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Menstrual disorders in adolescence

Early Pregnancy and Adolescent Gynaecology GP Study Afternoon

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Introduction

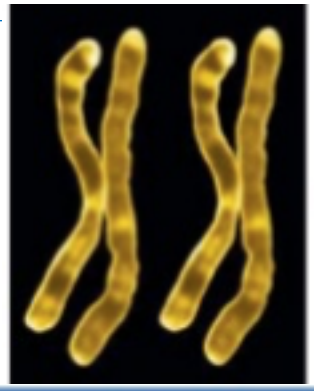
- Menstrual dysfunction in adolescence common
- 25% had significant dysfunction affecting life activities and school absence
- Serious pathology rare
- Specialist input may be required
- Challenging as most evidence based on adult women

Aims and learning objectives

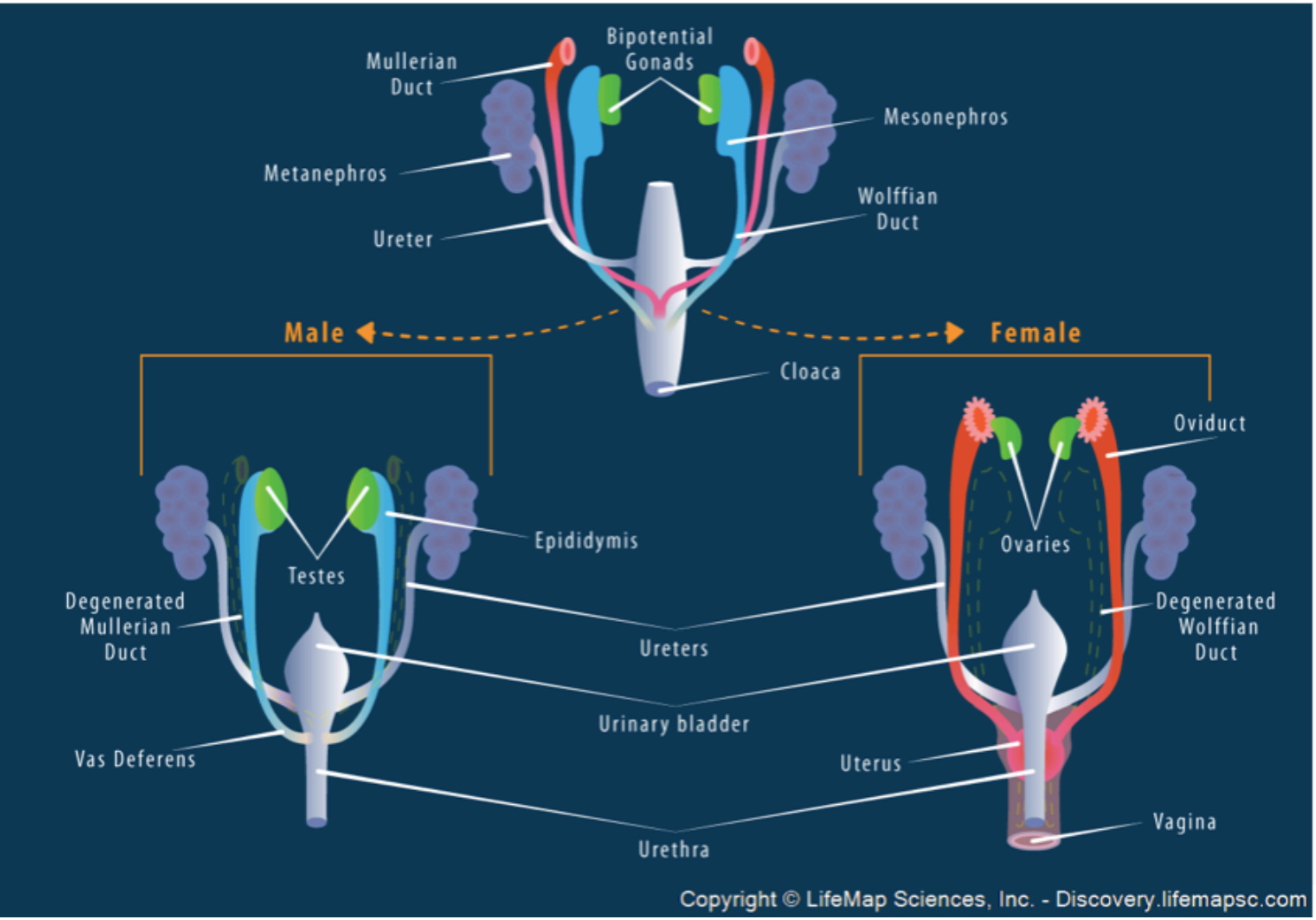
- Female development and menstrual cycle
- Describe common features of menstrual dysfunction
- Investigations that should be performed
- Describe treatment options
- Management of complex patients

XX embryology

- In females, genital organs comprise gonads, reproductive ducts and external genitalia
- Gonadal differentiation occurs before the end of the embryonic period
- Both reproductive ducts and external genitalia differentiate before end of first trimester
- Development of female genital tract continues in utero. The gonads descend in utero in XX
- Maturation of genital tract is continuous during childhood and puberty



In-utero development female gynaecological tract	
Phase of genital development	Gestational age weeks
Indifferent gonadal phase	4–6
Gonadal differentiation	7
Ductal differentiation	9–11
External genitalia differentiation	10–12



Bipotential stage differentiation to the male and female genital system.

Top: At the bipotential stage, both Mullerian and Wolffian ducts are present.

Bottom left: In the male embryo, the Mullerian duct degenerates under the influence of AMH, secreted by the testicular Sertoli cell, and each testis connects to the Wolffian duct through a series of tubules. During further development, the Wolffian duct gives rise to the efferent ductules, epididymis, ductus deferens, ejaculatory duct, and the seminal vesicle under the control of androgens produced by Leydig cells.

Bottom right: In the female embryo, the Wolffian duct degenerates, while the Mullerian duct contributes to the formation of the female reproductive organs. The distal ends of the paired Mullerian ducts fuse to form the vagina and uterus. The proximal un-fused portions become the oviduct (Fallopian tubes).

Development Upper Genital Tract - Fallopian Tubes, Uterus, Cervix, upper Vagina

Paramesonephric (Müllerian) ducts arise from mesoderm lateral to the mesonephric ducts (week 7). They grow caudally, coursing lateral to the urogenital ridges (week 8). They fuse (Müllerian organogenesis) - initial development **upper two-thirds of vagina, cervix, uterus and fallopian tubes**

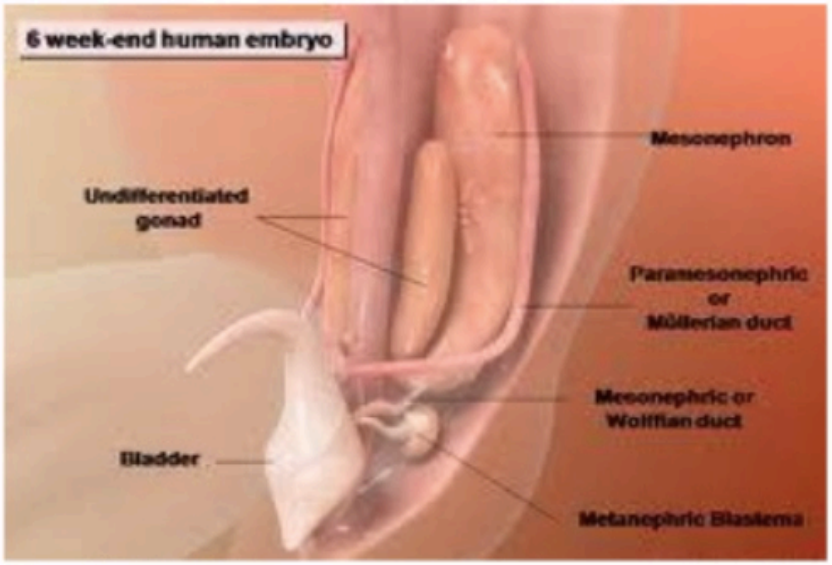
The cranial end of the fused ducts the future uterus contains mesoderm that forms **uterine endometrium and myometrium**.

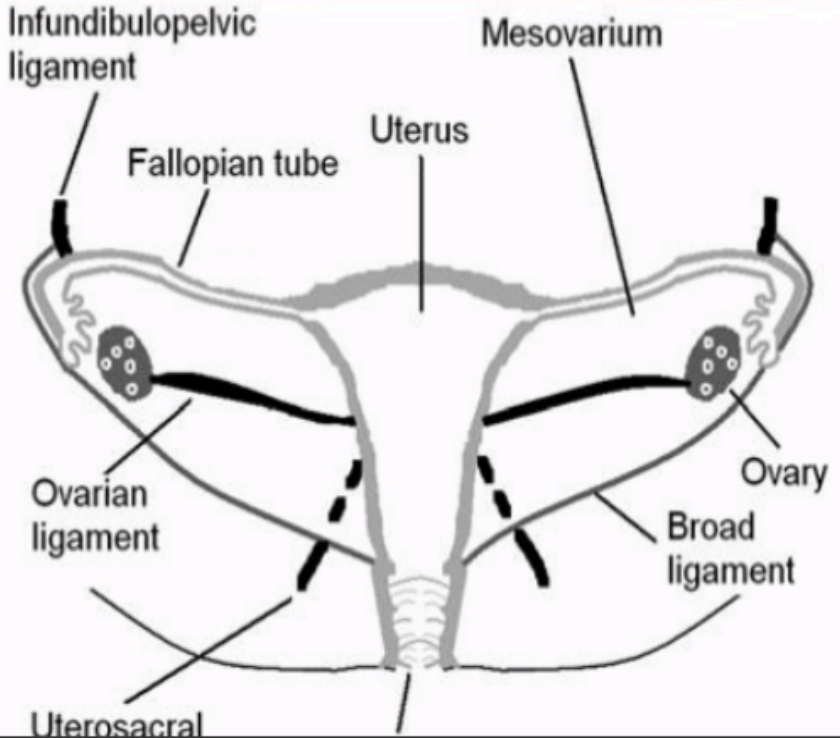
Unfused cranial ends of the paramesonephric ducts (funnel shaped) remain open as the fimbriale of the **fallopian tubes.**

Caudal end of fused ducts forms **upper two-thirds of the vagina**.

Lateral fusion of the paramesonephric ducts (week 7-9) when lower parts paramesonephric ducts fuse. At this stage midline septum is in the uterine cavity regresses week 20 (uterine septal defects).

Vertical fusion (week 8) when the lower most fused paramesonephric ducts fuse with the ascending endoderm of the sinovaginal bulb. **Lower third of the vagina** is formed as the sinovaginal node (bulb) canalises.





Menarche and Physiological Changes

Puberty

At birth, females have a predetermined number primordial follicles arrested during meiosis 1 at the diplotene stage of prophase until stimulation at puberty. Hypothalamus is in a quiescent state.

At approximately 8 years, GnRH is synthesized in the hypothalamus and released.

The adrenal cortex begins to produce DHEA initiating **adrenarche** (ie, the development of sexual hair).

Progression of puberty begins with breast budding **(thelarche),** accelerated growth, and menses **(menarche).**

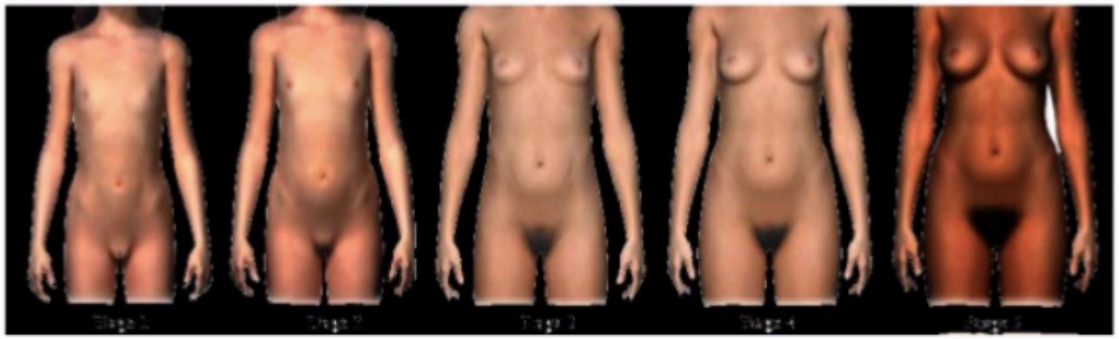
Pubarche, which is independent from GnRH function, typically occurs between breast budding and accelerated growth but may occur anywhere along the puberty timeline.

USA average age at menarche is 12.6 years, (R 9-15 years). 16 years is 3 SD's above

Menarche and sustained menstrual cycles requires normal function of the endocrine **HPO axis** any disruption in this axis may result in amenorrhea/oligomennorhoea.

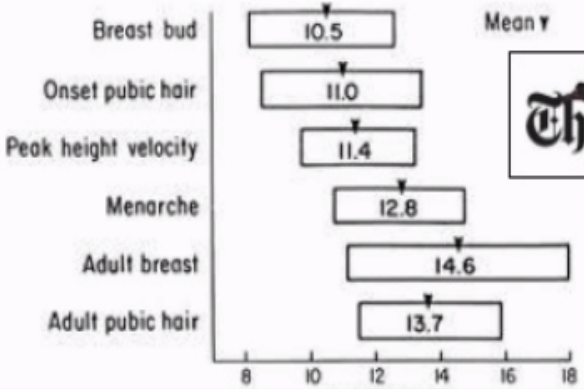
Defining the level of primary dysfunction is critical in determining the pathophysiology of amenorrhea.

Tanner stages 1-5



Stage I (Preadolescent) - Only papilla elevated above level of the chest wall.
Stage II - (Breast Budding) - Elevate breasts/ papillae (small mounds), wider areola
Stage III – breasts/areolae continue enlarging no separation of contour.
Stage IV – areola/papilla elevated, secondary mounds, increase overall breast tissue.
Stage V – Mature breasts, papillae extend above breast contour, areolar recession.

Stage I - Vellos hair over pubes, no more than on abdominal wall. No sexual hair.
Stage II - Sparse, long, pigmented, downy, straight/slightly curled, hair along labia.
Stage III - Darker, coarse, curlier hair now spread sparse over junction of the pubes.
Stage IV – Adult hair distribution , reduced total quantity, no hair on medial thighs.
Stage V - Hair adult quantity/type, appears as inverse triangle (feminine type). Spread to medial surface thighs but not above the base of the inverse triangle



Puberty Before Age 10: A New 'Normal'?

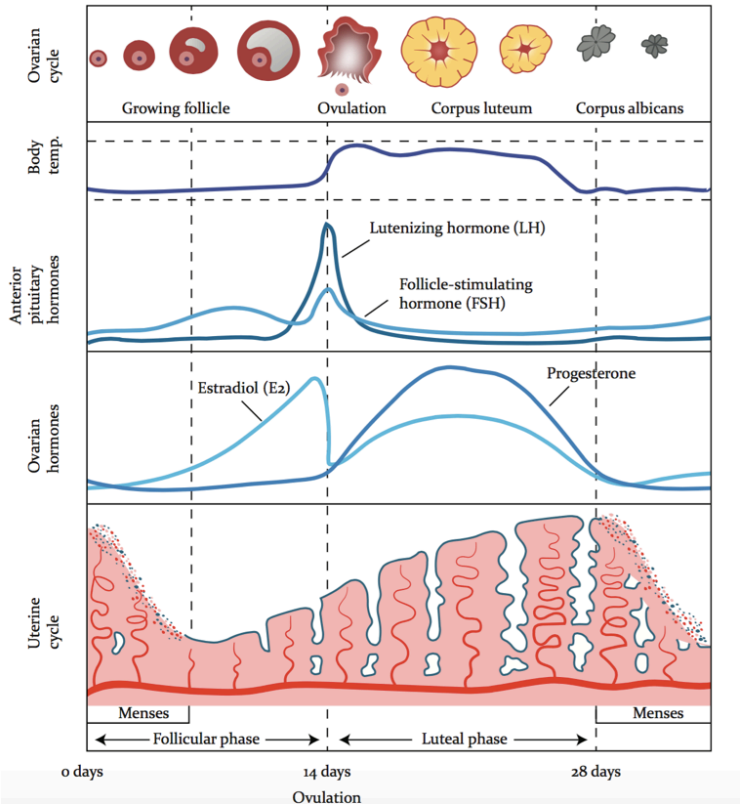
30th March 2012

Puberty and Menarche

- Age determined by general health, socio-economic and nutritional factors
- Mean age 12-13 year olds
- Cycle 2- 7/ 21-45
- Three to six pads or tampons per day

Menarche and the HPO axis

- Immaturity of HPO axis in first 2 years following menarche, results in more than 50% of cycles being anovulatory
- Irregular cycles – 20 to 90 days;
  - >90 days are 95th percentile for length – warrants investigation
- After 1-2 years, capacity for oestrogen positive feedback to anterior pituitary develops with subsequent LH surge and ovulation results in more cycle regularity



Result of anovulatory cycles

- Heavy bleeding
- Prolonged bleeding
- Painful periods – if heavy; become more painful in ovulatory cycles due to circulating prostaglandins
- Iron deficiency anaemia

Amenorrhoea

PRIMARY

- Hypothalamic/pituitary disease
  - Hypog hypog
- Congenital GnRH deficient
  - Idiopathic hypog hypog
- Constitutional delay of puberty
- Hyperprolactinemia
- Ovarian aetiologies
  - Gonadal dysgenesis
  - Turner syndrome
  - PCOS
- Congenital anatomic lesions
  - Imperforate hymen
  - Transverse septum
  - MRKH

SECONDARY

- PREGNANCY
- Hypothalamic causes
  - Idiopathic
  - Endocrinopathies
  - Stress/exercise/eating disorders
  - Weight loss
  - Chronic illness
  - PCOS
- Pituitary causes
  - Lesions, trauma
- Ovarian causes
  - Premature ovarian insufficiency
  - Ashermans syndrome

History

- With and without parents if appropriate
- Menstrual history – age of menarche, regularity, duration, associated pain, acne, hirsutism
- Bleeding history – dental procedures, surgery, nosebleeds
- Sexual history
- Past medical history
- Medications
- Family history – bleeding d/c, VTE,
- Social history – school attendance, bullying
- Safeguarding

Examination

- Observations, height, weight, BMI
- Assessment of anaemia, hirsuitism, acne, acanthosis nigricans, bruising
- Assessment of secondary sexual characteristics
- Tanner staging
- Presence/absence of pubic hair
- Abdominal palpation - ?pelvic masses
- Clitoral size (pre-menarchal is 3mm)
- Configuration of hymen
- Signs of oestrogenisation – moisture, thickness, pinkish
- Hygiene
- Abnormalities – discharge, discoloration, trauma, anatomical defects
- Internal exam – only if sexually active

Basic investigations

- FBC/ferritin
- Clotting
- Pelvic ultrasound (TA): ET may be thick, multifollicular ovaries
- Urinary pregnancy test if appropriate

- Consider:
  - Thyroid function
  - Prolactin
  - LH/FSH
  - Oestradiol
  - SHBG
  - Testosterone
  - Sexual health screen
  - Von willebrand panel

  - Additional: Karyotype, MRI brain

Treatment

- Depends on cause
- Consider if needs contraception, other symptoms e.g. hirsutism, acne
- Treat anaemia
- If anovulatory cycles – reassurance, non hormonal treatments

Drug	Dose	Timing and duration of therapy	Efficacy rates	Common side effects	Contraindications
NSAIDS (Mefenamic Acid)	500mg TDS (licensed for girls 12-17years)	To be taken on first day of heavy bleeding, maximum duration 3 days.	25-50% reduction in menstrual blood loss[16].	GI discomfort, diarrhoea (discontinue if occurs), nausea, vomiting, headache, dizziness.	History of GI bleed, recurrent GI ulcers, IBD
Tranexamic Acid	1g TDS or QDS (licensed for girls 12-17years)	To be initiated when menstruation has started, to be taken for up to 4 days; maximum 4g per day.	50% reduction in menstrual blood loss[17-18].	Diarrhoea (reduce dose if occurs), nausea, vomiting	Fibrinolytic conditions, history of convulsions, thromboembolic disease

Hormonal treatment
Progesterone

- Progesterone: good for regularity and reducing flow
- Cyclical (21 days) or continuous: can be used to defer period e.g. for exams etc; start 5 days before event
- Options:
  - Norethisterone 5mg TDS
  - Medoxyprogesterone acetate (provera/MPA) 10mg tds
- No RCT to compare
- NE more androgenic: acne, hirsutism
- SE: headache, mood disturbance, breast tenderness
- NOT contraception
- Very low VTE risk (background risk 1.83/10,000 women per year aged 15-19yo)

POP

- Low dose oral progesterone, continuous
- Thickens cervical mucus and in some cases inhibits ovulation
- Contraception (3 hour window)
- E.g. desogestrel 75mcg, micronor (NET 350mcg)
- By 11-13 months, 50% will have infrequent bleeding or amenorrhoea in some
- SE: BTB, prolonged bleeding
- Can increase to double dose 150mcg (off license but effective)

COCP

- Inhibits production of LH/FSH from anterior pituitary and so stops ovulation
- Contraception
- Treats irregular menses, heavy bleeding and dysmenorrhoea
- Tricycle or continuous
- SE: breast tenderness, mood changes, nausea and vomiting, headache
- VTE risk: 2x higher with COCP; 3rd generation (desogestrel, gestodene) 2x higher than 2nd generation (NET, levenorgestrel)
- No evidence of weight gain
- Will not affect final height
- NO evidence will bring forward sexual debut
- Risk of breast cancer overall low
- CI: thrombophilias, migraine with aura, hypertension,
- Check BP and annual review

Types of COCP and their progestogens

- Usually EE with a progestogen
- Progestogens classified according to the time of introduction

1st Generation	2nd Generation	3rd Generation	4th Generation
Norethisterone e.g. Loestrin	Levonorgestrel e.g. microgynon/ rigevidon	Desogestel e.g. Marvelon, merclinon	Drospirenone e.g. Yazmine or Yaz
		Gestodene e.g. gynera/ femodene	Dienogest e.g. Qlaira (E2valerate)
		Norgestimate e.g. cilest now lizinna (consider if acne or BTB)	
Moderate androgenic	Strong progesterone and androgenic property	Anti-ovulatory, suppression gonadotrophin	Anti-androgenic and anti-mineralocorticoid

Activity of Progestin Agents

Generation	Progestin	Estrogenic	Progestational	Androgenic
First	Norethindrone	++	++	++
	Ethynodiol diacetate	++	+++	+
	Norgestrel	–	+++	+++
	Norethindrone acetate	++	++	++
Second	Levonorgestrel	–	++++	++++
Third	Norgestimate	–	++	++
	Desogestrel	+/–	++++	++
Fourth	Drospirenone	–	+/–	–

+/– indicates low to no activity.
– indicates no activity.

Adverse Effects Associated with Type of Hormonal Activity

Estrogenic	Progestational	Androgenic
Bloating	Headache	Acne/oily skin
Nausea/vomiting	Breast pain/tenderness	Weight gain
Breast fullness	Hypertension	Hirsutism
Breakthrough bleeding		Fatigue
Irritability		Depression
Headache		
Hypertension		

Risk of DVT
Lidegaard et al, 2011

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Group	Women years	No of events*	Crude incidence per 10 000 user years*	Adjusted relative risk† (95% CI)
Non-use	4 960 730	1812	3.7	1 (reference)
Progestogen with 50 µg ethinylestradiol:				
Norethisterone	6848	11	16.1	5.66 (3.12 to 10.3)
Levonorgestrel	23 691	31	13.1	3.54 (2.48 to 5.05)
Progestogen with 30-40 µg ethinylestradiol:				
Norethisterone	27 355	10	3.7	1.57 (0.84 to 2.92)
Phasic levonorgestrel	105 970	89	8.4	2.28 (1.85 to 2.83)
Levonorgestrel combined	104 251	78	7.5	2.19 (1.74 to 2.75)
Norgestimate	267 664	165	6.2	2.56 (2.18 to 3.01)
Desogestrel	170 249	201	11.8	4.21 (3.63 to 4.87)
Gestodene	668 355	738	11.0	4.23 (3.87 to 4.63)
Drospirenone	286 859	266	9.3	4.47 (3.91 to 5.11)
Cyproterone	120 934	109	9.0	4.10 (3.37 to 4.99)
Progestogen with 20 µg ethinylestradiol:				
Desogestrel	470 982	322	6.8	3.26 (2.88 to 3.69)
Gestodene	472 118	321	6.8	3.50 (3.09 to 3.97)
Drospirenone	23 055	23	10.0	4.84 (3.19 to 7.33)
Progestogen only:				
Norethisterone	44 168	9	2.0	0.56 (0.29 to 1.07)
Desogestrel	29 187	6	2.1	0.64 (0.29 to 1.42)
Levonorgestrel releasing intrauterine device	155 149	55	3.5	0.83 (0.63 to 1.08)

When to start the pill…

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- If not sexually active, can start anytime in cycle

- If sexually active and to be used as contraception, start on day one (first day of period) of next cycle

LARCs
Mirena IUS

- E.g. Mirena t shaped IUS: 20mcg levonorgestrel/ day
- Prevents endometrial proliferation, thickens cervical mucus, inhibits ovulation in 25%
- Contraception, treatment of HMB and dysmenorrhoea
- Lasts 5 years
- 65% at one year have amenorrhoea or light bleeding
- Requires insertion – screen for STI beforehand
- can be done under GA if not SA
- SE: initial irregular PVB, mood changes, acne, headache,

LARCs
Depo provera and Implant

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- 150mg Depoprovera MPA every 12 weeks
- 68mg nexplanon implant
- Long acting reversible contraception
- Inhibits ovulation
- 70% amenorrhoeic within 12 months with depo / 20% with implant
- SE with depo
  - bleeding problems eg. Irregular or BTB
  - weight gain (in those already overweight)
  - reduce BMD (caution in those at high risk e.g. immobility, steriod use)

	Does it reduce blood loss?	Can it reduce pain?	Can it regulate cycle?	Does it provide contraceptive cover?	Contraindications	License
Tranexamic acid	Yes, 50% reduction in blood loss	No	No	No	Personal or family history of thromboembolic disease	Used in children >1 month
Mefenamic acid	Yes, 20% reduction in blood loss	Yes, anti-inflammatory, inhibits prostaglandin synthetase	No	No	Caution in asthma and renal impairment	Age >12 years (used off-label in under 12-year-olds)
Cyclical progestogens	Yes, 80% reduction in blood loss	No	Yes	No	Liver tumours, genital/breast cancer, severe arterial disease, acute porphyria	Age appropriate to stage in puberty
POP	No	No	No	Yes	Liver tumours, genital/breast cancer, severe arterial disease, acute porphyria	Refer to individual preparation
COCP	Yes, >40% reduction in blood loss	Yes, 50% reduction in menstrual cramping	Yes	Yes	Previous thromboembolism or multiple risk factors, migraine with aura, BP >160/95 mm Hg, pulmonary hypertension, liver disease/tumours, systemic lupus erythematosus, acute porphyria, gallstones, breast cancer, haemolytic uraemic syndrome	Refer to individual preparation
Depo-Provera	Yes, 70% amenorrhoea by 12 months	Yes, secondary to amenorrhoea	Yes, can cause amenorrhoea, but 50% discontinuation due to irregular bleeding	Yes	Liver tumours, genital/breast cancer, severe arterial disease, acute porphyria	Age >12 years
Mirena IUS	Yes, 65% amenorrhoea or light bleeding by 12 months	Yes, effective treatment for dysmenorrhoea and endometriosis	Yes, can cause irregular bleeding for first 3–6 months, then 65% amenorrhoea/ light bleeding	Yes	Breast cancer in last 5 years, untreated sexually transmitted infection or pelvic inflammatory disease	Age >18 years (used off-label in under 18-year-olds)

How t
bleedi

- History
- Exami
- Think:
  screen

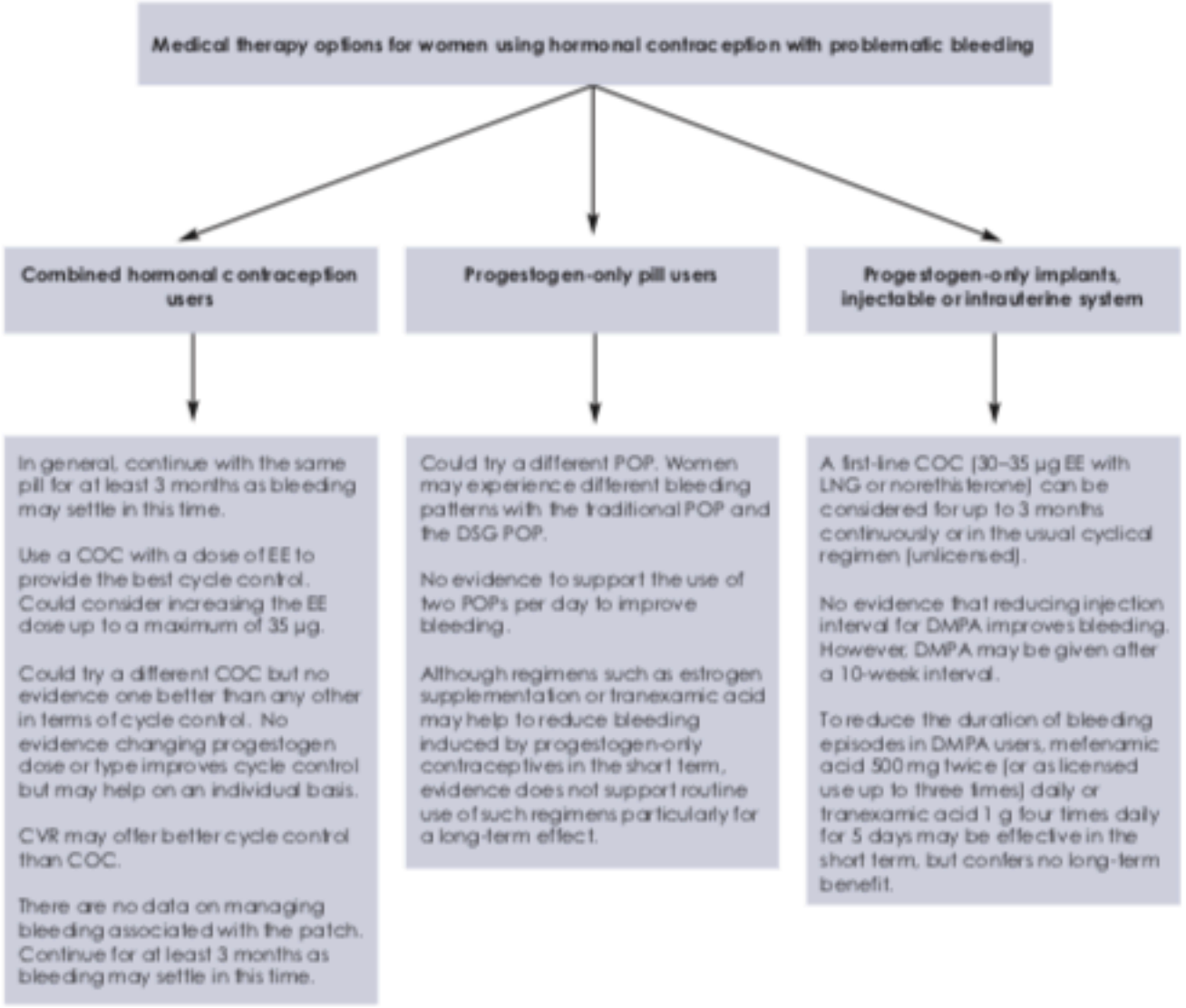

Figure 2 Medical therapy options for women using hormonal contraception with problematic bleeding. COC, combined oral contraceptive pill; CVR, combined vaginal ring; DMPA, depot medroxyprogesterone acetate; DSG, desogestrel; EE, ethinylestradiol; LNG, levonorgestrel; POP, progestogen-only pill.

Special considerations
Epilepsy

- Epilepsy/enzyme inducing drugs
- Young girls notice more seizures during periods
- Enzyme inducing drugs increase metabolism of oestrogen and progesterone
- Reduces contraceptive efficacy – use barrier contraception
- OCP – double dose
- Cannot use implanon
- Otherwise, use LARC e.g. mirena IUS, depot provera Or cyclical progesterone e.g. NET (but not contraceptive)
- If contraception: copper IUD, barrier

Special considerations
Adolescents with learning difficulties

- Periods can be distressing for girl and carer
- Hygiene may be difficult
- Mood changes can mean increased aggression
- Best to aim for amenorrhoea
- Things to consider: route of delivery, poly-pharmacy and interactions, risks of VTE/bone, need for contraception
- Options:
  - Continuous NET or COCP or POP
  - Mirena
  - Depo provera

Special considerations
Haematological disorders

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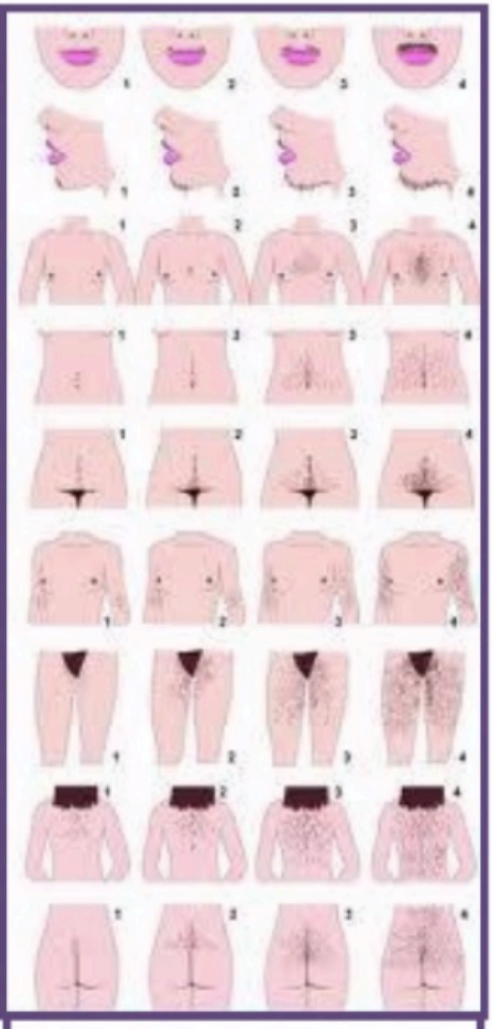
- Bleeding disorders in 1-2% of general population but 20% of those with HMB and 33% who are hospitalised
- Associated with HMB
  - Von willebrands disease
  - Acquired haemophilia
  - Carriers of haemophilia
  - Factor XI deficiency
- Manage as part of MDT
- Hormonal treatments safe
- Aim for amenorrhoea

Hirsutism -

Affects 5-15% XX



Ferriman Gallwey Score –
Clin assessment of body hair growth in women. Clin Endocrinol Metab. 1961;21:1440-1447.

Causes -

1. **PCOS** (70–80% hirsutism cases)
2. **Endocrine:** Hypothyroid , acromegaly, hyperprolactinemia, cushings -cause hyper androgenism
3. **Androgen tumours** ovary (arrhenoblastomas; Leydig, hilar , thecal)/ adrenal (1in 300-1000) hirsutes/50% malignant)
4. **Drugs** – indep androgens, phenytoin, minoxidil, diazoxide, streptomycin,high-dose steroids, psoralen, penicillamine.
5. **Non-classic CAH (AR)** -1.5–2.5% cases, 21-hydroxylase deficient, >17-hydroxyprogesterone (androgenic)
6. **Idiopathic** (hyperandrogenism/hirsutism)6-7%

Management - (9–12 months for maximum effect)

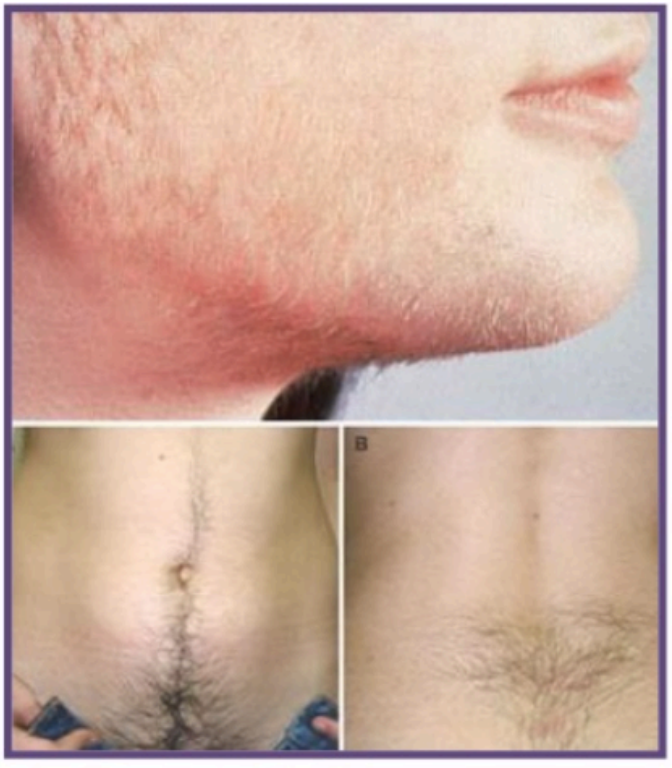
Lanugo- soft hair covers fetus shed by 4 months old
Vellus - soft, >lanugo (2 cm), non-pigmented covers body
Terminal hair-long pigment, eyebrows, scalp, axilla, pubis

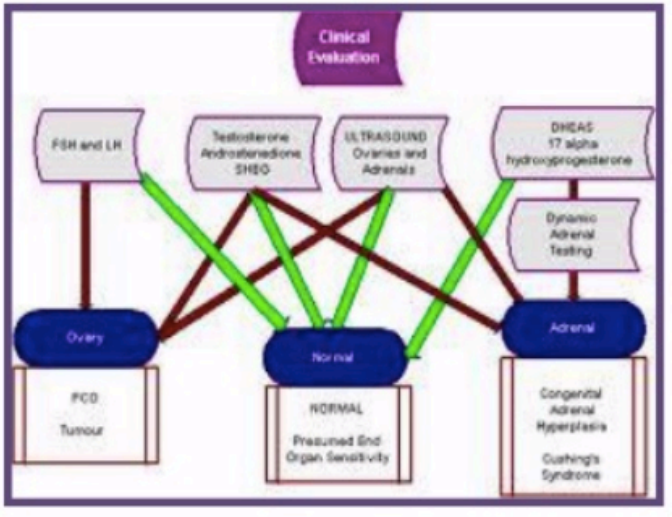
3 phases hair growth-
Anagen- active growing; **Catagen-** involuting, stops growing;
Telogen- resting phase, hair is shed.

Testosterone converted follicle by 5-reductase to potent dihydrotestosterone (DHT). Weaker- androstenedione , DHEA metabolised skin to testosterone , DHT - induce s hair growth.

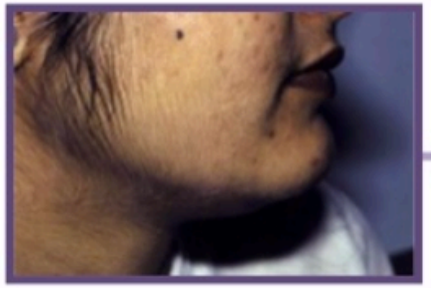
Mild (FG score 8–15)- Treat cosmetic
Moderate–Severe (FG 15) - androgen excess investigate causes Free testosterone level most sensitive . Levels > 1.5–2 ng/ml suggest neoplasm – If DHEA (adrenal) as not produced ovary. Non-classic CAH- testosterone , 17-hydroxy progesterone (17-OHP), elevated early morning. PCOS – (17-OHP)slightly elevated Levels >200 ng/dl suggest CAH. USS look for PCO change.

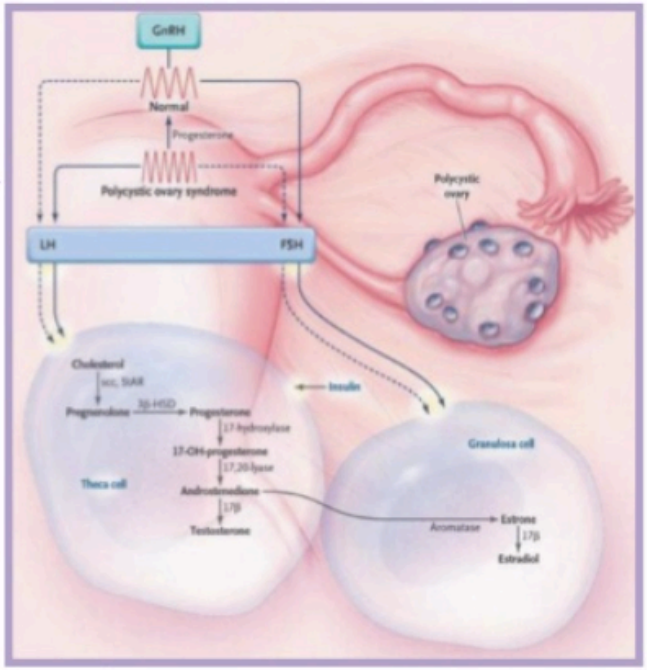
Treat- OCP (Dianette), cyproterone acetate, cosmetic- laser, electrolysis, bleaching, waxing, shaving), topical facial eflornithine (Vaniqa®). **Off license use** -spironolactone, antiandrogens, such as flutamide, finasteride high-dose cyproterone acetate.





PCOS





PCOS- 5-10% XX reproductive age

- **Oligo or amenorrhoea**
- **Infertility and Miscarriage**
- **Acne**
- **Hirsutism, alopecia**
- **Weight gain and obesity**
- **High blood pressure**
- **Elevated insulin/insulin resistance (IR)/NIDDM**
- **PCO > 12 cysts 2-8mm diameter (20% XX PCO versus 5-10% XX PCOS)**

Diagnosis PCOS exclude - thyroid dysfunction, CAH, elevated PL, androgen-secreting lesion, Cushing syndrome.

Diagnosis PCOS based 2/3 criteria Rotterdam Consensus 2004 (ESHRE) -

1. USS – PCO> 12 peripheral follicles (ring of pearls) or ovarian volume >10 cm3
2. Oligo- or anovulation/oligo or amenorrhoea
3. Clinical +/or biochemical signs of Hyper-androgenism

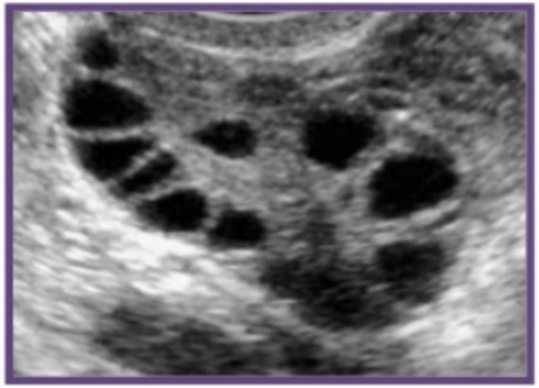
LH/FSH raise not a diagnostic criteria (not consistent)

Baseline screening tests exclude other Diagnoses-

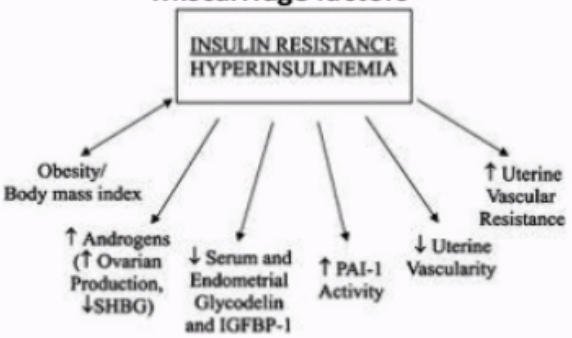
TFT, Prolactin, Free androgen index (total testosterone (T)/SHBG x 100= free T).
If free T >5nmol/l –check 17-Ohprogesterone exclude androgen-secreting TU.
Suspicion Cushings Syndrome investigate according to local protocol.

Complications of PCOS - NIDDM, IHD, CVA.

50% PCOS NIDDM/borderline GTT by 40. Hypertension (> 40 yrs).
LDL:HDL cholesterol increased, IHD risk young age – 40% coronary calcification <45 vs 20% no PCOS , 50% > number of coronary events with PCOS.
Cancer-endometrial hyperplasia & malignancy.



Miscarriage factors



PCOS in Adolescents

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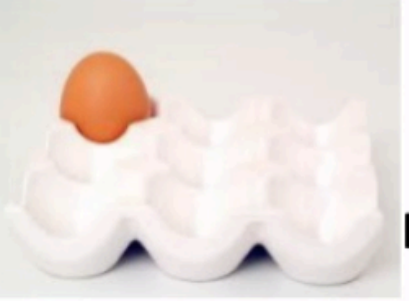
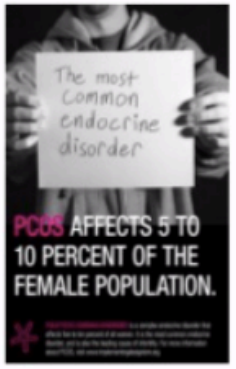
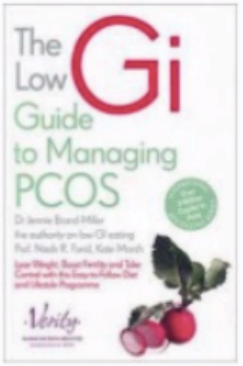
- Based on clinical and biochemical signs of hyperandrogenism and irregular menses
- Diagnosis should be defered until at least 2 years after menarche and once you have excluded other causes
- For girls who not fullfill the diagnostic criteria – focus should be on treatment of symptoms (WITHOUT the label)
- USS should not be used as part of diagnosis – until 8 years after menarche

Witchel et al, Curr Opinino in Paeds, Aug 2019



PCOS DIET SUPPORT





PCOS Management

Website: wellbeingofwomen@rcog.or.uk

Lifestyle: exercise, weight loss, dietary change
Infertility: Irregular and infrequent ovulation. Ovulation induction clomiphene 5 days (D2-7/50-100mg), Superovulation (OHSS risk), IVF, Laparoscopic ovary drill.

Excessive hair growth: Depilatory creams, shaving, waxing, bleaching, plucking, electrolysis, useful/repeated. Anti-androgen effect- OCP or Cyproterone acetate reduce hair growth (use min 9 mths). Laser hair removal best long-term method.

Obesity/IR: Obesity profound effect natural/ART conception rate, also pregnancy complication rate. (>BMI /> IR).
40–50% PCOS overweight: Ovary hyper-androgenism driven by (LH) slim PCOS, >BMI insulin augments effect LH.
IR present in 10–15% slim/20–40% obese PCOS. Screen- BMI/waist circumference. If fasting, BG<5.2 mmol/l risk IGT low. 2-hour/75 g OGTT >risk -BMI> 30 or >25 S.Asia (>IR <BMI).

Metformin inhibits hepatic gluconeogenesis, > insulin sensitivity at cellular level, direct effect ovarian function. Insulin lowering/ insulin sensitising agents – metformin, thiazolidinediones (rosiglitazone, pioglitazone) ? improve symptoms, reproductive outcome. These drugs unlicensed for PCOS, counsel patients.

PCOS best managed with controlled diet, vigorous exercise and weight reduction. Orlistat, sibutramine may help reduce BMI and hyper-androgenism of PCOS. Bariatric surgery may be indicated in select morbid obesity.

Drug therapy

Metformin, troglitazone - beneficial short-term effect IR XXPCOS but not DM. **Metformin** <effect obesity/BMI >35. Doses 500–3000 mg/day common regime 500TDS or 850BD. Long-acting agent <GI effects. Metformin not > lifestyle intervention in improve cardio-metabolic risk/progress T2 DM

Androgen -Metformin may reduce androgen levels by 11% and modest reduce BMI (>37 BMI poor response).
CVD - No robust evidence prevent CVD in PCOS
Cochrane review – Metformin vs COCP no diff in effect on PCOS -hirsutism/acne. 20yrs after lap ovarian drill. Persistent ovulation/norma androgens/SHBG >60% XX (+ if normal BMI)

Health Check

- BP measurement and a fasting blood glucose taken.
- BMI>30 or strong FHx NIDDM OGTT
- All overweight PCOS provide diet and lifestyle advice.
- Amenorrhoeic/severely oligomenorrhoeic PCOS induce regular (3-4mths) withdrawal bleeds. Reduces risk endometrial hyperplasia/CA. Cyclical gestogens (provera/NET at least 12/7), COCP, Mirena® IUS.
- No increase risk Breast CA (routine screen)
- XXPCOS (or partners) ask about snoring/day fatigue /somnolence risk of sleep apnoea, investigation /treatment if necessary.

Ovarian cysts

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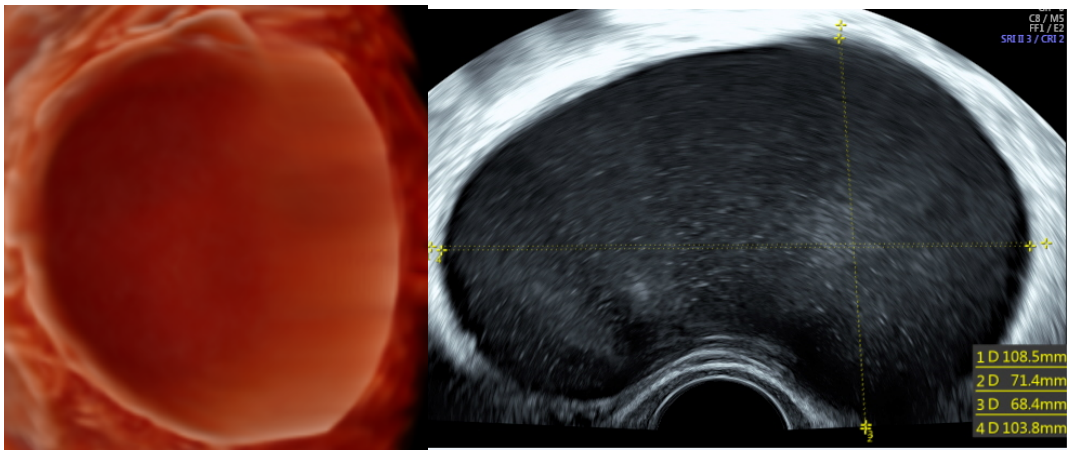
- Very common between menarche and 18 yo
- Mature follicles that fail to ovulate (follicular) or involute (CL)
- Functional vs non functional
- Mostly asymptomatic; incidental
- Can cause menstrual irregularity, pain, urinary frequency, constipation
- Rpt USS in 6-8 weeks – functional tend to resolve
- <5-6cm – observe +/- OCP
- >5-6cm – non functional ; consider laparoscopic cystectomy



Chronic pelvic pain

- 3-6 months of pain
- Can lead to missed school etc
- Investigation into non-gynae causes
- Offer support and empathy, be non-judgemental and be thorough
- Laparoscopy – low yield
- Safeguarding

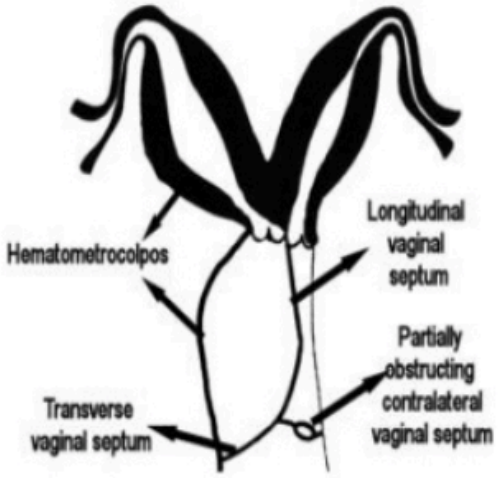
Endometriosis

- Chronic oestrogen dependent disorder of reproductive age
- Pelvic pain, secondary dysmenorrhoea, dyspareunia, infertility
- Less likely in early menarche – primary dysmenorrhoea most likely due to anovulation
- Janssen et al, 2013 – SR published in HRU – 2/3 with CPP or dysmenorrhoea have laparoscopic evidence of endometriosis
  - This does not include those who did not have surgery
  - Includes those resistant to medical therapy – more severe end of spectrum
  - Surgery NOT the mainstay or long-term treatment
- Diagnosis : history, examination, USS, Laparoscopy
- Treatment: hormonal , lifestyle

Classification - Mullerian/Uterine Malformations

Incidence: 1 in 200-1 in 600

American Fertility Society classification 1988 - Müllerian duct anomalies or DES, ∗=uterus may be normal or take variety abnormal forms, ∗∗=may be two cervices. AFS classification system framework for description anomalies, communication among physicians, comparison of therapeutic modalities, often confusion about reporting of certain anomalies, particularly those with features >one class. MRI gold standard-accuracy, detailed outline uterovaginal anatomy. Laparoscopy and hysteroscopy reserved for those where interventional therapy may be undertaken.



Incidence Type 111 -Didelphus – 0.1-0.5%

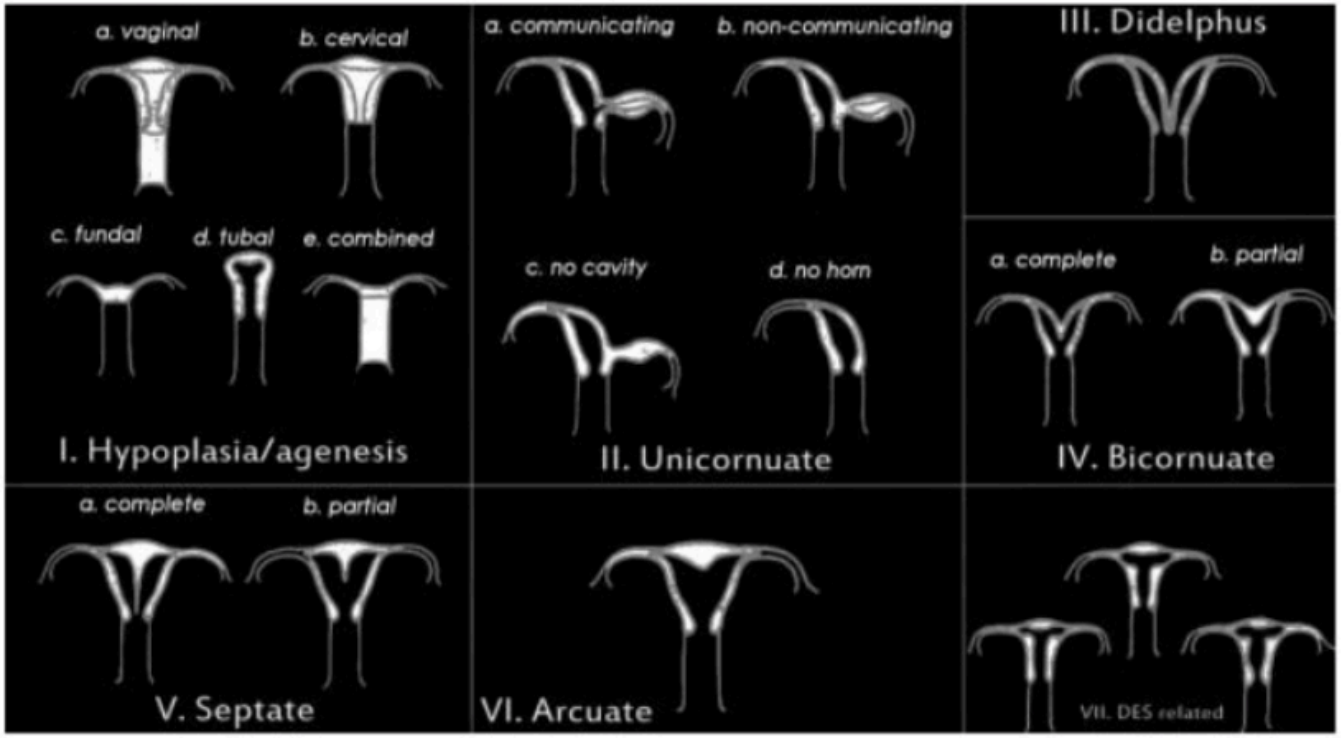


Diagram vaginal anomalies –

Longitudinal vaginal septum is the result of failure of lateral fusion of the Mullerian ducts.

Transverse vaginal septum is the result of failure in vertical fusion between the Mullerian duct and the urogenital sinus and is usually ipsilateral to the side of renal agenesis.

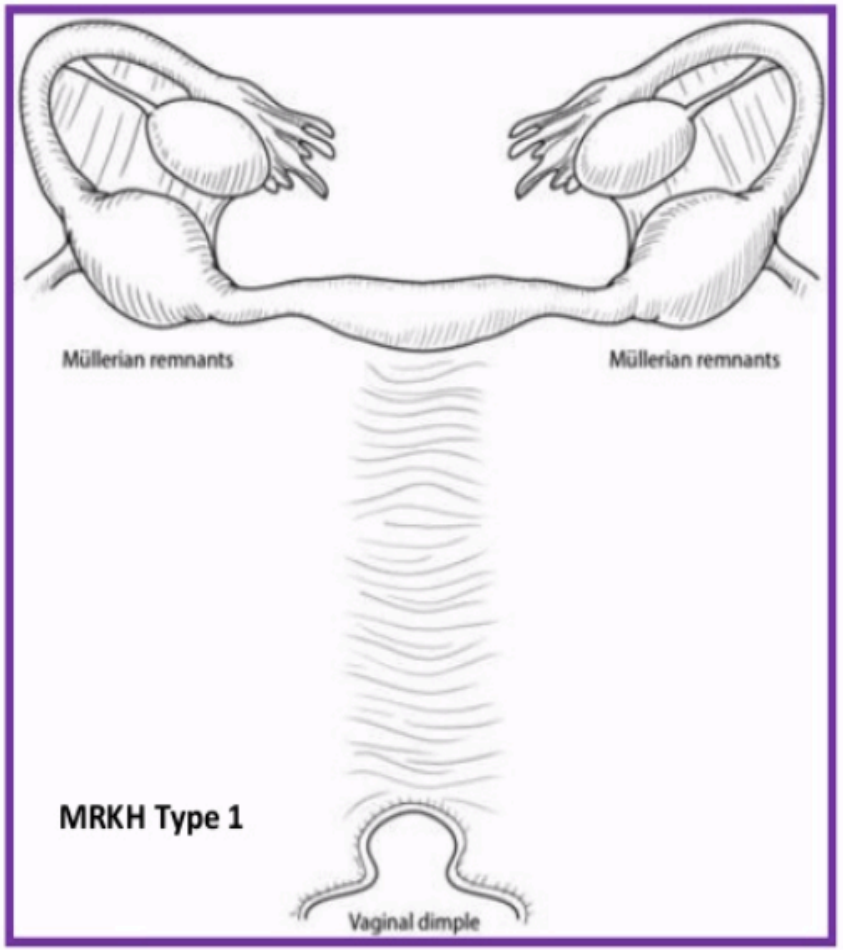
MRKH Syndrome (1 in 4500)

Table 1. American Fertility Society classification of congenital uterine abnormalities (1988)

Class I: Hypoplasia/uterine agenesis
Class II: Unicornuate uterus
Class III: Uterus didelphys
Class IV: Bicornuate uterus
Class V: Septate uterus
Class VI: Arcuate uterus
Class VII: T-shaped uterus results from DES

Classification – MRKH 3 Types:

1 Typical (64%): Isolated symmetrical UV aplasia/hypoplasia
2 Atypical (24%): Asymmetrical UV aplasia/hypoplasia, absence or hypoplasia 1 or 2 F tubes. Malform. ovaries +/- renal system
3 MURCS (12%): (mullerian duct aplasia, renal dysplasia, cervical somite anomalies) syndrome: UV aplasia/hypoplasia, skeletal, renal, +/- heart malformation.
Associated renal abnormalities (40%): renal agenesis, ectopic or horseshoe kidney, ectopic ureter
Genetics: sporadic-polygenic/multifactorial inheritance or familial clustering- autosomal dominant, with variable penetrance, expressivity



Mayer–Rokitansky–Küster–Hauser syndrome: diagnosis and management.
S Valappil, U Chetan, N Wood, A Garden.
TOG 2012;14(2):93-98

MRKH
Sexual function

Mayer-Rokitansky-Küster-Hauser syndrome: a review of 245 consecutive cases managed by a multidisciplinary approach with vaginal dilators

D. Keith Edmonds, F.R.C.O.G., Gillian L. Rose, F.R.C.O.G., Michelle G. Lipton, D.Clin.Psy., and Julie Quek, R.N.

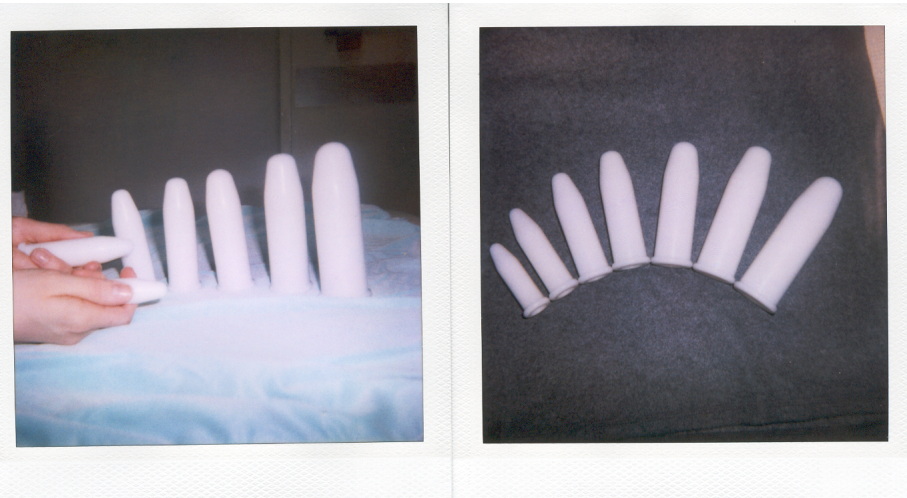
Queen Charlotte's and Chelsea Hospital, Imperial College Healthcare NHS Trust, London, United Kingdom

Objective: To understand the efficacy of vaginal dilators in the management of Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome.
Design: Retrospective sequential study.
Setting: Hospital.
Patient(s): 245 women.
Intervention(s): Vaginal dilators.
Main Outcome Measure(s): Functional vaginal length and sexual satisfaction.
Result(s): Of the patients who completed the program, 232 (94.9%) achieved a successful vaginal length (defined as greater than 6 cm in length and maximum width throughout the vagina and especially at the apex) and sexual function. When the program was completed by all patients, 100% of patients were successful.
Conclusion(s): Vaginal dilator therapy is the treatment of first choice for creation of the vagina in MRKH syndrome, and the success rates suggest that surgery is rarely, if ever, required. (Fertil Steril® 2012;97:686–90. ©2012 by American Society for Reproductive Medicine.)
Key Words: MRKH syndrome, neovagina, vaginal dilators

MRKH
Sexual satisfaction

- There was **NO** difference between women with MRKH and other women for sexual desire, arousal, and satisfaction with sexual relationship

- Difference in vaginal lubrication – use lubrication



J Pediatr Adolesc Gynecol. 2005 Feb;18(1):39-42. doi: 10.1016/j.jpag.2004.11.008.

Sexual function in women treated with dilators for vaginal agenesis

S Nadarajah [1], J Quek, G L Rose, D K Edmonds

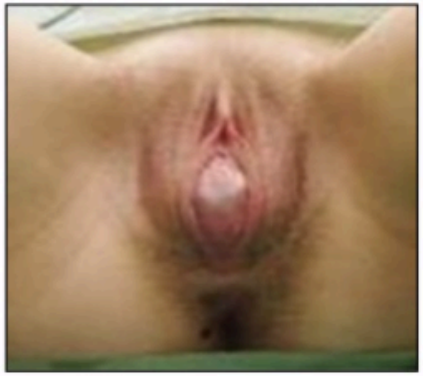
Types vaginal septum

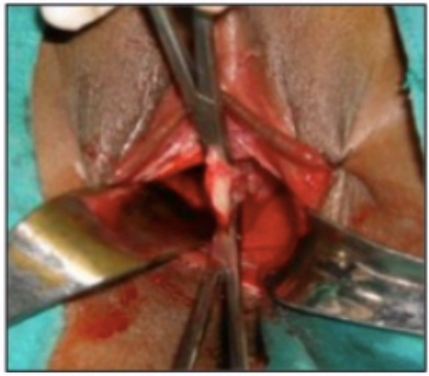
Longitudinal septums -
"double vagina" complete or incomplete, often with uterus didelphys, many combinations encountered, including normal uterus /cervix. Sometimes one cervix may be blocked (retention mucus/ blood). EG usually normal, septums often not diagnosed unless painful sex or labor dystocia . Simple incision, ligation bleeding points all that is required. Fistula formation or vaginal stenosis may result with incautious or extensive surgery. Longitudinal septums occur x2 frequently as transverse septums.

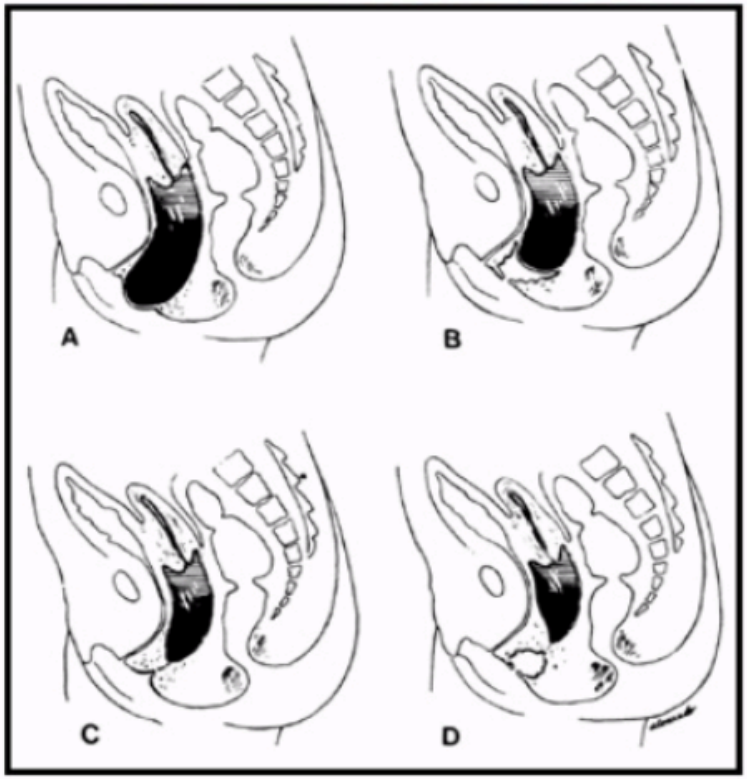
Transverse septums –
varies occlusion to mild constriction. Frequently, small opening allows secretions/ blood to drain. Unlike bulging membrane associated with imperforate hymen, no external sign of blockage. In nearly all internal organs are normal and pregnancy not infrequent. Transverse septums represent failure of complete canalization of the vaginal epithelial mass. Stenosis may be caused by a constricting fibromuscular band. Menstruation and coitus occur without trouble, condition may not be detected until pelvic examination reveals its presence.

Symptoms -
Depends on the presence of adequate uterine drainage. Complete vaginal atresia, lower abdomen mass (hematometrocolpos), pelvic abscesses, may cause dystocia and cesarean section the safest method of delivery.

Treatment-
Depends on degree stenosis and rigidity of constricting band. No treatment or 2/3 longitudinal incisions may suffice. Complete excision of annular segment of vaginal wall may result in scarring or fistula .







Websites
Vaginal Hypoplasia: http://www.medhelp.org/www/ais/31_HPLASIA.HTM
A Guide for Teens: http://www.youngwomenshealth.org/vaginalagenesis.html
Vaginal Agenesis: http://www.urologyhealth.org/adult/index.cfm?cat=01&topic=150
Vaginal Atresia: http://www.emedicine.com/ped/topic2999.htm
Rokitansky-Mayer-Küster-Hauser Syndrome MRKH Foundation: http://mrkh.org/

Lesions causing hydrometrocolpos-
A) Imperforate hymen B)Transverse septum C) and D) Low and high vaginal atresia. Spencer R, Levy DM: Hydrometrocolpos: Report of three cases review of the literature. Ann Surg 155: 558, 1962.)

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DRD TEAM

Summary

- Most menstrual dysfunction in adolescence is associated with anovulatory cycles
- Baseline history, examination and investigations
- Reassurance is key
- Non hormonal vs hormonal treatments
- If concerns, please refer or contact us for help

Thankyou

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Menstrual disorders in adolescence

Early Pregnancy and Adolescent Gynaecology GP Study Afternoon

Thursday 13th May 2021

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