PAEDIATRIC AND ADOLESCENT GYNAECOLOGY

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Definitions

- **Paediatrics:** The study and treatment of children in health and disease during development from birth through adolescence.

- **Adolescence:** WHO has defined it as progression from appearance of secondary sexual characteristics to sexual and reproductive maturity and development of adult mental process.
  WHO: 10-19 years
  RCH-II: 10-19 years
  Govt. of India (National Youth Policy): 13-19 years

Normal development

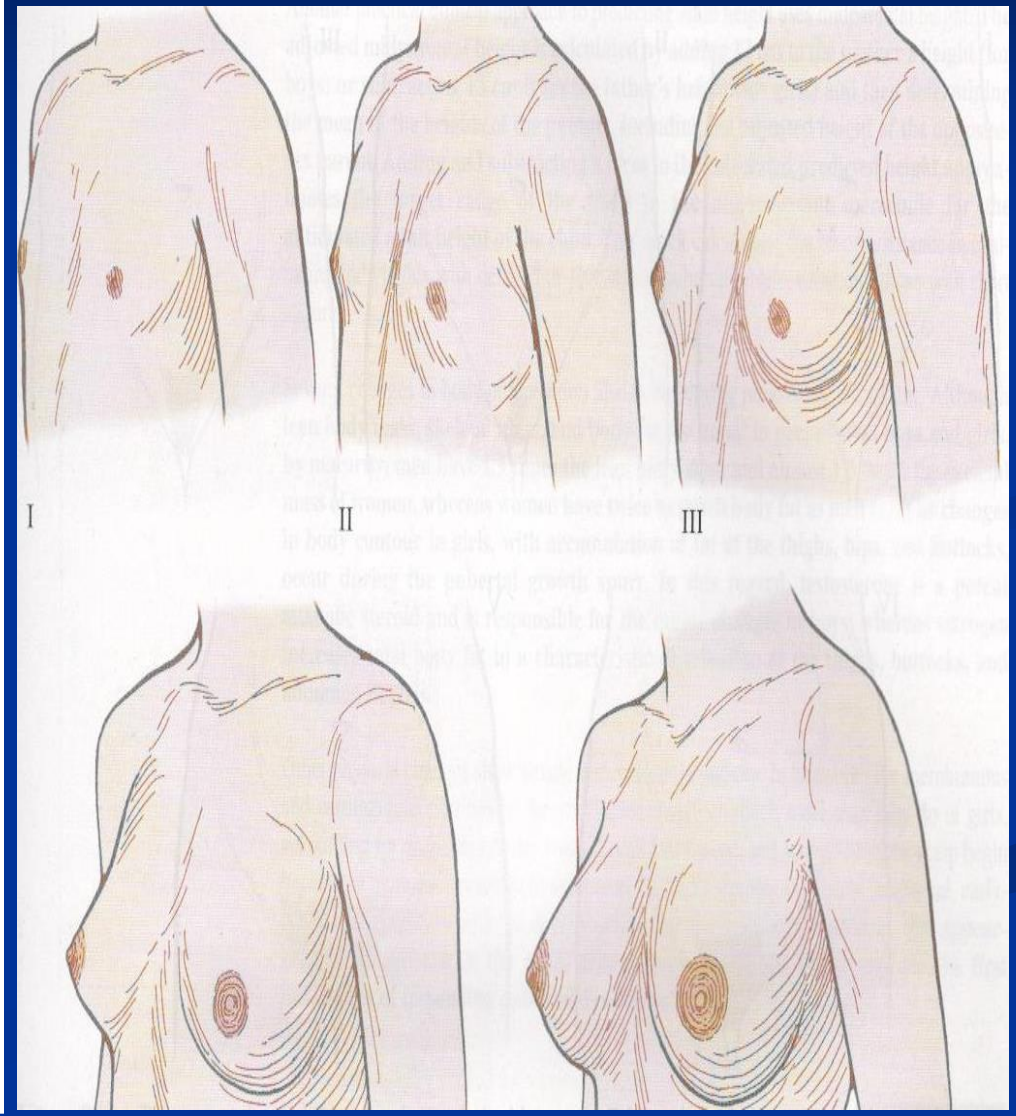
	NEWBORNS (first 28 days) (maternal estrogen)	YOUNG CHILDREN (upto 7 yrs) (↓ estrogen)	LATE CHILDHOOD (7-10 yrs) (↑ estrogen)	YOUNG ADOLESCENTS (10-13 yrs) (puberty)
EXTERNAL GENITALIA	▪Labia majora bulbous ▪Labia minora thick protruding ▪Clitoris relatively large	▪Labia majora flattened ▪Labia minora and hymen thin ▪Clitoris relatively small	▪Mons pubis thickened ▪Labia majora fill out ▪Labia minora rounded ▪Hymen thickened	▪Adult appearance ▪Bartholin's glands begin to secrete mucus

VAGINA	■4 cm long at birth ■Vaginal discharge - mucus+ exfoliated cells (sterile) ■pH – 4-5	■Atrophic mucosa with few rugae ■Doderlien's bacilli absent ■Susceptible to infection ■pH > 7 ■Cervix flush with vault	■Length 8 cm ■Mucosa thickened ■Cervix flush with vault ■pH > 7	■Length 10 -12 cm ■More distensible ■Lactobacilli reappear ■Development of fornices ■pH – alkaline to acidic
UTERUS	■Length 4 cm ■Cervix: corpus – 3:1 ■Columnar epithelium protrudes through external os	■Regresses in size ■Regains size by 6 years	■Uterine growth begins ■Myometrial proliferation ■Pre menarchal rapid endometrial proliferation ■Cervix:corpus- 1:1	■Cervix:corpus – 1:2

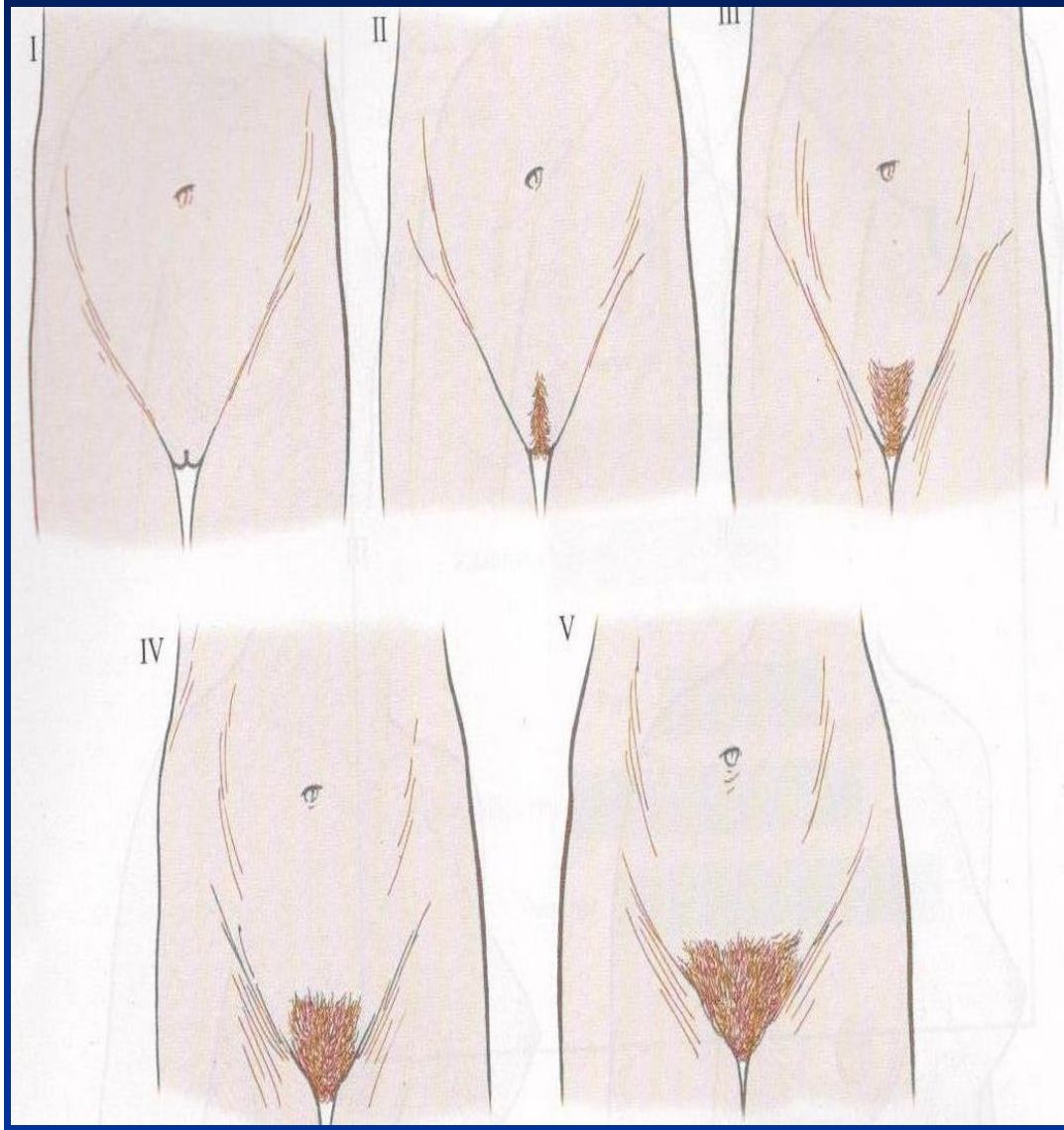
OVARIES	Abdominal organs	▪Begin to enlarge and descend into true pelvis ▪Number and size of ovarian follicles increase	▪Further descent ▪Number of follicles increases ▪Ovulation generally does not occur	▪Ovaries descend into true pelvis ▪Secondary sexual characteristics appear

Tanner staging of development of secondary sexual characteristics

STAGE	BREAST
1.	Elevation of papilla
2.	Elevation of breast and papilla as a small mound, increased areolar diameter (median age 9.8 years)
3.	Further enlargement without separation of breast and areola (median age 11.2 yrs)
4.	Secondary mound of areola and papilla above breast (median age 12.1 years)
5.	Recession of areola to contour of breast (median age 14.6 years)



	PUBIC HAIR
1	No pubic hair (prepubertal)
2	Sparse, long pigmented hair mainly along labia majora (median age 10.5 years)
3	Dark, coarse, curled hair sparsely spread over mons (median age 11.4 years)
4	Adult type, abundant hair, but limited to the mons (median age 12 years)
5	Adult type spread in quantity and distribution (median age 13.6 years)



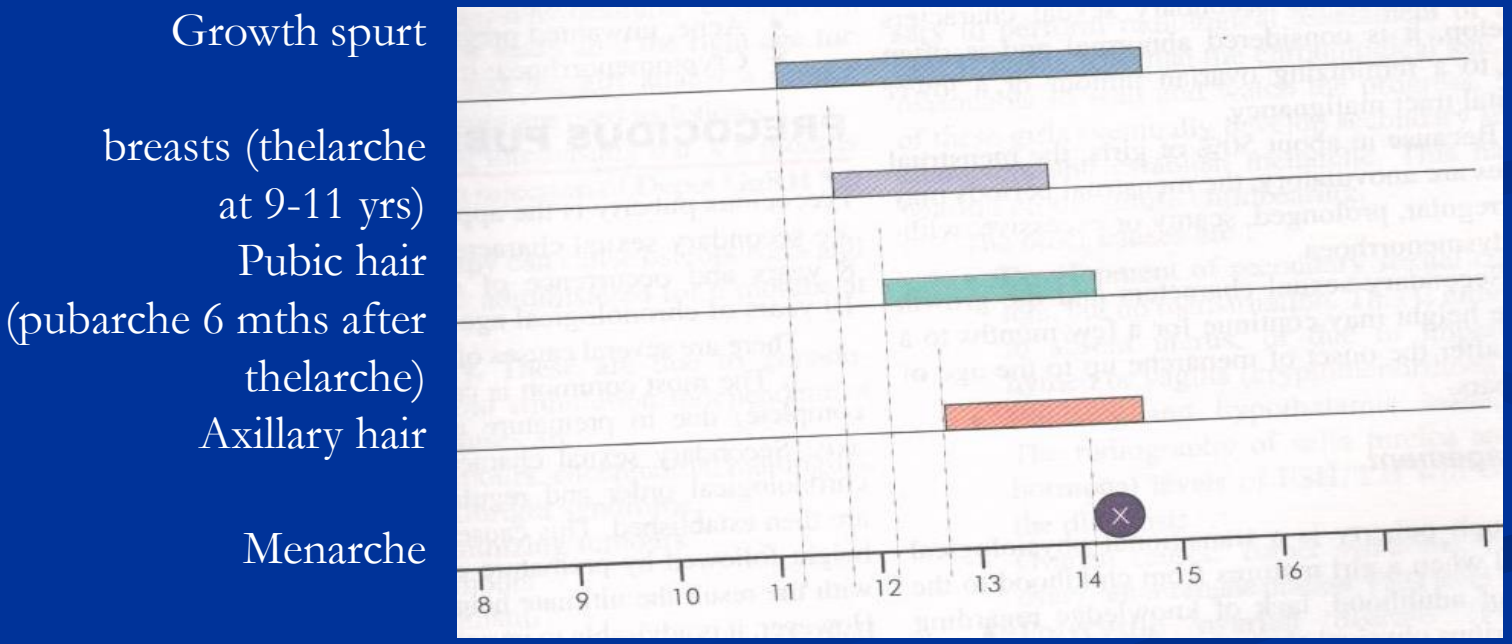
Puberty

- It is the state of becoming functionally capable of procreation
- First menstrual period usually occurs between ages of 10 and 16 years

  average 12.5 years in India

  13 years in Europe

  12.5 years in North America



Development of secondary sexual characters related to age

Summary of pubertal events

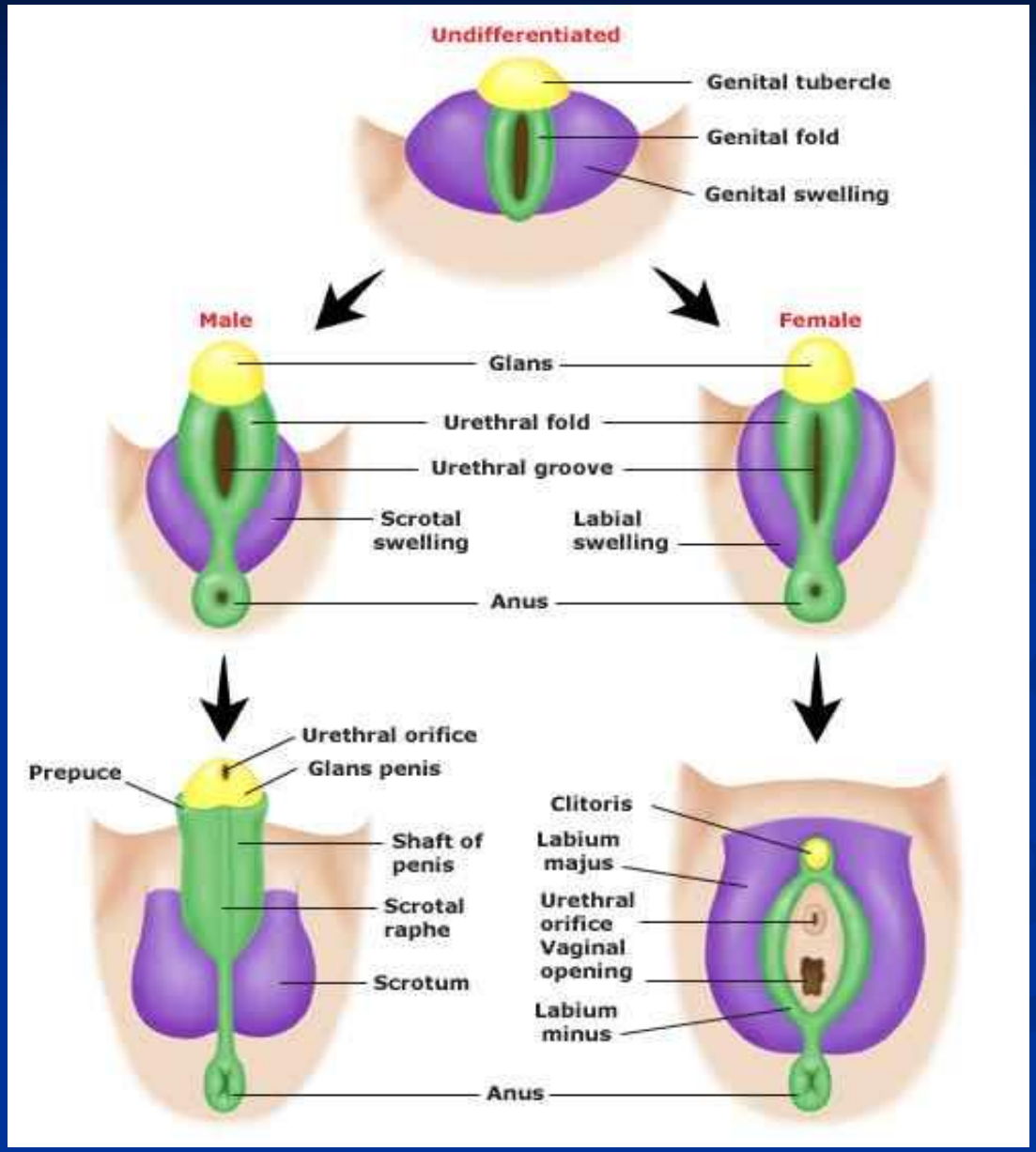
1. FSH and then LH levels rise moderately before the age of 10 yrs and are followed by rise in estradiol.
   Increasing LH pulses first seen only in sleep but gradually extends throughout the day.
   In adults it occurs at 1.5 to 2 hourly intervals
2. As gonadal estrogen increases (gonadarche)
   breast development (thelarche at 9 -12 yrs), female fat distribution, vaginal and uterine growth occur.
   Skeletal growth rapidly increases as a result of initial gonadal secretion of low levels of estrogen, which stimulates IGF-1 production
3. Adrenal androgen (adrenarche) and to a lesser degree gonadal androgens cause pubic and later axillary growth (functionally unrelated to gonadarche)
4. At midpuberty, sufficient gonadal secretion proliferates the endometrium – menarche occurs
5. Postmenarchal cycles are initially anovulatory. Sustained, predictable positive LH surge responses to estradiol with ovulation are late pubertal events

Common clinical problems in different age groups

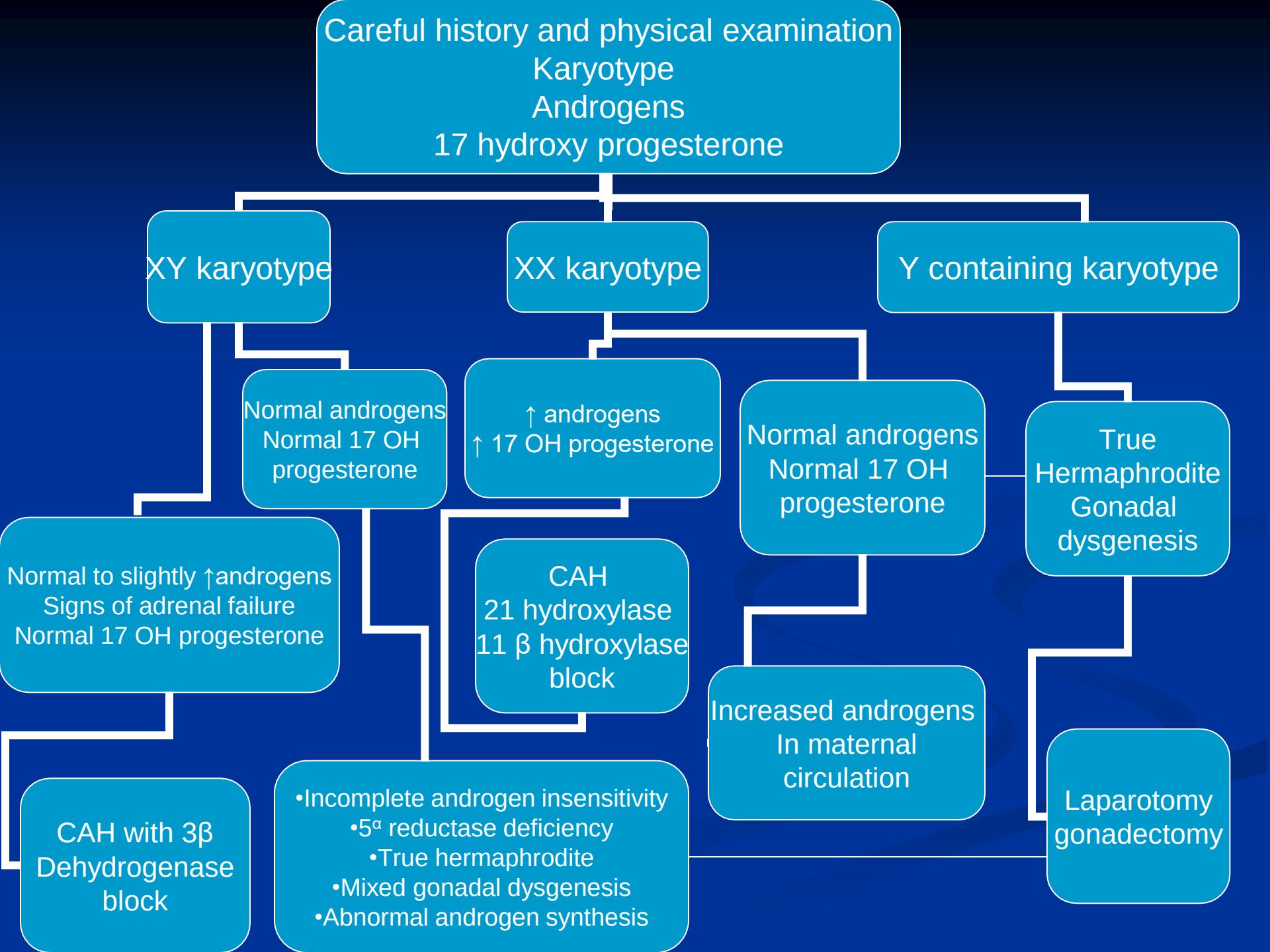
<5 years (neonates, toddlers and infants)	▪Ambiguous genitalia ▪Genital crisis ▪Labial adhesions ▪Imperforate hymen ▪Ectopic anus ▪Nipple discharge
6-11 years (premenarchal)	▪Vulvovaginitis in childhood ▪Abnormal vaginal discharge ▪Vaginal bleeding ▪ precocious puberty ▪neoplasms
12-18 years (perimenarchal to adolescence)	▪Menstrual abnormalities ▪Delayed puberty ▪Neoplasms ▪Leucorrhea ▪Congenital anomalies ▪miscellaneous

AMBIGUOUS GENITALIA

Normal development of external genitalia



Androgen exposure at 9-14 weeks superimposes variable external ambiguity on basic female phenotype (clitoral hypertrophy, hypospadias, scrotalisation of non fused labia)


Careful history and physical examination
Karyotype
Androgens
17 hydroxy progesterone
XY karyotype
XX karyotype
Y containing karyotype
Normal androgens
Normal 17 OH progesterone
↑ androgens
↑ 17 OH progesterone
Normal androgens
Normal 17 OH progesterone
True Hermaphrodite
Gonadal dysgenesis
Normal to slightly ↑androgens
Signs of adrenal failure
Normal 17 OH progesterone
CAH
21 hydroxylase
11 β hydroxylase
block
Increased androgens
In maternal
circulation
CAH with 3β
Dehydrogenase
block
•Incomplete androgen insensitivity
•5α reductase deficiency
•True hermaphrodite
•Mixed gonadal dysgenesis
•Abnormal androgen synthesis
Laparotomy
gonadectomy

Treatment

Surgery:

feminizing genital surgery-

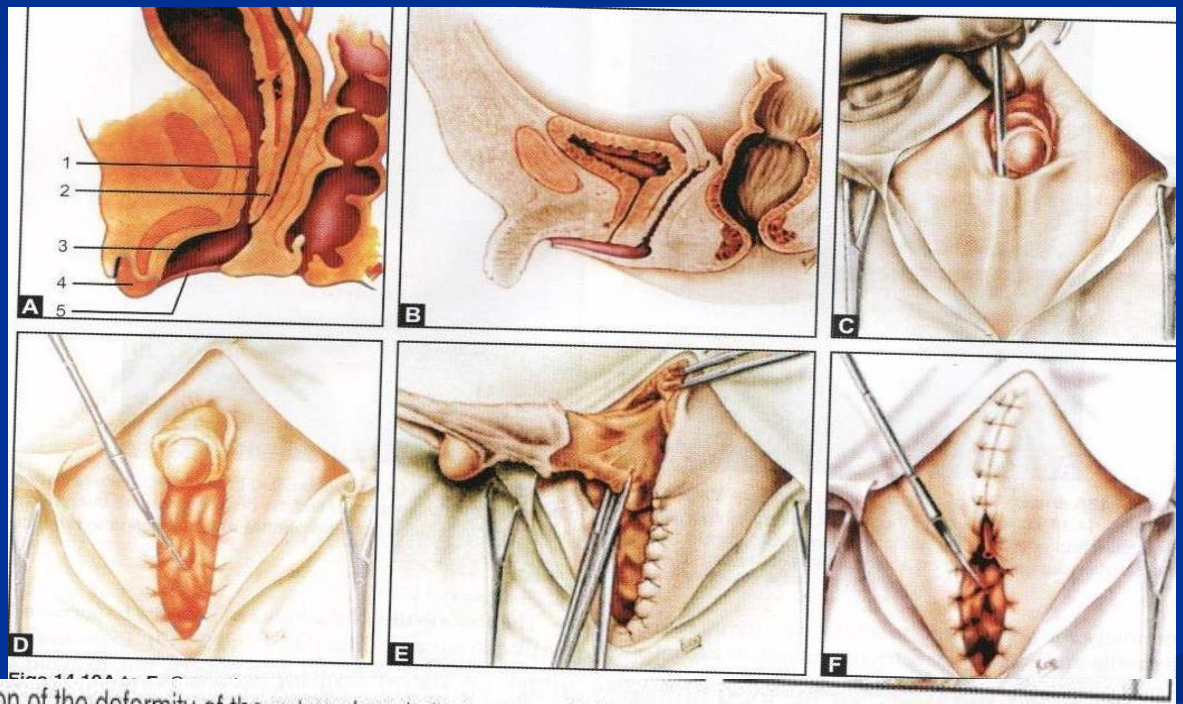
clitoral surgery for clitoral hypertrophy

urogenital sinus mobilization

labioplasty

neovaginal construction

possible gonadectomy



Correction of the deformity of the external genitalia in congenital adrenal hyperplasia. (A) The normal development of the vulva, vagina and urethra at the fourth month (after Hamilton, Boyd and Mossman). (1) urethra; (2) vagina; (3) vestibule forming from the urogenital sinus; (4) clitoris; (5) site of cloacal (urogenital) membrane, (B) The deformity in the adrenogenital syndrome represents essentially a fusion of the genital folds to form a perineal membrane which obscures the introitus and vagina, (C) The occluding membrane demonstrated (D) Incision of the membrane reveals the urethra and vagina, (E) Removal of the enlarged clitoris, (F) The ultimate result;

- **Gonadectomy:** done because of
  - risk of malignancy
  - risk of ongoing virilization with continued androgen exposure
- **Medical:**
  - maintenance dose of adrenal steroids in congenital adrenal hyperplasia
  - sex steroids in hypogonadal patients (post gonadectomy, Turner's, premature ovarian failure)

GENITAL CRISIS

Seen within few days of birth

Effect of passive stimulation from maternal estrogen

1. **Bleeding PV** : within 10 days of birth, blood stained discharge or frank bleeding. Decline in levels of estrogen leading to withdrawal bleeding.

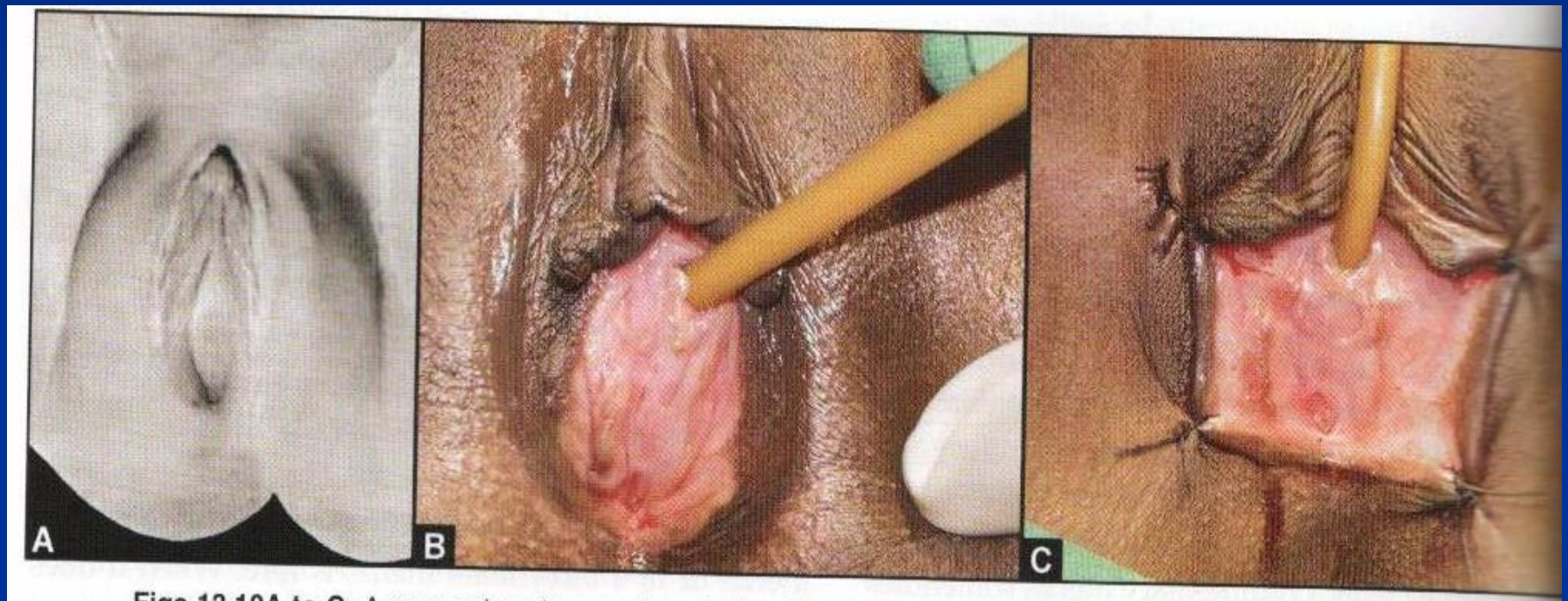
2. **Enlarged breasts:** Maternal estrogen and progesterone lead to development of duct and alveolar system of breasts. Withdrawal of hormonal suppression of prolactin leads to discharge from breasts (Witch'smilk)

3. **Neonatal leucorrhea:** maternal estrogen leads to hypertrophied cervical glands

Treatment: reassurance

MUCOCOLPOS

Imperforate hymen may present in neonates in case of excessive secretion from cervix under influence of maternal estrogen



Figs 13.10A to C: A mucocolpos in a newborn baby with the imperforate lower vaginal membrane bulging. This was incised when the baby was 3 days and 500 ml of mucoid fluid escaped. (By permission of Mr H.H. Francis)

Labial adhesions

Cause: absence of adhesions

↓

Lack of local defense

↓

Mild infection of vulva

↓

Denudation of surface epithelium of labia minora

↓

Adhesions

Maternal androgen intake is a rare cause

Appears within 2-3 years of birth

Management:

- Asymptomatic: observation
- Symptomatic (difficulty in micturition, periodic UTI, rarely incontinence, inflammation of vestibule and vagina)
  retention: surgical separation with fingers or probe
  raw area treated with topical estrogen or antibiotic ointment to prevent re-agglutination
  no retention: topical estrogen twice a day for 7-10 d
- Tendency of recurrence:
  await spontaneous cure at puberty.
  Improve perineal hygiene and removal of irritants

VULVOVAGINITIS

- Premanarchal age group is susceptible because

1. Decreased circulatory estrogen

↓

Lack of stratification of vaginal epithelium

↓

Lack of glycogen and absence of lactobacilli

↓

No acid formation

↓

Raised vaginal pH

2. Inadequate perineal hygiene
3. Lack of protective pubic hair and fatty pads of labia majora

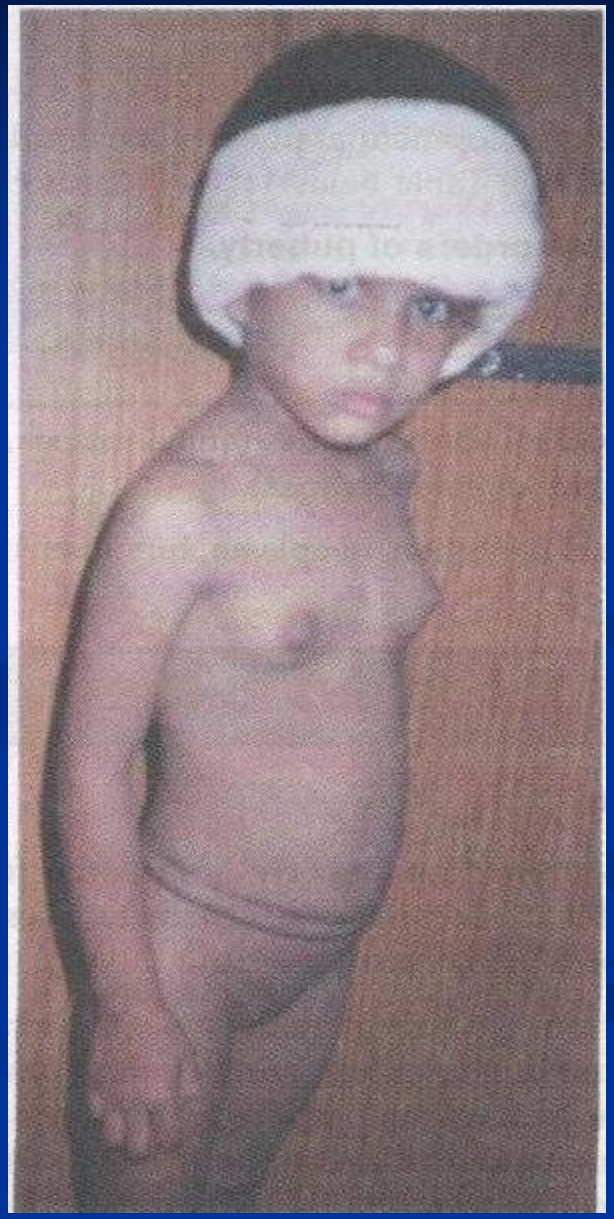
- Most common cause is polymicrobial
- Symptoms: vaginal discharge (purulent or blood stained)
  pruritus or sorenes in external genitalia
  painful urination
- Signs: vulva becomes edematous, red even ulcerated
  vaginal epithelium is congested with pent up discharge
- Sequelae: vulval inflammation
  adhesions
  neglected STD → future infertility, dyspareunia, pelvic congestion syndrome
- Treatment: perineal hygiene
  medical: Amoxycillin 20-40 mg/kg/d in 3 div doses
  refractory: local estrogen twice a day for 3 weeks
  specific treatment
  consider sexual abuse

LEUCORRHEA

- As puberty approaches, there is excessive production of mucus from cervical glands and increased transudation from the vaginal epithelium (increased endogenous estrogen)
- Non offensive and non irritant vaginal discharge
- No treatment required

PRECOCIOUS PUBERTY

- Onset of menstruation, accompanied by other evidences of puberty eg. breasts, pubic hair before age of 8 years (menarche before age 10) taking mean ± 2 SD as encompassing normal range
- Thelarche and adrenarche before age 6 warrants evaluation



Girl aged 4 years showing precocious puberty

- Classification:

1. Complete isosexual precocity (true; gonadotropin dependant)
    a. idiopathic
    b. CNS lesions: hamartomas, craniopharyngiomas etc.
    c. primary hypothyroidism
    d. post treatment for CAH
    e. abnormal skull development (rickets)
    f. injury, encephalitis, meningitis
2. Incomplete isosexual precocity (gonadotropin independent)
    a. isolated precocious thelarche
    b. isolated precocious menarche
    c. estrogen secreting tumors of ovary or adrenals
    d. ovarian cysts
    e. McCune Albright syndrome
    f. Peutz-Jeghers syndrome
    g. iatrogenic
3. Isolated virilization
    a. isolated precocious adrenarche
    b. congenital adrenal hyperplasia
    c. androgen secreting ovarian or adrenal neoplasms
    d. iatrogenic

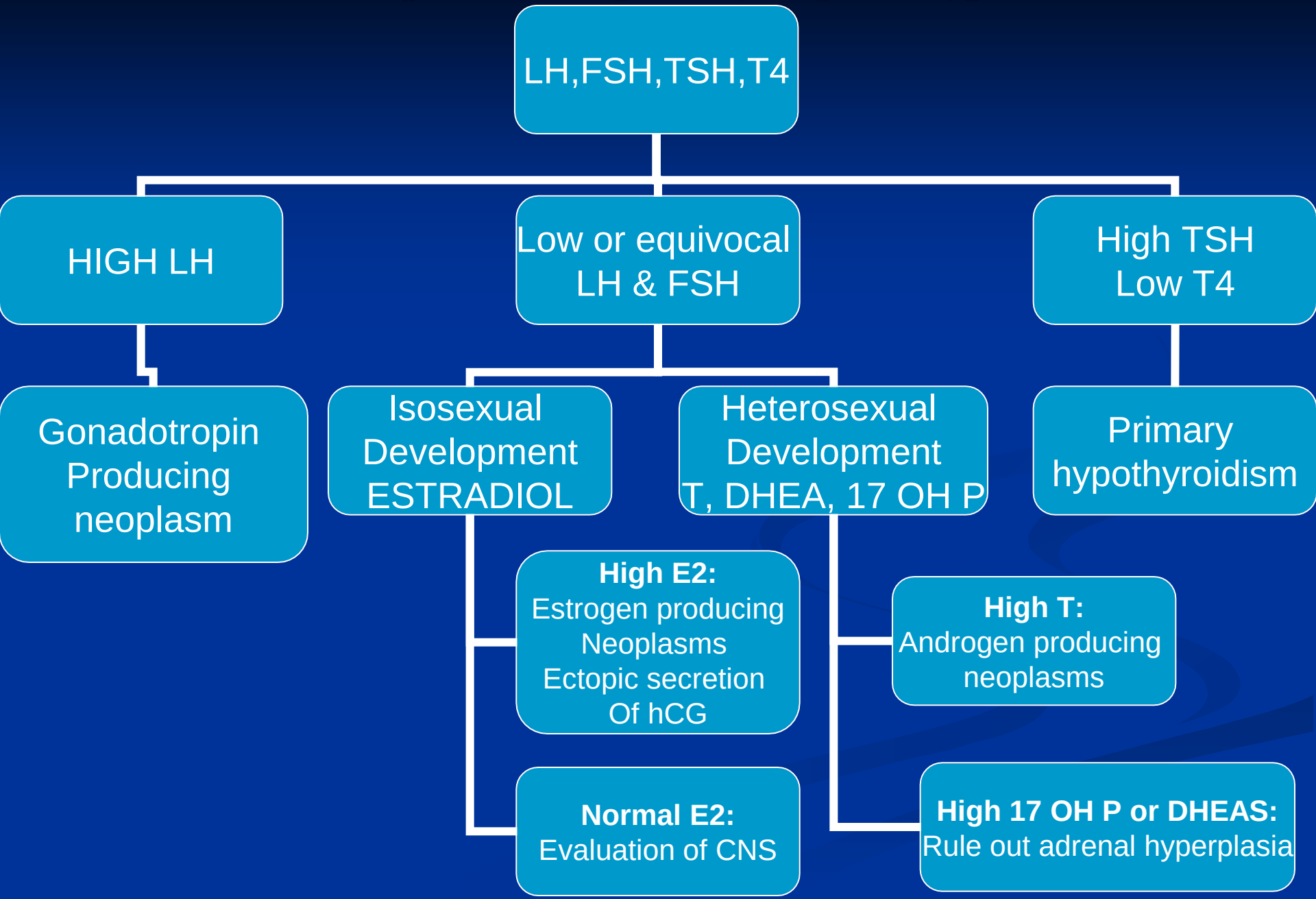
Gonadotropin dependent

- Constitutional: premature maturation of HPO axis leads to production of gonadotropins and sex steroids.
  Runs in families
  usually occurs close to borderline age of 8 years
- Idiopathic: does not run in families
  occurs much earlier in childhood
- Diagnosis of these made only by exclusion
- Clinical progression is variable. Usually slower in idiopathic cases
- Most serious effect : adult short stature (early epiphyseal fusion)
- Intellectual and psychosocial development commensurate with chronological age rather than age of puberty
- Ectopic gonadotropin production (<0.5%): chorioepithelioma and dysgerminoma of ovary, liver hepatoblastoma

Gonadotropin independent

- Common cause: congenital adrenal hyperplasia
- 11%- ovarian tumour. Bleeding is irregular and menorrhagic because of anovulatory cycles
- Drugs- OCP, anabolic steroids, hair or facial creams. Associated with dark pigmentation of nipples and breast areolae.
- McCune Albright syndrome 5%: multisystem disorder
- Benign or follicular or luteal cyst: absence of gonadotropin pulsations; variable response to GnRH; lack of suppression of puberty by long acting GnRH agonist

Evaluation of precocious puberty in phenotypic females



- FSH > 7.5 IU/L or LH > 15IU/L → pituitary secretion suspected
- Normal examination and MRI → idiopathic
- Rule out life threatening diseases like neoplasms
- Exclude nonendocrine causes like trauma, foreign body, vaginitis, neoplasm

Treatment:

- Objectives: diagnose and treat intracranial disease
  arrest maturation until normal maturation age
  attenuate and diminish established precocious characters
  maximize eventual adult height
  facilitate avoidance of abuse
  decrease emotional problems

- Drugs: GnRH analogues (less side effects, drug of choice in GnRH dependent)
  Buserelin nasal spray 100µd daily or depot once a month goserelin or leuprolide
  Medroxy progesterone acetate(30 mg daily or 100-200 mg im weekly)
  Cyproterone acetate(70-100 mg/m^2/d oral for 10 days from 5 day of cycle)
  Danazol

Dose monitored by E2 levels <10 pg/ml

- Treatment maintained until epiphses are fused or until appropriate pubertal and chronological ages are matched. There is prompt reactivation of ovulatory function

Specific:

- GnRH secreting hamartomas of hypothalamus- suppression of gonadal steroidogenesis (medroxyprogesterone acetate or aromatase inhibitor)
- CNS tumors: neurosurgical treatment or radiation
- Ovarian or adrenal tumors: surgical excision
- Ovarian cyst: multiple and bilateral – usually secondary to gonadotropins
  solitary and contralateral ovary immature - resection
- Primary hypothyroidism – thyroid replacement
- CAH – glucocorticoids
- Management of psychosocial problems

DELAYED PUBERTY

- Puberty is said to be delayed when secondary sexual characteristics (breast tissue and/or pubic hair) have not appeared by 13 years and when menstruation does not begin before 15 years age

- **Causes:**

1. Anatomic abnormalities of the genital outflow tract (Eugonadism)
   a. Mullerian dysgenesis (Meyer-Rokitansky-Kuster-Hauser syndrome)
   b. Distal genital tract obstruction
      -imperforate hymen
      -transverse vaginal septum

2. Hypergonadotropic (FSH>30mIU/ml) hypogonadism (gonadal 'failure') low estrogen <25 pg/ml
   a. gonadal dysgenesis with stigmata of Turner's syndrome
   b. pure gonadal dysgenesis
   -46XX - 46XY
   c. early gonadal 'failure' with apparently normal ovarian development
3. Hypogonadotropic (LH & FSH <10 mIU/ml) hypogonadism
   a. constitutional delay
   b. isolated gonadotropin deficiency
   - associated with midline defects (Kallman's)
   - independent of associated disorders
   - Prader-Labhardt-Willi syndrome
   - Laurence-Moon-Bardet-Biedl syndrome
   - other rare causes

c. Associated with multiple hormone deficiencies

d. Neoplasms of hypothalamo-pituitary area
- craniopharyngioma
- pituitary adenoma
- others

e. Infiltrative processes (Langerhans cell histiocytosis)

f. Irradiation of CNS

g. Severe chronic illnesses and malnutrition

h. Anorexia nervosa and related disorders

i. Severe hypothalamic amenorrhea

j. Anti dopaminergic and GnRH inhibiting drugs (psychotropic agents, opiates)

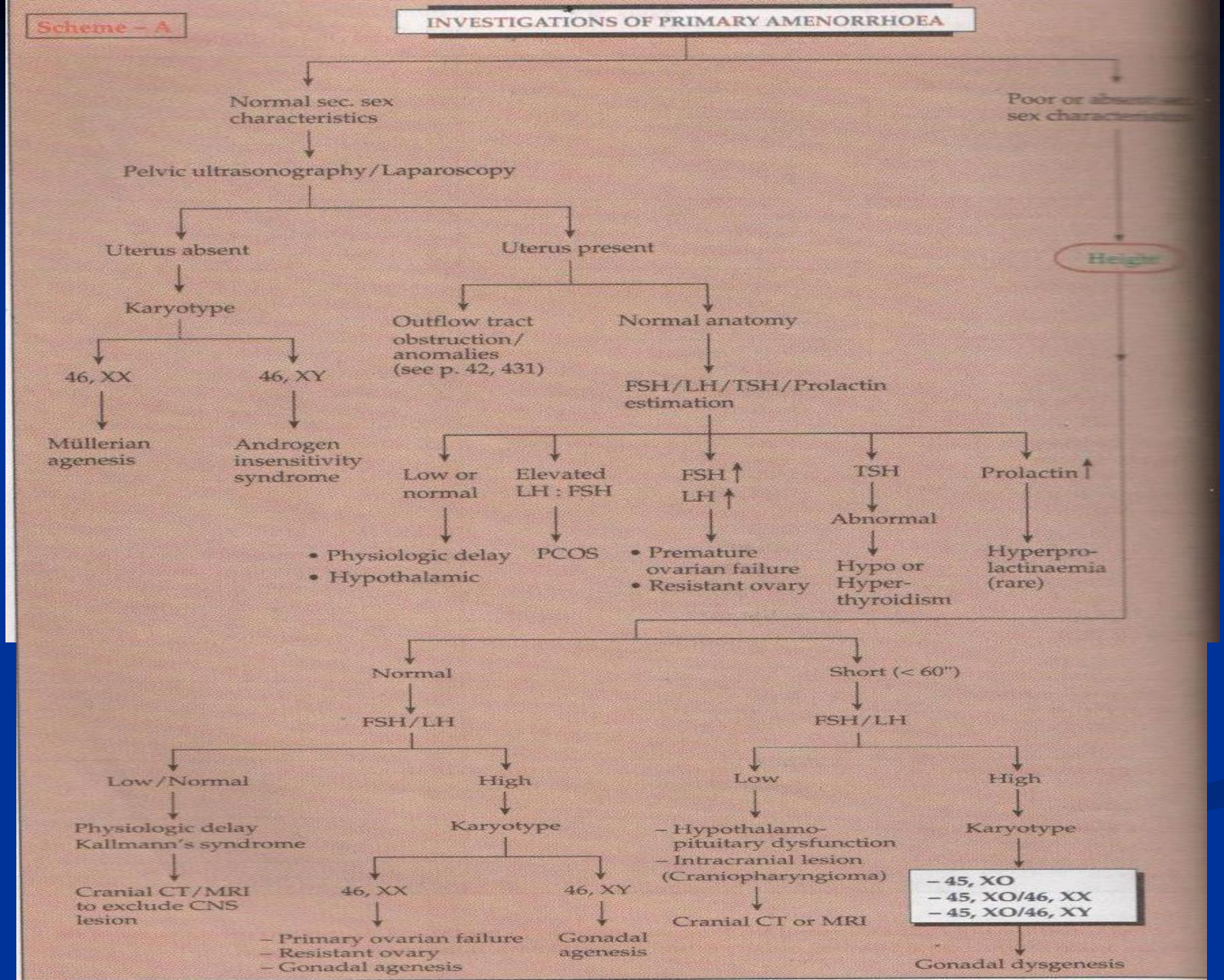
k. Primary hypothyroidism

l. cushing's syndrome

Primary amenorrhea

- Defined as the absence of menses by 13 years of age when there is no visible secondary sexual characteristic development or by 15 years of age in the presence of normal secondary sexual characteristics.

Scheme – A

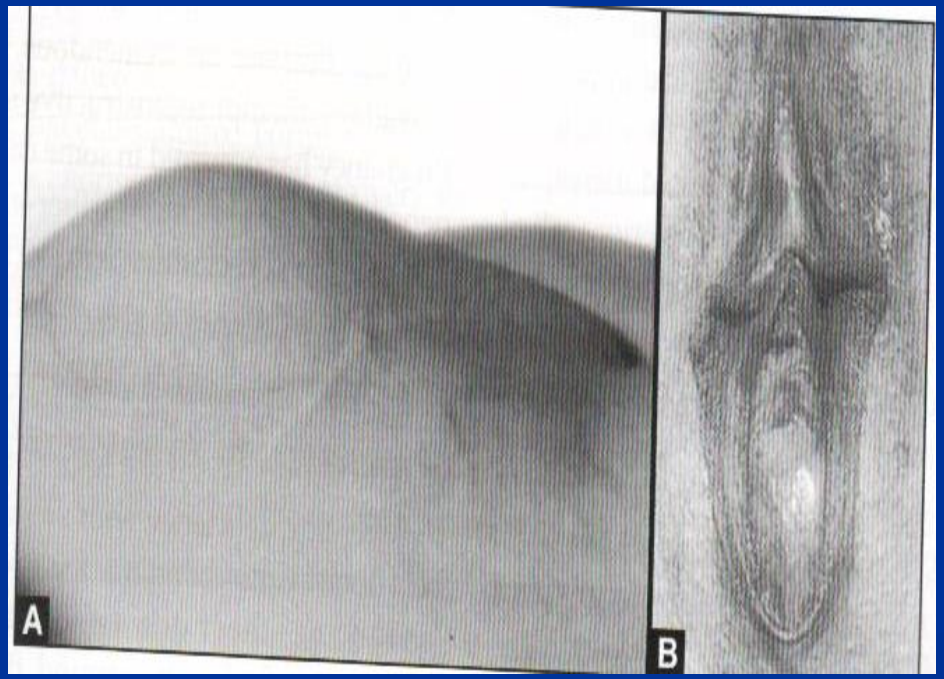

INVESTIGATIONS OF PRIMARY AMENORRHOEA
Normal sec. sex characteristics
Poor or absent sec. sex characteristics
Pelvic ultrasonography/Laparoscopy
Uterus absent
Uterus present
Height
Karyotype
Outflow tract obstruction/ anomalies (see p. 42, 431)
Normal anatomy
46, XX
46, XY
FSH/LH/TSH/Prolactin estimation
Müllerian agenesis
Androgen insensitivity syndrome
Low or normal
Elevated LH : FSH
FSH ↑ LH ↑
TSH
Prolactin ↑
Abnormal
• Physiologic delay
• Hypothalamic
PCOS
• Premature ovarian failure
• Resistant ovary
Hypo or Hyper-thyroidism
Hyperpro-lactinaemia (rare)
Normal
Short (< 60")
FSH/LH
FSH/LH
Low/Normal
High
Low
High
Physiologic delay Kallmann's syndrome
Karyotype
– Hypothalamo-pituitary dysfunction
– Intracranial lesion (Craniopharyngioma)
Karyotype
Cranial CT/MRI to exclude CNS lesion
46, XX
46, XY
– 45, XO
– 45, XO/46, XX
– 45, XO/46, XY
– Primary ovarian failure
– Resistant ovary
– Gonadal agenesis
Gonadal agenesis
Cranial CT or MRI
Gonadal dysgenesis

Diagnosis of amenorrhea based on clinical exam

PHYSICAL APPEARANCE	EXTERNAL GENITALIA	INTERNAL GENITALIA	PROBABLE DIAGNOSIS
▪Normal breast development ▪Normal sexual hair ▪Average stature	Normal	▪Absent vagina ▪Absent uterus	▪Mullerian agenesis
▪Normal breast development ▪Normal sexual hair ▪Average stature	Normal	Normal	▪Unresponsive endometrium (receptor defect) ▪Uterine synechiae ▪Obstructive lesion
▪Tall and lanky	Under-developed	Underdeveloped	Primary ovarian failure
▪Poor breast development ▪Scanty pubic hair ▪Average stature	Under - developed	Underdeveloped (vaginal rugosity absent)	Hypogonado-trophic hypogonadism

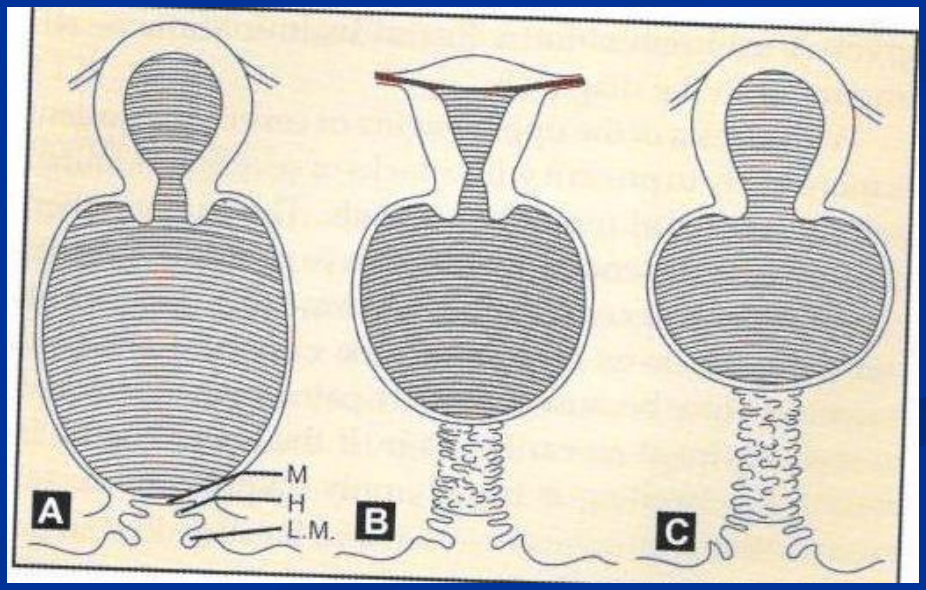
Distal outflow tract obstruction

- Imperforate hymen
  A. abdominal swelling
  B. bulging hymen



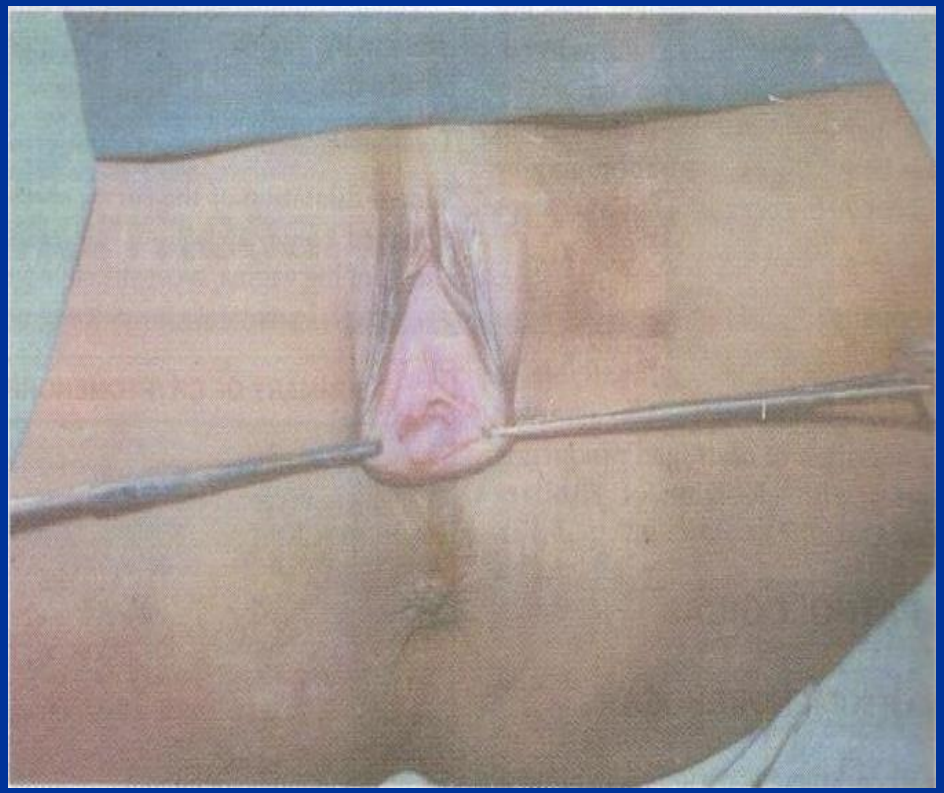
- Haematometra

1. Imperforate hymen
2. Lower part of vagina not developed
3. Whole vagina not developed

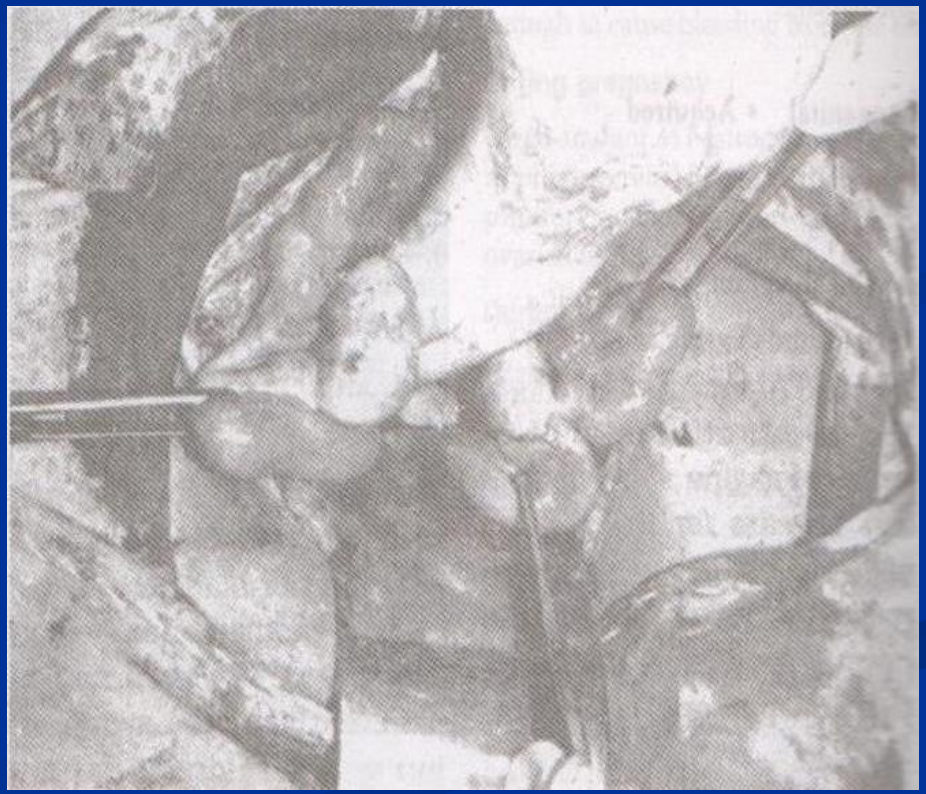


Mullerian agenesis

- Complete absence of vagina

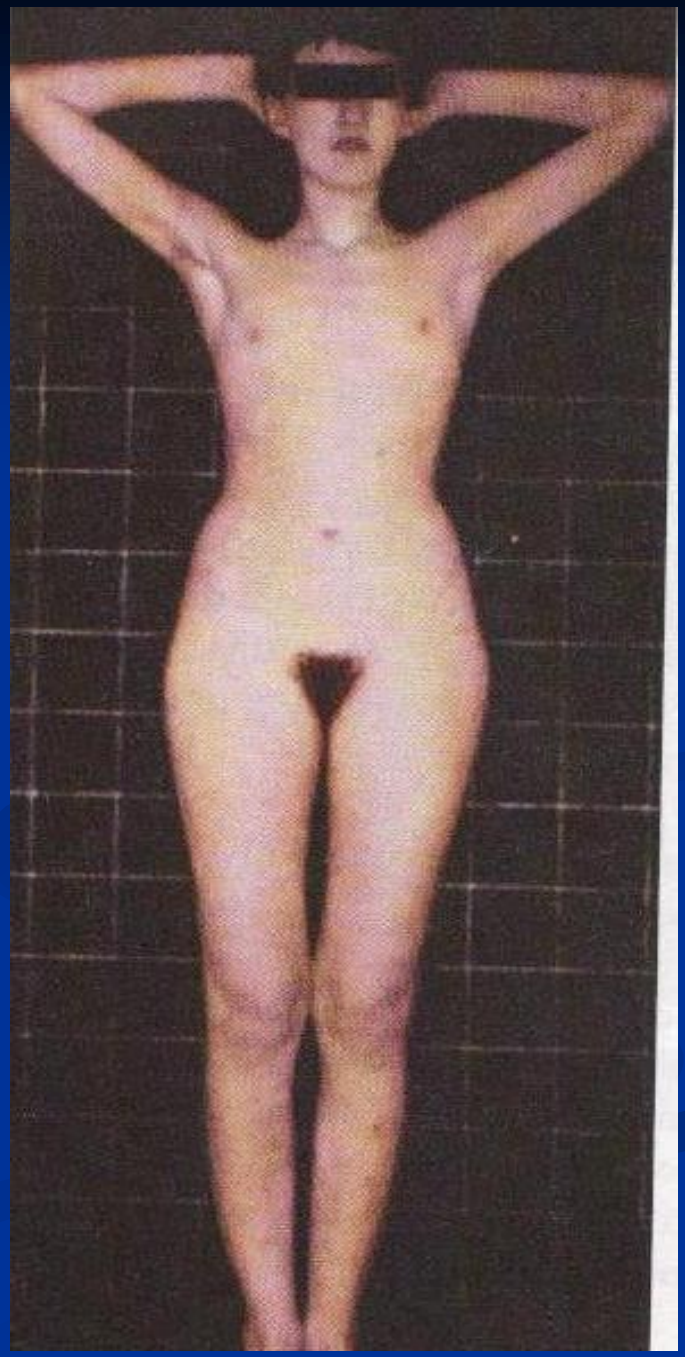


- Mullerian agenesis with normal gonads



Kallmann syndrome

- 21y 6mths age
- Scanty pubic and axillary hair
- Bone age 16 yrs

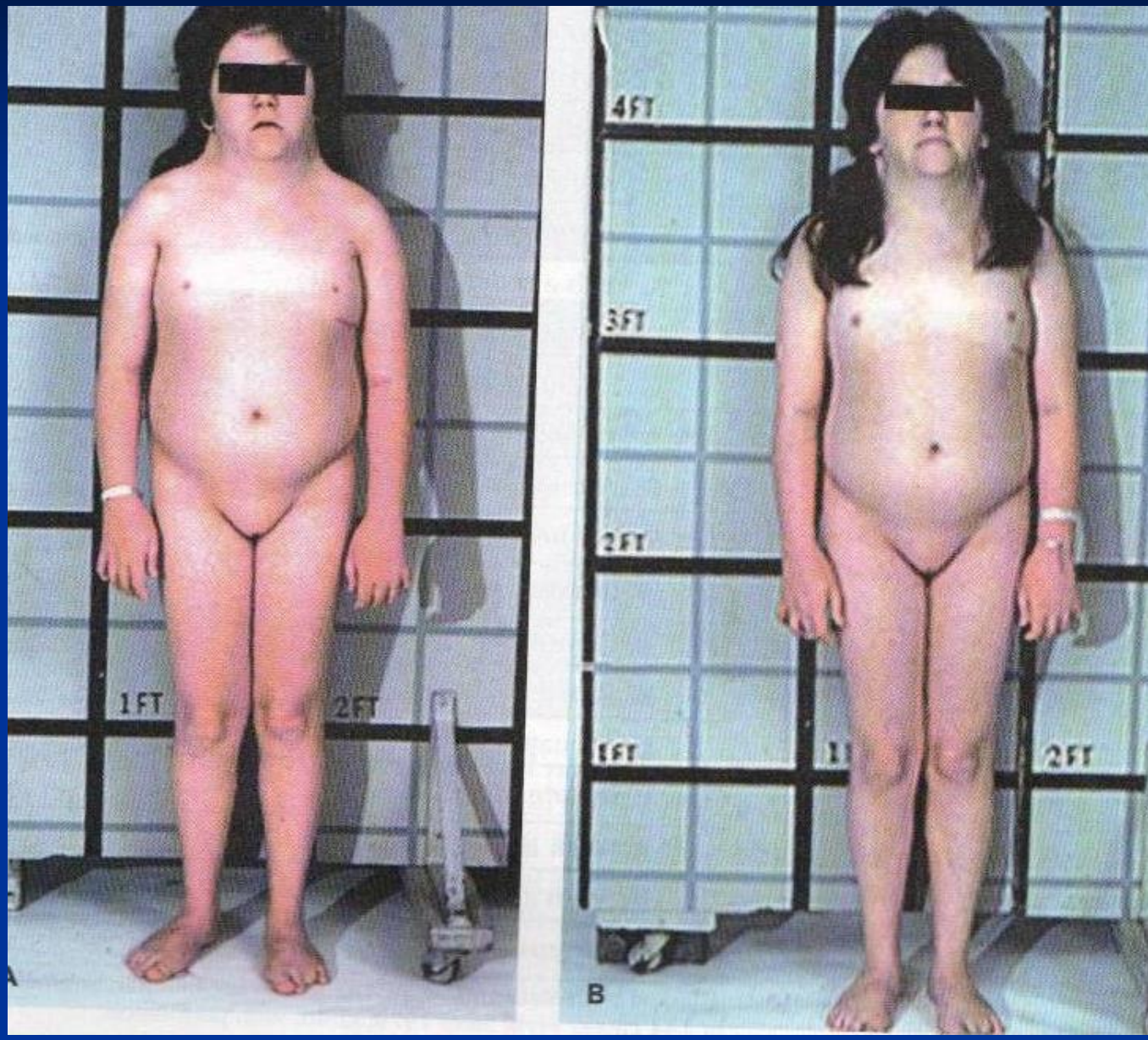


▪Poor secondary sexual characteristics ▪Short stature ▪Webbing of neck ▪Cubitus valgus ▪Congenital malformations of cardiac, renal or great vessels	Under – developed	'streak' gonads	Turner's syndrome
▪Phenotypically female ▪Average height ▪Delayed secondary sexual characters	Normal	Bilateral 'streak' gonads	Pure gondal dysgenesis
▪Normal breast development without areolar pigmentation ▪Scanty pubic and axillary hair ▪Average stature	Labial or inguinal gonads	▪Short blind vagina ▪Absence of uterus	Androgen insensitivity syndrome

Turner's syndrome

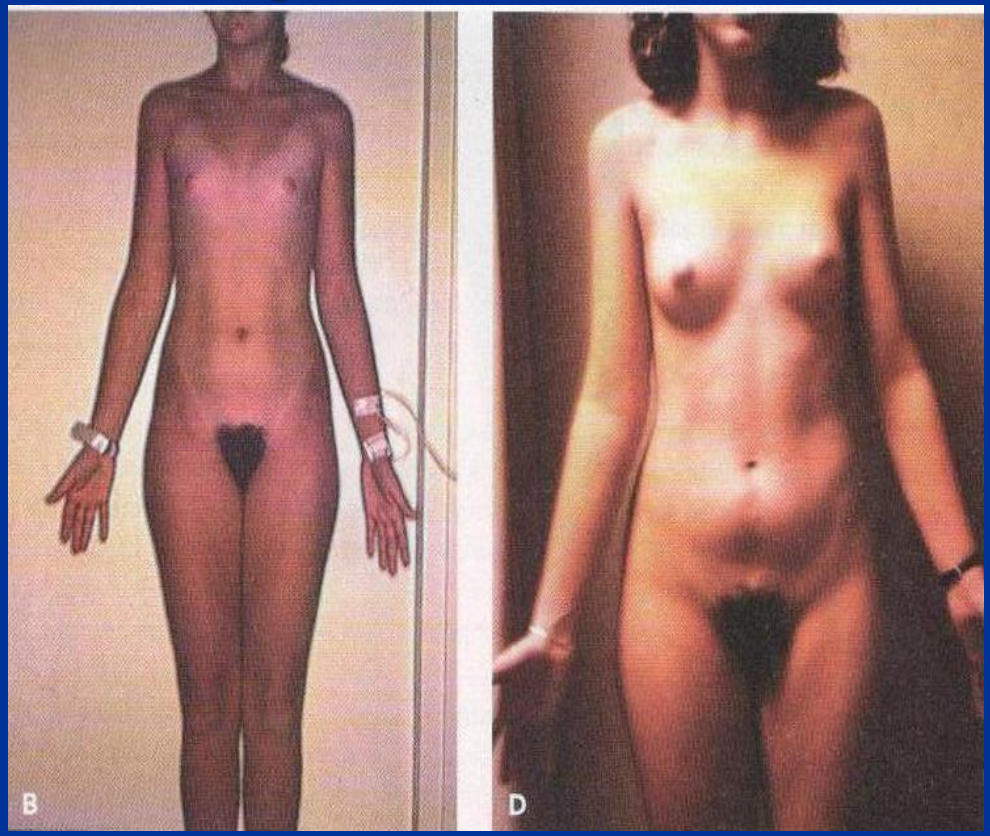
A. 16 yr old with short stature, webbed neck, shortened fourth metatarsals, underdeveloped secondary sexual characteristics

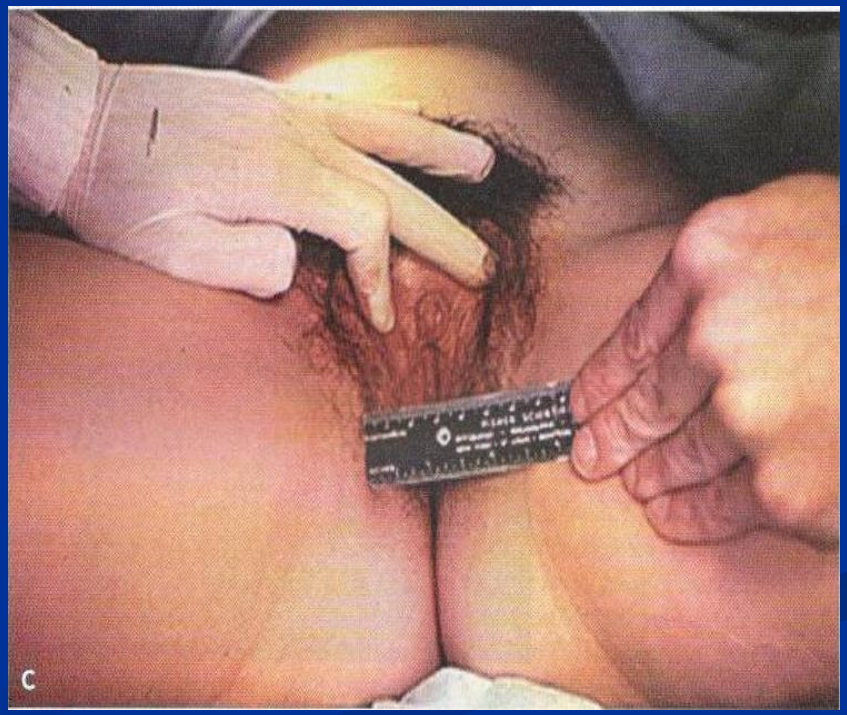
B. 11 yr old with similar features



16 yr old with 46 XY gonadal dysgenesis

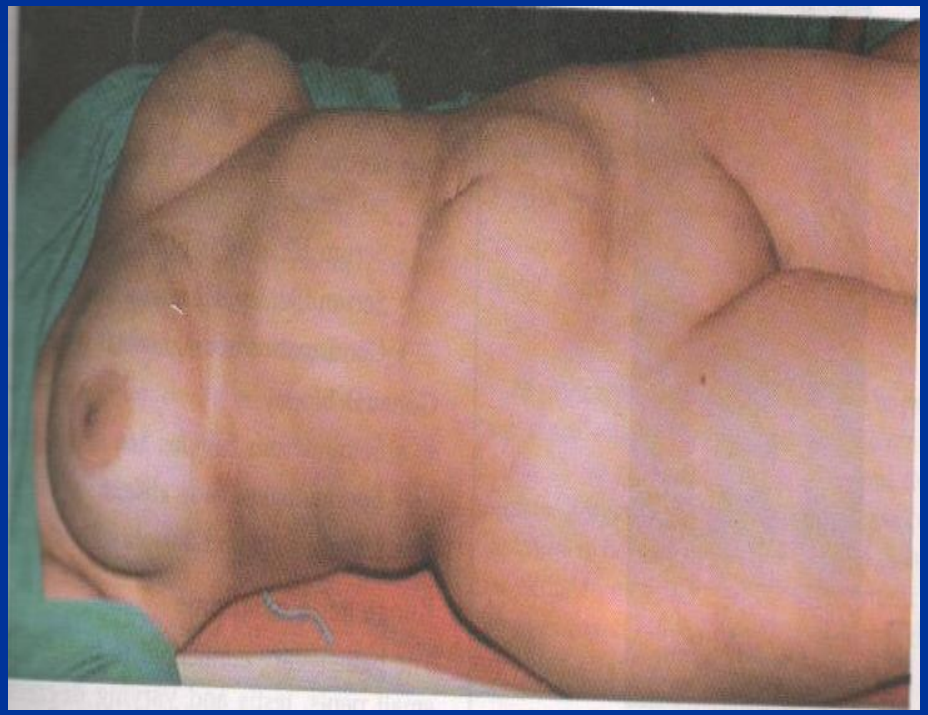
- Before and after gonadectomy and replacement with exogenous estrogen
- Clitoromegaly

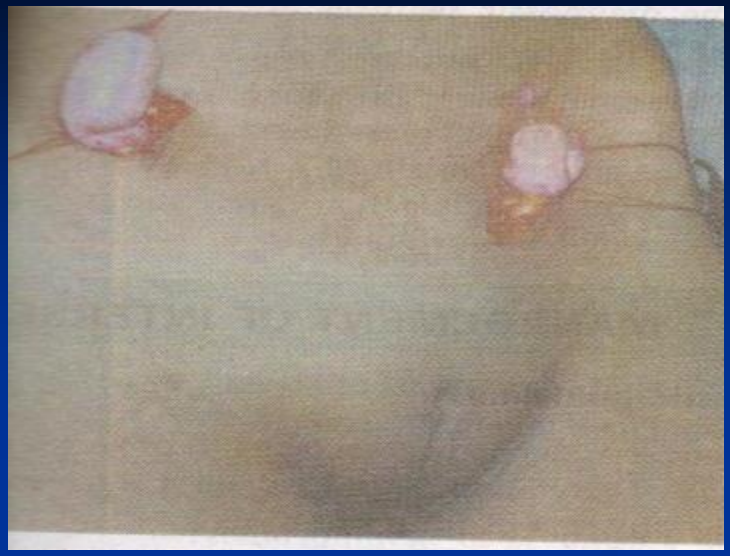




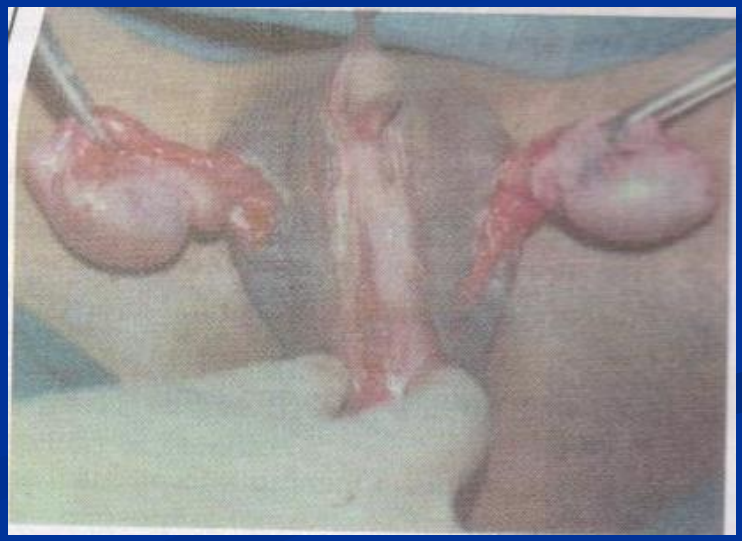
Androgen insensitivity syndrome

- Normal breast development with scanty pubic hair





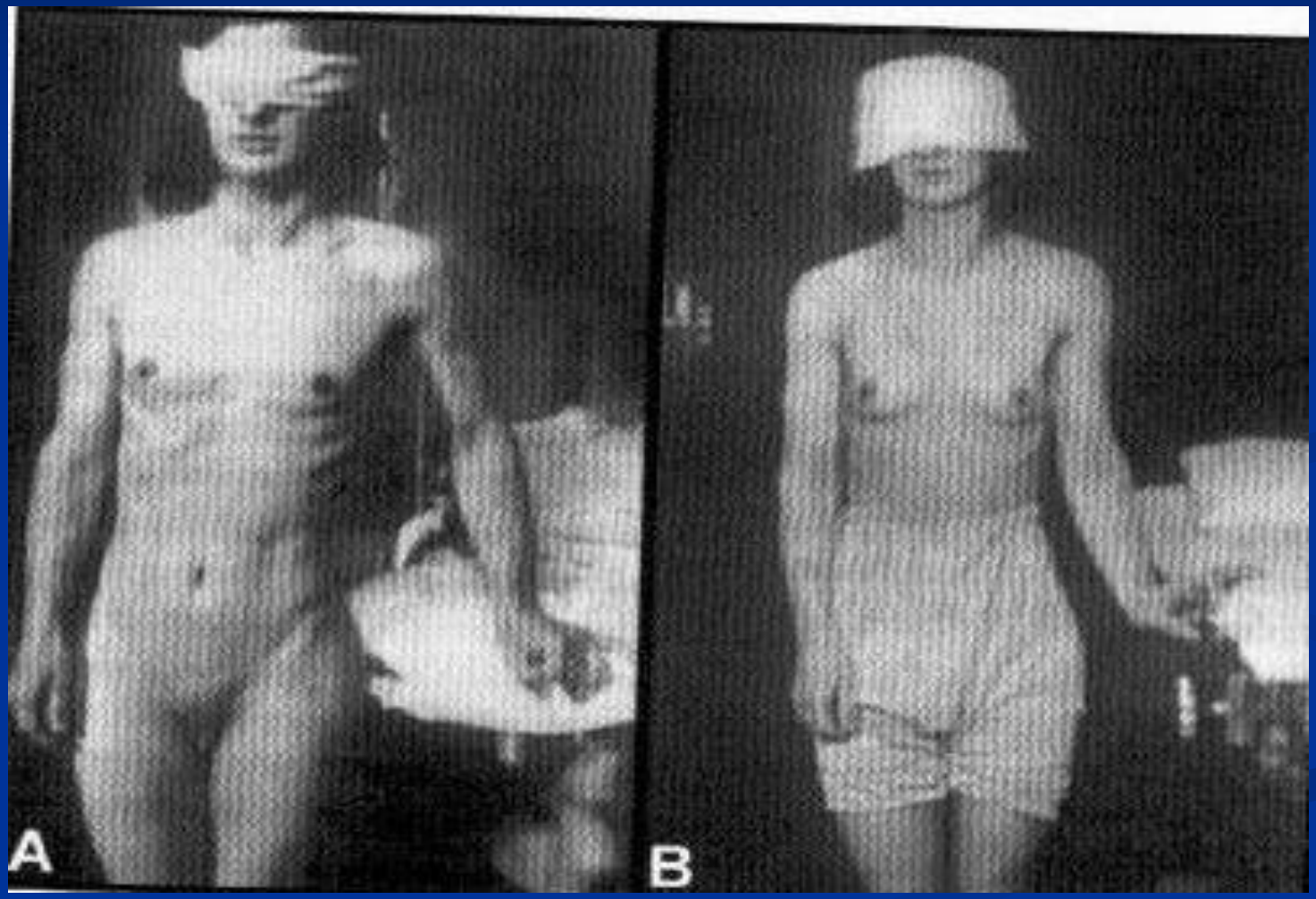
Bilateral inguinal gonads



Bilateral labial gonads with short blind vagina

▪Normal phenotypically female ▪Average stature	▪Labial fusion ▪Enlargement of clitoris	Normal	Adrenogenital syndrome (late onset)
Features of hypogonadism ▪Short stature ▪Mental retardation ▪Obesity	Under-developed	Under-developed	▪Cretinism due to hypothyroidism ▪Hypothalamo-pituitary dysfuntion (hypogonadotropic) -kallman syndrome -Prader Labhardt Willi syndrome -Laurence Moon Bardet Biedel syndrome

Late onset adrenogenital syndrome caused by adrenal tumour before and after treatment by tumour resection



Treatment

Hypergonadotrophic:

- Testicular feminising syndrome – gonadectomy
- Streak ovaries -

  cyclic estrogen and progesterone for development of secondary sexual characters and prevention of osteoporosis
- Resistant ovarian syndrome: not amenable to treatment

Hypogonadotropic:

- Hormone therapy - start with unopposed estrogen 0.3 mg conjugated estrogen or 0.5 mg estradiol daily. After 6 months to 1 year, 0.625 mg conjugated or 1 mg estradiol and 5 mg medroxy progesterone acetate or equivalent progesterone for last 14 days each month)
- Pulsatile GnRH: not practical
- Bromocriptine may be used for hypothalamic lesions

General treatment:

- Correction of diet (obesity, anorexia nervosa)
- Proper working conditions
- Proper home environment

Eugonadotropic:

- Mullerian agenesis: functional vagina formation by surgery
- Cryptomenorrhea: cruciate incision of hymen
- Septate vagina or atresia: excision and vaginoplasty
- If no cause found – treat as secondary amenorrhea

PUBERTY MENORRHAGIA

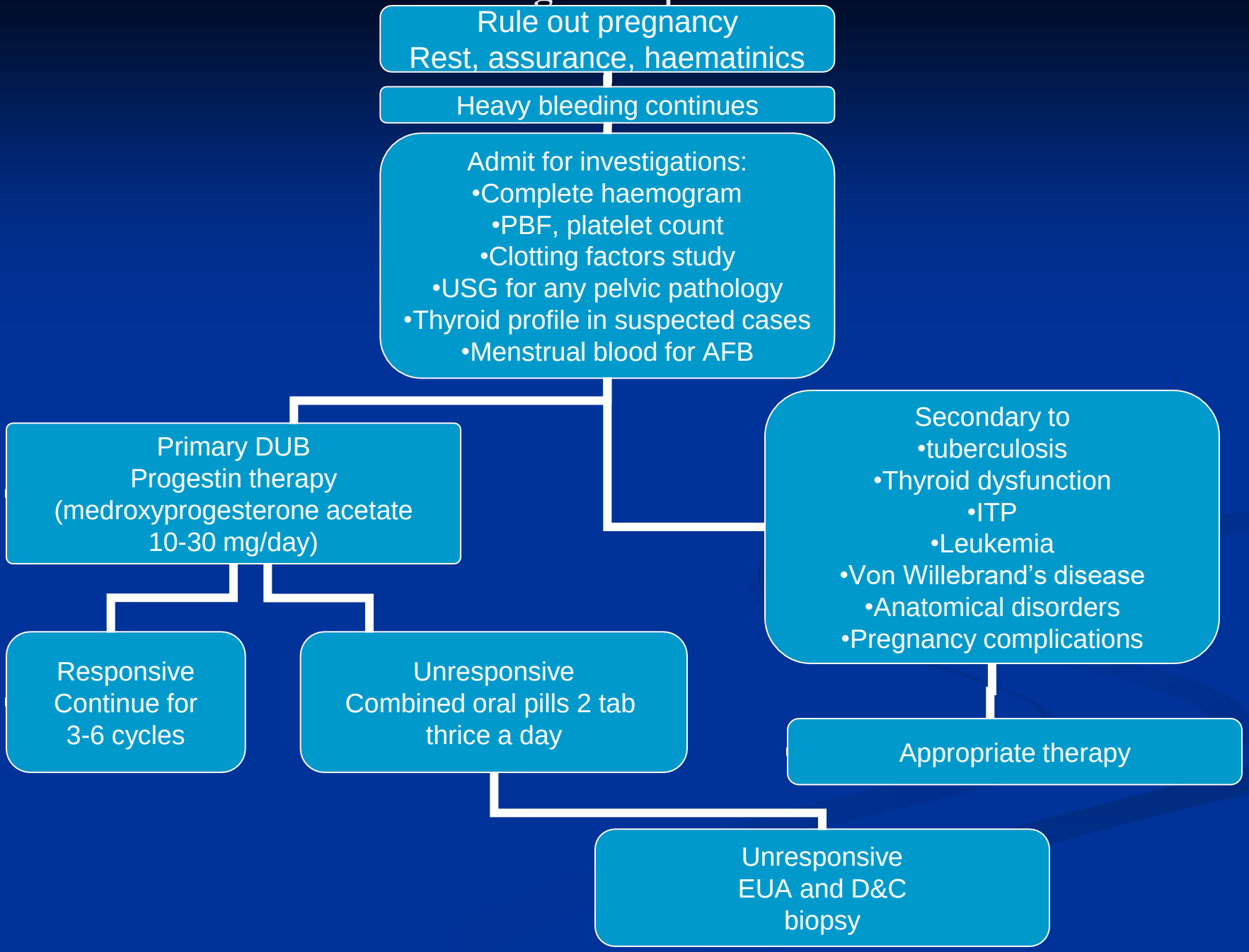
- Causes:

1. Dysfunctional uterine bleeding (95%)
2. Tuberculosis
3. Endocrine dysfunction – PCOS
   hypo or hyper thyroidism
4. Haematological – ITP
   von Willebrand's disease
   leukemia
5. Pelvic tumours – fibroid uterus
   sarcoma botryoides
   estrogen producing ovarian tumour
6. Pregnancy complications

Pathology

- It is usually anovulatory bleeding
- Estrogen withdrawal bleeding
- Focal breakdown of overgrown and structurally fragile endometrium under continuous estrogen stimulation
- Regression of most recent follicular cohort

Management protocol



Treatment:

- Explanation, reassurance, psychological support
- Haematinics
- Blood transfusion may be required in emergency
- Refractory cases:

  progesterone – medroxy progesterone acetate or norethisterone 5 mg TDS till bleeding stops (controlled in 3-7 days)
- Condition usually becomes normal following 2-3 courses. Regular menstrual cycles established once HPO axis matured

DYSMENORRHEA

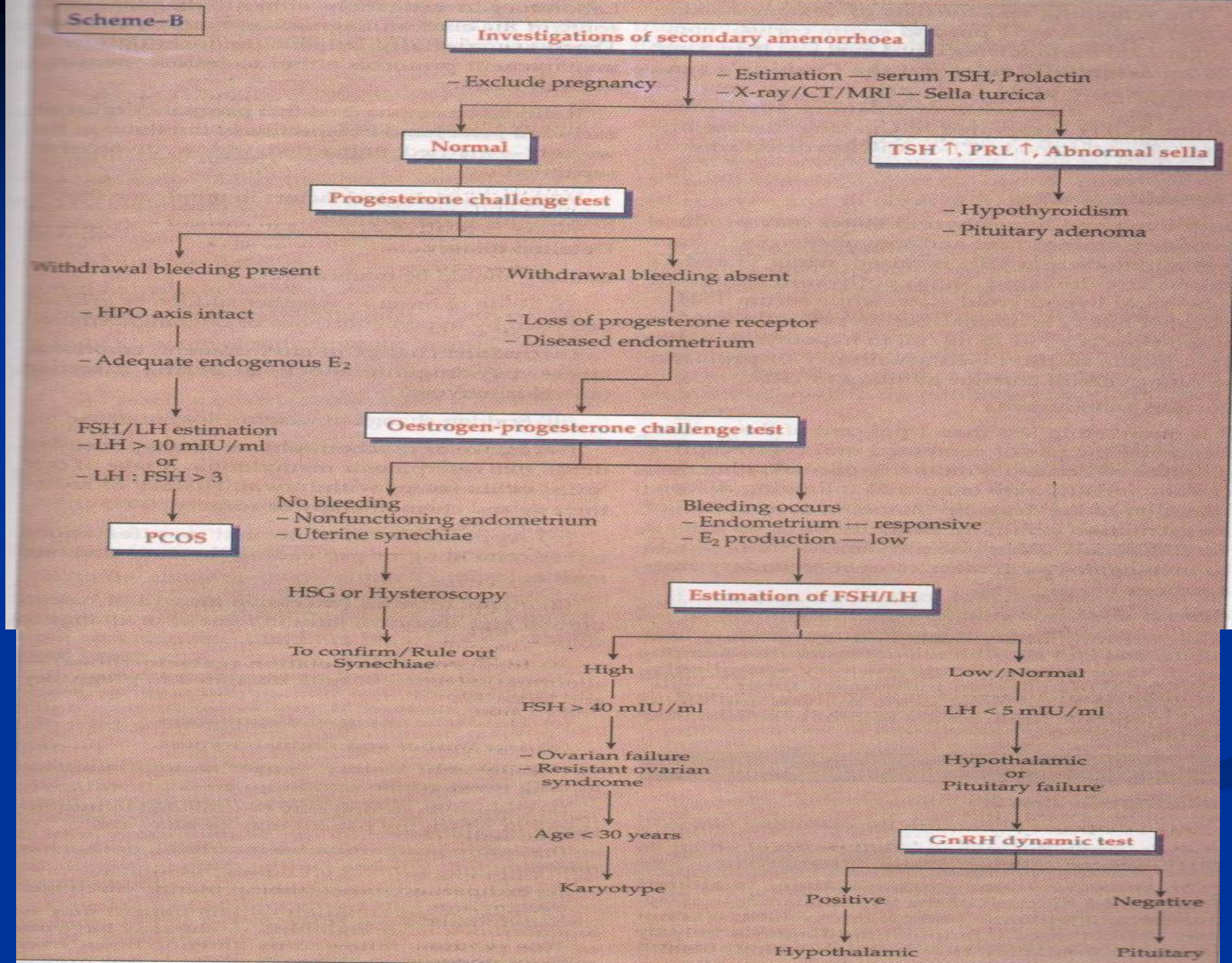
- Common in adolescence with onset of ovulation
- Spasmodic and colicky pain beginning premenstrually and lasting until day 2-3 of cycle, associated with vomiting and diarrhea in severe cases
- Treatment: 1. simple analgesics eg. Aspirin
  2. if ineffective prostaglandin synthetase inhibitors such as mefenemic acid 250-500mg TDS can be tried
  3. severe cases – OCPs
- Rule out endometriosis if no response seen to therapy – diagnostic laparoscopy
  usually begins 3 or more years after menarche
  Laufer and colleagues have reported endometriosis in 67% adolescents with refractory dysmenorrhea
- Intractable dysmenorrhea: uterosacral nerve ablation

SECONDARY AMENORRHEA

Defined as absence of menstruation for longer than 6 months after menstruation is established

Causes:

Physiological	pregnancy
Pathological	hypothalamic/pituitary stress weight loss hypogonadotropic hypogonadism prolactinoma panhypopituitarism
Ovarian	polycystic ovarian disease (25%) hormone secreting tumour chemotherapy, radiotherapy surgery
systemic causes	thyroid adrenal tuberculosis

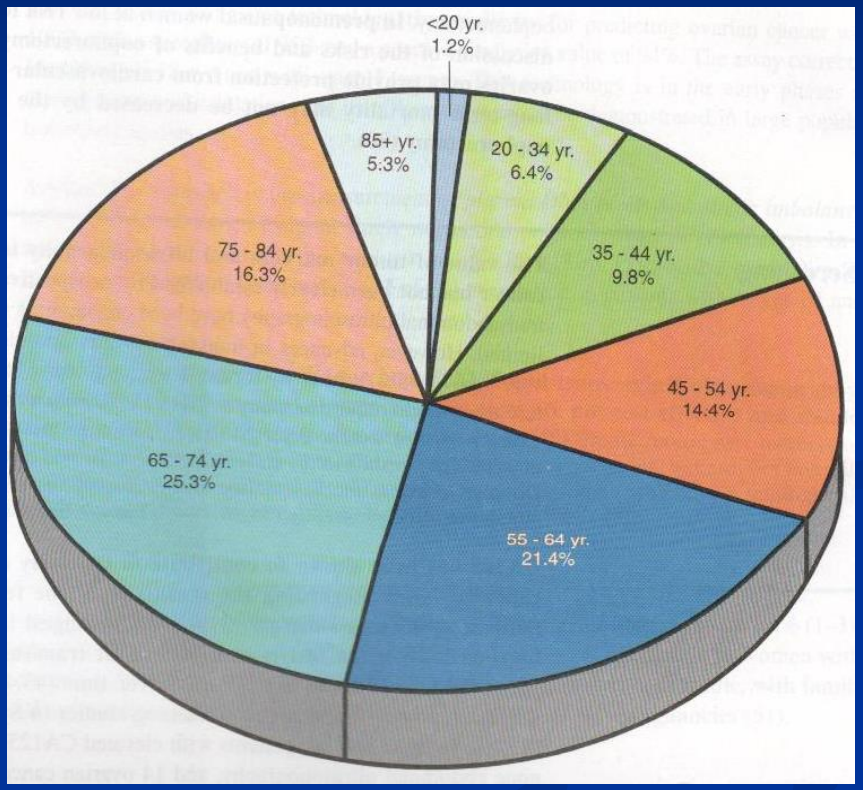

Scheme–B
Investigations of secondary amenorrhoea
– Exclude pregnancy
– Estimation — serum TSH, Prolactin
– X-ray/CT/MRI — Sella turcica
Normal
TSH ↑, PRL ↑, Abnormal sella
– Hypothyroidism
– Pituitary adenoma
Progesterone challenge test
Withdrawal bleeding present
– HPO axis intact
– Adequate endogenous E_2
FSH/LH estimation
– LH > 10 mIU/ml
or
– LH : FSH > 3
PCOS
Withdrawal bleeding absent
– Loss of progesterone receptor
– Diseased endometrium
Oestrogen-progesterone challenge test
No bleeding
– Nonfunctioning endometrium
– Uterine synechiae
HSG or Hysteroscopy
To confirm/Rule out Synechiae
Bleeding occurs
– Endometrium — responsive
– E_2 production — low
Estimation of FSH/LH
High
FSH > 40 mIU/ml
– Ovarian failure
– Resistant ovarian syndrome
Age < 30 years
Karyotype
Low/Normal
LH < 5 mIU/ml
Hypothalamic or Pituitary failure
GnRH dynamic test
Positive
Hypothalamic
Negative
Pituitary

Management

- Stress/ weight loss : reassurance and diet control
- Prolonged amenorrhea : hormone replacement
- Space occupying lesions of hypothalamus : neurological / neurosurgical assessment
- PCOD: cyclic estrogen/progesterone or OCP
- Tuberculosis: ATT category I

NEOPLASMS

- Gynaecological tumours represent 1.5 to 2% of all malignancies in this age group
- Nonepithelial tumours predominate
- Ovarian tumours represent 1.5% of all childhood tumours
- Germ cell tumours make up one half to two thirds of ovarian neoplasms in <20 yrs age group
- Ovarian neoplasms are malignant in about 25% in childhood



incidence of ovarian cancer by age

Ovarian tumours

Functional ovarian cysts/ benign tumours:

- Among most common pelvic masses in adolescence
- Usually 6-8 cm in diameter
- Present with abdominal pain
- USG for diagnosis
- Treatment:

  usually regress within 3-6 months

  simple cyst resection

  rarely salpingo-oophorectomy if there is torsion with loss of tissue viability

Germ cell tumours:

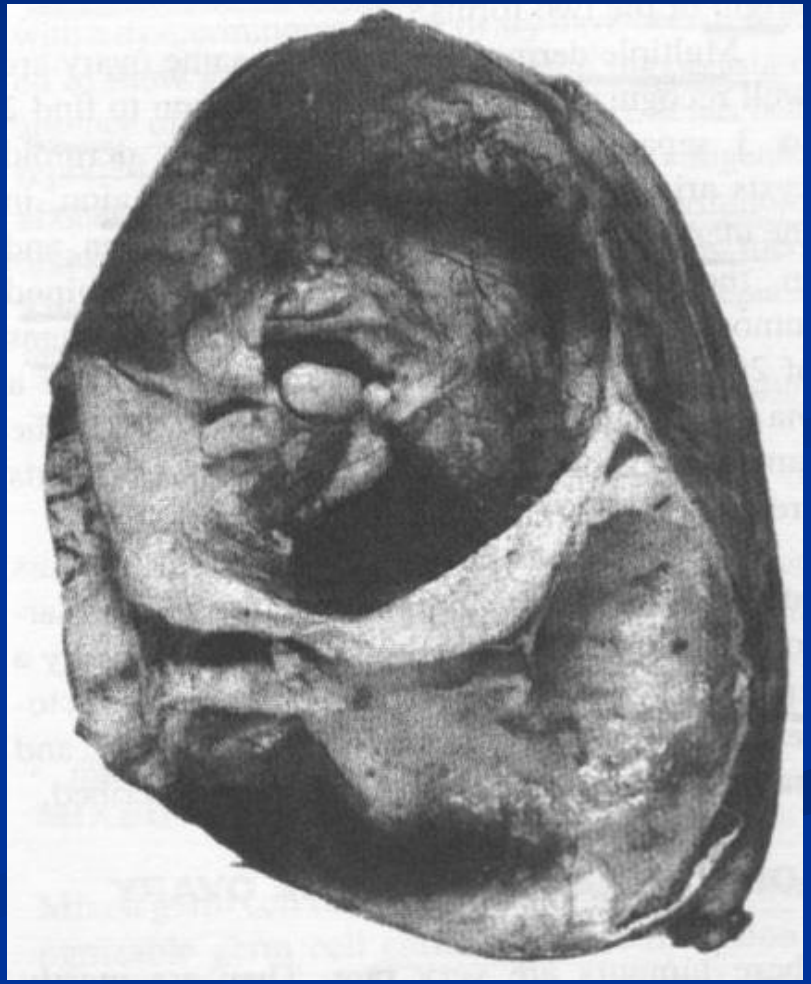
below the age of 20 yrs, 60% tumours are of germ cell origin

<10 yrs age almost 85% belong to this group, invariably malignant

Teratomas:

- Unilateral or bilateral (10%)
- About 50% of pure immature teratomas occur between 10 to 20 yrs age
- Represent 10-20% of all ovarian malignancies in <20 yrs age group
- Contain ectodermal, mesodermal and endodermal tissue
- Present with asymptomatic abdominal mass or abdominal pain
- USG : multicystic tumour with mixed solid and cystic areas
- X-ray: calcifications or teeth like structures
- Treatment: surgical removal

preserve as much part of ovary as possible



Dysgerminomas

- Develop from primordial germ cells
- Termed as malignant, though do not have high malignant potential. Early progression to lymph nodes.
- 60% - seen in first 2 decades of life
- Present as rapidly growing tumour
- Treatment:

  obvious malignancy with involvement of opposite ovary, peritoneum, ascites – TAH with BSO followed by radiation

  less aggressive – unilateral oophorectomy and lymphadenectomy followed by chemotherapy
- Follow up necessary

Yolk sac tumours:

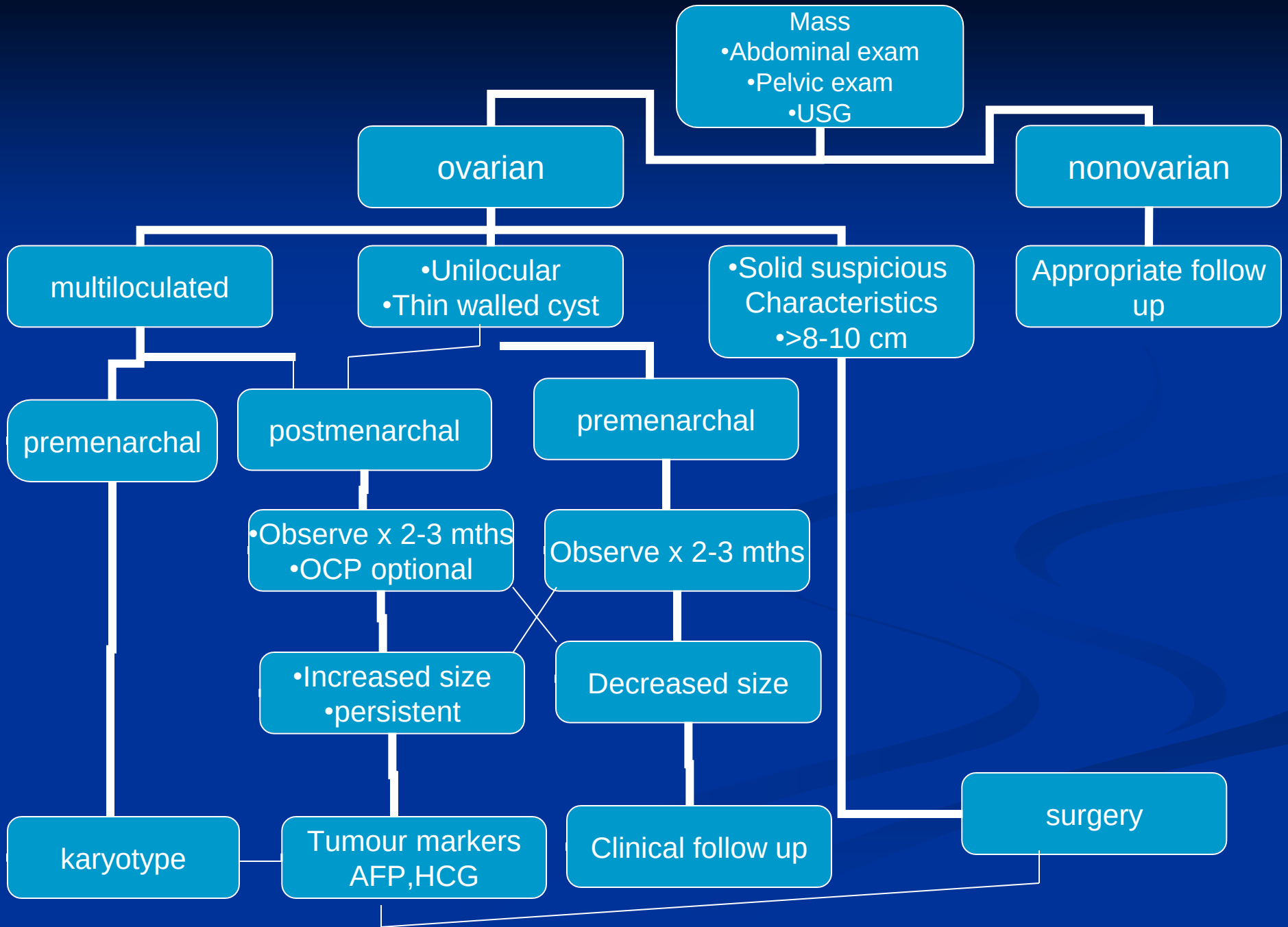
- Secrete αFP and hCG
- Present as pelvic abdominal masses
- Highly malignant with short survival
- Mostly between 15-20 years age group
- Treatment : surgical staging and unilateral salpingo-oophorectomy followed by chemotherapy

Sex cord stromal tumours:

Granulosa cell tumours:

- Rare <5% in prepubertal girls
- Secrete estrogen and sometimes prolactin – precocious pseudopuberty, galactorrhea
- Present as pelvic mass
- Good prognosis
- Treatment: unilateral oophorectomy with long term follow up

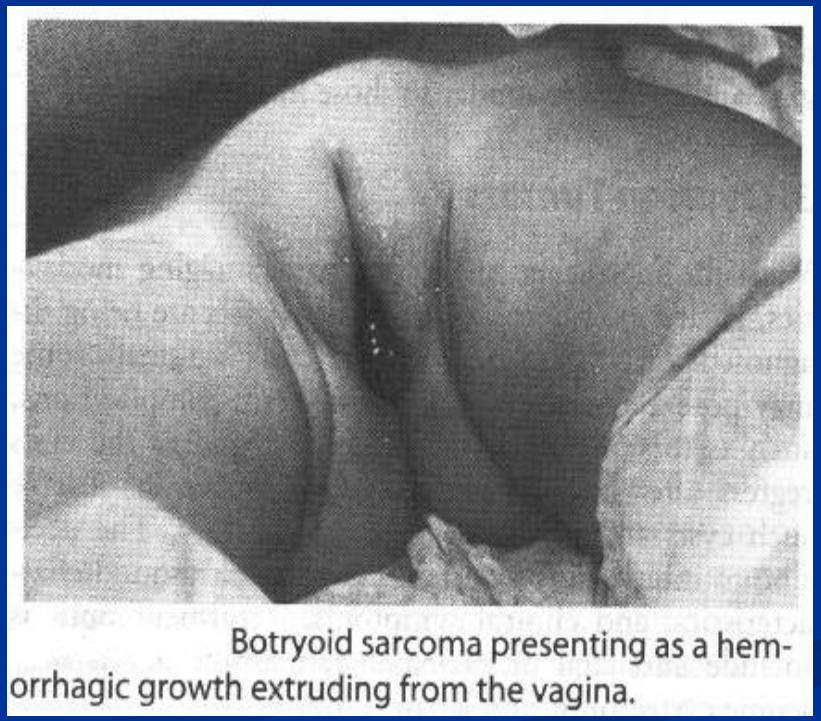
Management of pelvic masses in premenarchal and adolescent girls



Cervical vaginal tumours

Botryoid sarcoma (rhabdomyosarcoma):

- Frequently affecting <2 years age group
- May occur in cervix in older girls and adolescents
- Presents with blood stained vaginal discharge, occasional frank bleeding
- Exam: fleshy haemorrhagic lesion (grape like)
- Tumour originates in subepithelial layer of cervical/vaginal epithelium and spreads widely within this layer producing classical polypoid appearance before invading vaginal wall. Later spreads via lymphatics and blood stream
- Treatment: combination chemotherapy over 6 mths followed by extended hysterectomy and vaginectomy
- Response to chemo good – continued for 6-12 mths
- Prognosis - poor



Botryoid sarcoma presenting as a hemorrhagic growth extruding from the vagina.

Clear cell adenocarcinoma:

- Affects vagina and cervix
- Secondary to DES exposure in utero
- DES exposure – pseudopolyp formation
  hypertrophy of endocervical tissue
  incomplete vaginal septa
  fibrous bands
  vaginal adenosis
- Present as vaginal bleeding
- Risk of cancer is 0.14 to 1.4 per 1000
- Treatment: extended hysterectomy and vaginectomy with adjunctive radiotherapy

Fertility preservation after treatment of neoplasm

- Substitution of alkylating agents, cyclical rather than continuous regimens
- Surgically taking ovaries out of field of radiation like laparoscopic suspension of ovarian tissue and ovarian transplantation in an ectopic site
- Cryopreservation of oocytes and ovarian tissue prior to treatment

OBESITY

- BMI>30 kg/m²
- Cause: overeating, hypothyroidism, polycystic ovaries
- Complication: early age of menarche, insulin resistance, infertility, oligomenorrhea, increased risk for hypertension, hypercholesterolemia, type 2 DM, osteoarthritis, sleep apnea, CHD, cancers
- Minor obesity disappears by 20 years
- Gross obesity : strict diet control 1000 kcal in 24hrs
- Goal of 10% weight loss over 6 months
- Periodic weight check necessary
- Medications: sibutramine, orlistat, rimonabant

HIRSUITISM

- Excessive growth of androgen dependent sexual hair in facial and central part of the body
- Laboratory evaluation consists of measuring circulating levels of testosterone and 17 OH progesterone, and screening for Cushing's syndrome
- Consider possibility of hyperinsulinemia
- Rapidly progressive- consider androgen secreting tumour
- Treatment: weight reduction
  remove source of androgens
  drugs: COC(low dose)
  does not respond- antiandrogen
  (spironolactone or finasteride)
  impact seen in about 6 months

PREGNANCY IN ADOLESCENTS

Complications more common in adolescents

- Anemia
- Pregnancy induced hypertension
- Pre term birth
- Prolonged and obstructed labour
- Low birth weight
- Perinatal and neonatal mortality
- Post partum depression
- Too early repeat pregnancies
- Inadequate child care and breast feeding
- Maternal mortality increased 2-5 fold

ADOLESCENT CONTRACEPTION

OCPs:

- Preferred
- Advantage of regularisation of menstrual cycle

Barrier method:

- protects against STDs

Emergency contraception:

- Education required

MTP services:

- Presently, in India it is not permitted to prescribe MTPill in unmarried girls
- Surgical methods are effective
- 38-68% of abortion complications in women <20 years age

Strategy for addressing adolescent health in RCH II

- Incorporate adolescent health issues in all RCH training programmes including unmet need for contraception and care in pregnancy
- In select districts: reorganize services at PHCs on dedicated days and dedicated timing for adolescents

THANK YOU