pyrexia etc. Ranges from asymptomatic to acute abdomen with peritoneal inflammation, pelvic abscess, septicemia. Cilia may be destroyed causing later infertility or ectopic pregnancy.

**Chronic** – usually due to lower grade organism e.g. chlamydia. Causes pain, deep dyspareunia, heavy periods, infertility. Laparoscopy may show exudates, thickened tubes, fibrosis, hydrosalpinges, adhesions, clubbed fimbriae (chronic damage), tubal blockage. Treat acute infection and overcome infertility by tubal surgery or IVF.

**Cancer of fallopian tube**
Rare. Bloody watery vaginal discharge, adnexal mass, raised CA125.
Bilateral oophrectomy and TAH with chemotherapy as ovarian cancer.

**Ovarian cysts**
Cause symptoms if press on bladder or rectum, lead to abdominal distension, tort, bleed or rupture or affect hormone production.

**Physiological cysts** – not after menopause, <10cm, get smaller after 3 months. Follicular – follicles which fail to rupture, anovulatory cycles or fertility drugs. Corpus luteum cysts – esp in pregnancy, otherwise amenorrhoea followed by heavy bleeding, which can be intraabdominal. Can cause pain (e.g. if bleed into cyst or tort), in these cases remove, otherwise rescan in 3m.

**Complications of benign cysts** – torsion, haemorrhage into cyst, rupture.

**Endometriomas** – invagination of endometrial deposits on surface of ovary.

**PCOS** – numerous small subcapsular follicular cysts.

**Ovarian tumours**
Can be benign (common) or malignant (14 per 100000).

**Types of tumours:** Epithelial - 90%, either benign, borderline or malignant, serous (like Fallopian tube lining), mucinous (like endocervix), endometrioid, Brenner (like urinary tract, almost always benign, solid, associated with Meigs syndrome). Germ cell teratomas – totipotent, variety of tissues, commonly benign dermoid cyst, 10% bilateral, typically younger women, solid teratoma is malignant. Stromal – rare, secrete oestrogen, malignancy depends on cell, granulomas mostly malignant, thecomas mostly benign, fibromas almost always benign but can cause ascites. Androblastomas – virilisation. Secondary deposits – Krukenberg tumours = bilateral solid tumours, transcoelomic spread e.g. from stomach, large bowel, uterus.

For small cysts (<3 cm) with low risk of malignancy manage expectantly, rescanning in 3 months at different time in cycle.

**Malignancy** – generally symptomless, in advanced disease abdo distension, venous obstruction of legs, pain, supraclavicular lymphadenopathy. Present late with spread unless chance finding on USS, endocrine symptoms.

**Factors suggesting malignancy** – older women, fast growing, fixed, bilateral, ascites, raised CA125 (also increased with PID and endometriosis), >5cm, loculations, solid appearance on USS, pleural effusion (R sided), hepatomegaly. Other risk factors – nulliparity, long HRT, family history.

**Staging** – Ia = on ovary, Ib = both, Ic = surface of ovary (progression to this is the important distinction as once this rapid spread), II = extension within pelvis, III = extension to peritoneal cavity (70% present at this stage), IV = distant spread.

**Management** – preop USS and serum CA125 to estimate chance of malignancy. CA125 – only raised in serous carcinomas, also raised in endometriosis, >35 is significant but generally malignant is in 100s, if raised can be used to monitor post-treatment. Laparotomy for staging (rapid spread within peritoneum - inspection, peritoneal washings, biopsies of peritoneum, omentum etc.) and removal of all visible deposits (= debulking, typically TAH, BSO, omentectomy, lymph node sampling).
After cystoreductive surgery, chemotherapy – taxol, platinum, generally 1 day/month for 6m, typically myelotoxic and nausea but good QOL.

**Prognosis** – 5 year survival: average <20%, Ia 85%, II 40%, IV <5%, generally present late. Kills 6000 per year, 4th most common cancer.

**Screening** – may be good for people at increased risk (FH = >2 1st degree relatives, or BRCA gene). Could use USS (good sensitivity but high false positives) and CA125 (less sensitive more specific) (UKCtox trial), problem is false positives (40 for every case).

## Pelvic pain

Investigations – urine culture (for UTI), pregnancy test, swabs (for infection), USS (masses, cysts, swollen tube, uterine size, fibroids, fluid in pouch of Douglas).

## Vulva and Vagina

**Pruritis vulvae** – generally infection (has discharge esp TV, candida), sensitivity to chemicals e.g. soaps, iron deficiency anaemia, glycosuria (probably due to fungal infection), oestrogen deficiency or vulval dermatoses (lichen sclerosis – scarring and resorption of labia minora, check with punch biopsy and potent steroids, Paget’s disease = adenocarcinoma, VIN – hyperkeratotic, needs excision or laser). Chronic epithelial change should be biopsied.

**Vulvodynia** – cause rarely found. Usually symptomatic relief – esp amitryptiline, trimovate.

**Bartholin’s cyst** – gland is deep to posterior introitus, secretes lubricating fluid for intercourse. Duct can dilate forming cyst. Can get infected causing abscess. Treat by marsupialisation.

**Cancer of vulva** – uncommon, SCC, mostly older postmenopausal women and associated with lichen sclerosus. Rise in younger women associated with HPV and smoking. Biopsy, excision (vulva and both groins, poss with inguinofemoral nodes) or chemoradiation.

**Vaginal discharge** – specific infection, physiological (sexual excitement, cervical erosion, IUD), if pus or blood - vaginitis, endometritis, carcinoma.

**Atrophic vagina** – due to lack of oestrogen. Looks thin with no folds, bleeds easily and petechial haemorrhages.
Gynaecological Infections

General management – if one infection is present always exclude others. Trace and treat all contacts.

Symptoms – vaginal discharge, vulvovaginitis.

Candida – most common. May be asymptomatic, not necessarily sexually transmitted. White, particulate, non-offensive, irritant discharge. Red vulva with white discharge. Treat with topical antifungals (creams and pessaries) or systemic antifungals e.g. fluconazole. Predisposing factors – pill, pregnancy, antibiotics, steroids, immunosupression, diabetes.


Bacterial vaginosis – offensive watery grey vaginal discharge, positive amine test, pH >5, clue cells. Gardnerella vaginalis and anaerobes. Treat with oral metronidazole (avoid alcohol) or clindamycin. Not sexually transmitted.

HPV – genital warts (condylomata acuminata) and implicated in cervical neoplasia. Can incubate for up to 8 months. Treat by podophylin, electrocautery, cryocautery, excision. Advise barrier contraception. Worsen in pregnancy.

Herpes simplex – usually type 2, but also type 1. Commonest cause of ulceration. Primary infection – prodromal discomfort, vesicles, ulceration, inguinal lymphadenopathy. Painful. May have secondary bacterial infection. Recurrence is common, but less florid. Acyclovir is useful in primary attack, and may prevent recurrences with continuous treatment. Risk of transmission is greatest within 1yr of first attack, during symptomatic episode, in people with frequent recurrence, with type 2. Advise barrier contraception when visible lesions.

Chlamydia and gonorrhoea – usually asymptomatic in women in lower genital tract. Chlamydia treat with doxycycline (not in pregnancy), erythromycin, azithromycin (single dose). Gonorrhoea (G-intracellular diplococcus) treat with im ceftriaxone or cefotaxime or oral ciprofloxacin.

Non-specific urethritis – chlamydia, ureaplasma, mycoplasma.

Syphilis – treponema pallidum, spirochaetes on dark ground microscope, or serology VDRL. Primary painless chancre and lymphadenopathy, secondary (6 wks) rash, generalised lymphadenopathy, latent (positive serology but no symptoms), tertiary gummas, cardiovascular and neurological complications. Treat with parenteral penicillin.

Pelvic inflammatory disease
Infection of upper genital tract due to ascent of organisms from LGT. Generally chlamydia and gonorrhoea.

Acute PID - spectrum from silent infection to pelvic pain, dyspareuenia, fever, pyrexia, peritonism, cervical excitation, pelvic mass. Can get perihepatitis with chlamydia (Fitz-Hugh-Curtis syndrome). Treat with a/b, analgesia and surgery if fails to improve. Need to refer to GUM clinic for contact tracing. Should have raised WCC, may get positive swabs from cervix

Chronic PID – adhesions, pain, menorrhagia, discharge, infertility. Long term sequelae due to tubal damage – infertility in 15-20% cases and about 10x increased risk of ectopic pregnancy.
Urogynaecology

**Urge incontinence**
Involuntary loss of urine caused by uninhibited detrusor contractions which cause urgency and frequency and incontinence when intravesical pressure exceeds intraurethral pressure.

**Aetiology** – generally idiopathic detrusor instability, otherwise irritation e.g. infection, stones, tumour. **Management** – MSU for microscopy and culture, urodynamic investigations if any possibility of stress, PMH and DH to exclude causes e.g. diuretics, diabetes. A fluid and micturation diary may be useful to see if fluid restriction is needed. Cystometry shows unstable bladder. Treat by bladder drill (pass urine at time intervals which are gradually increased), drug therapy (calcium antagonists, anticholinergics e.g. tolterodine, oestrogen replacement if PM, ganglion blockers), surgery (bladder transection, clam cystoplasty and sacral neurectomy for difficult cases).

**Genuine stress incontinence**
Involuntary loss of urine when intra-abdominal pressure rises. **Aetiology** – urethral sphincter weakness, may be secondary to multiparity, prolapse, menopause, old age or surgery. Normally urethral neck is intraabominal so pressure transmitted and continence maintained. These factor cause urethral neck to descend and lose this mechanism. **Management** – demonstrate urine loss if patient coughs in Sims position with Sims speculum. Treat infection. Normal cystometry except low urethral closing pressure, check post-micturation US for chronic retention. Pelvic floor exercise with cones (help in most cases). Surgery to return urethral neck to abdomen – colposuspension (elevate bladder neck – 90% cure rate), TVT, anterior colporrhaphy (elevate urethra from below – 50% cure rate), injectables (to bulk up sphincter), endoscopic bladder neck suspension.

**Overflow incontinence**
Frequent involuntary loss of small volumes, poor stream, postmicturition feeling of incomplete emptying. **Aetiology** – obstruction to bladder outflow and bladder atony e.g. MND, drugs, uterovaginal prolapse, local inflammation. Enlarged bladder with large RV. **Management** – cystometry shows delayed first sensation and large bladder capacity. Treat cause or intermittent self-catheterisation.

**True incontinence** – continuous incontinence due to fistula secondary to obstructed labour, hysterectomy complications, carcinoma or radiotherapy. Micturating cystogram or IVP. Manage conservatively to allow to heal or with surgery to remove track.

**Neuropathic bladder** – due to spinal cord injuries, contraction not inhibited.

**Urodynamic investigations**: Fluid output charts. Cystometry – presence or absence of involuntary detrusor contraction. Residual urine measure by catheter.
Uterovaginal Prolapse

Downward displacement of uterus, may also involve bladder, urethra, rectum or bowel.

**Urethrocoele** = lower 1/3 of ant vaginal wall.
**Cystocele** = upper 2/3 of ant vaginal wall.
**Rectocoele** = lower post vaginal wall.
**Enterocoele** = upper post vaginal wall (involving pouch of Douglas). Differentiate this from rectocoele by PR.

**Uterine prolapase** – 1st degree = descent within vagina, 2nd = cervix at introitus, 3rd = procidentia, cervix beyond introitus (this also involves ureters so affects renal function and needs treating, squamous epithelium can get ulcerated). If hysterectomy has been performed may get vaginal vault prolapse.

**Symptoms** – feeling something coming down, dragging feeling, backache and stress incontinence – all are worse on standing. Typically discomfort and backache rather than pain. If cystocele also urinary symptoms e.g. incontinence, feeling of incomplete emptying, recurrent cystitis, residual volume, hesistancy. If rectocoele then constipation or having to reduce prolapse before opening bowels (digation). Haematuria, PMB, rectal bleeding are not related to prolapse and should be investigated.

**Normal support** – ant vaginal wall is supported by pubocervical fascia (symphysis pubis to cervix), post wall by fibrous tissue of rectovaginal septum and levator ani muscles (voluntary muscle inserting into the perineal body and supplied by pudendal nerve – S2,3,4) and uterus by cardinal and uterosacral ligaments. The round ligament holds uterus anteflexed but does not support.

**Risk factors** – multiparity (causes pelvic floor dysfunction and denervation), congenital weakness of pelvic supporting structures, postmenopausal atrophy of supporting tissues, increased IAP (heavy lifting, chronic cough, obesity).

**Prophylaxis and conservative treatment** – avoid traumatic deliveries, stop smoking, HRT.

**Physiotherapy** – pelvic floor exercises, cones and electrical stimulation. Particularly important to prevent recurrence after surgery.

**Pessaries** – can cause vaginal discharge, ulceration (at worse through to rectum) or discomfort (esp as likely to be atrophic vagina, so need to give topical oestrogen therapy), change 4-6 monthly, used if unfit for surgery, while awaiting surgery, if further pregnancies planned, to see if symptoms are due to prolapse. Need correct size – too small will be expelled, to large will cause discomfort. Ring pessary – more common, shelf pessary – used if ring pessaries don’t stay, these are inflexible and have holes to let secretions out, can’t have intercourse, Hodge pessary – no longer used, was to correct retroversion (rigid bent ring).

**Surgery** – most involve incision, making fascia tighter and closing skin, compression pack after to stop haematoma. Anterior repair (cystocele), posterior repair (rectocele), vaginal hysterectomy (for uterine prolapse), Manchester repair (for elongated cervix - shorten cardinal ligaments, amputate cervix and ant repair, used previously as less risk of peritonitis, but lower success rate). Complications – infection (1/3), hole in rectum or bladder, bleeding, narrowed vagina, urinary retention. Various repairs for recurrent prolapse involving stitching vaginal vault to sacrospinous ligament (sacrospinous fixation) or closing vaginal lumen.
The Menopause

Menopause = woman’s last period (avg age is 51). Diagnose when no periods for 6 months or more at climacteric age.
Climacteric = period of ovarian decline including menopause. Ovarian follicles become increasingly resistant to gonadotrophin stimulation.

Symptoms – due to decrease in oestrogen. 15% have moderate to severe symptoms. Immediate problems – irregular often heavy cycles, hot flushes (vasomotor instability), night sweats, genital atrophy, vaginal dryness, urinary frequency, headaches, mood changes. Atrophy occurs in all tissues which are sensitive to oestrogen e.g. breasts, skin, genitalia. Longer term – osteoporosis and CV disease (increase in cholesterol and LDL).

Investigations – FSH level over 30iu/l confirms ovarian failure. Assess bone density by DXA.

Osteoporosis – decrease in osteoblast function, leading to fractures esp radius, neck of femur (20% mortality in first year, earliest site of loss in Ward’s triangle) and vertebral spine (wedge fractures causing dowager’s hump). Maximum bone density in 30s (contentious), need adequate calcium and weightbearing exercise, after menopause lose about 3% per year.

Atrophic vaginitis – common, soreness, dysuria and dyspareunia, treat with topical oestrogens (must not be unopposed in women with uterus).

HRT – for hypooestrogenic symptoms and to prevent osteoporosis. Oral – continuous oestrogen and progestogens for 12 days per month; >1yr after LMP then both continuously. Parenteral – patches, creams, implants). If have uterus need progestogens as well. Generally take for 2-5 years. Increased risk of breast cancer and thromboembolic disease. Contra-indications – breast cancer or oestrogen dependent tumours, thromboembolic disorder, liver disease. Alternative is tibolone – oestrogenic, progestogenic and weak androgenic activity – treats hot flushes and prevents against osteoporosis (can also use as add-back therapy for hypooestrogenic symptoms associated with GnRH analogue treatment in pre-menopausal women). Selective oestrogen receptor modulators exist – beneficial effects on bone and arteries without stimulating endometrium and breasts e.g. raloxifene (at moment only for women at high risk of osteoporosis).

Contraception – generally can stop contraception 12 months after last period – HRT is not contraceptive.

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. 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.

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