D: **SGA:** birth weight < 20th percentile for gestational age or < 2.5 kg.
LGA: birth weight > 90th percentile for gestational age or > 4 kg.

A: **SGA:** may be familial, constitutional, or due to IUGR.
IUGR is defined as either symmetrical or asymmetrical:
Asymmetrical IUGR: relative sparing of head circumference in relation to weight and length:
- Due to impaired uteroplacental function 2° to maternal pre-eclampsia, DM or nutritional deficiency during the 3rd trimester.
- Occurs when foetal growth rate in 3rd trimester exceeds maximal supply from the placenta.
- There is preferential sparing of the cerebral perfusion at times of foetal distress.

Symmetrical IUGR: head circumference, weight, and length are all proportionally affected to equivalent degrees:
- Indicative of a prolonged period of poor intrauterine growth.
- Caused by congenital intrauterine infections (TORCH) in the 1st trimester, genetic factors such as single gene deletions and chromosomal disorders, maternal smoking, drug and alcohol abuse, chronic medical conditions (e.g. CRF), malnutrition, or multiple pregnancies.

LGA: macrosomia is a feature of infants of mothers with either gestational or poorly controlled/undiagnosed DM.

A/R: **IUGR:** previous SGA infant, low pre-pregnancy weight and poor pregnancy weight gain.

E: **SGA:** affects by definition 20% of the population and varies with ethnic background.
IUGR: 2/200 neonates; asymmetrical > symmetrical IUGR.
LGA: affects by definition 20% of live births; is more common in developed countries where there is a higher prevalence of DM.

H&E: **Antenatal:** maternal examination and accurate dating aid diagnosis. Oligohydramnios and poor foetal movements are indications of placental insufficiency.
Perinatal monitoring: foetal tachycardia, loss of variability of the baseline in the foetal heart trace, and late decelerations may indicate foetal distress on CTG.
Postnatal measurements: birth weight, length, and head circumference on centile chart.

P: See **A**.

I: **Radiology:** USS is the 1° method of diagnosing IUGR or macrosomia antenatally.
Cordocentesis: percutaneous umbilical blood sampling may be used for detection of hypoxia, lactic acidosis, hypoglycaemia, chromosomal analysis, and DNA diagnosis of congenital intrauterine infections.

M: **Antenatal:** maternal bedrest and limitation of activity for severe IUGR.
Perinatal: maternal administration of O_2, continuous assessment of foetal well-being.
Delivery (IUGR): if foetus becomes hypoxic *in utero*, an emergency Caesarean section is required.
Macrosomia: induce at 38/40 to prevent complications in a unit with good neonatal facilities.

Abnormal size at birth continued

C: **IUGR foetus:** intrauterine hypoxia, birth asphyxia, and death.
IUGR infant: hypothermia (relatively large surface area), hypoglycaemia (poor fat and glycogen stores), hypocalcaemia, polycythaemia, and meconium aspiration.
LGA: birth asphyxia due to prolonged/difficult delivery, birth trauma, especially shoulder dystocia, hypoglycaemia in the neonatal period due to hyperinsulinism, and polycythaemia.

P: Depends on the cause of abnormal size at birth. Infants with asymmetrical IUGR will rapidly put on weight in the postnatal period; symmetrical IUGR infants are more likely to remain small permanently. Studies have shown that IUGR infants are at ↑risk of developing ↑BP, Type II DM, and coronary heart disease.

D: Inflammation of the pilosebaceous duct. Classified as mild, moderate, and severe.

A: **Adolescent acne:**
- ↑Sebum production: androgenic stimulation of hyper-responsive pilosebaceous units.
- Impaired normal flow of sebum: obstruction of the pilosebaceous duct by hyperkeratosis.
- Propioni acne bacteria: may play a role by producing cytokines and lipolytic enzymes.

Infantile acne: <3 months of life; transient and usually due to maternal androgens.

A/R: Puberty, may ↑ premenstrually, POS, excess cortisol (Cushing syndrome).

E: **Developed world:** affects 79–95% of the adolescent population, peaking at 14–18 years; tends to recede by early twenties.
Developing world: acne incidence is considerably lower; likely combination of environmental and genetic factors.

H: Usually self-diagnosed, acute onset, greasy skin, may be painful.

E: **Open comedones:** whiteheads; flesh-coloured papules.
Closed comedones: blackheads; black colour is due to oxidation of the melanin pigment.
Other features: pustules, nodules, cysts, scarring, and seborrhoea.
Distribution: primarily affects the face, neck, chest, and back (where sebaceous glands are most numerous).

P: Gross distension of the pilosebaceous follicle with neutrophil infiltration. Closed comedones may contain serous fluid. Severe acne can create fistulae between inflamed glands.

I: Normally none required. Investigate for endocrine disorder if acne develops during 2–10 years of age.
Bloods: FSH, LH (if female, suspect POS).
Urine: 24-h-urinary cortisol (if Cushing syndrome is suspected).

M: Many cases may not need treatment. Indication for treatment based on classification and degree of psychosocial impact. In severe acne, therapy should be commenced early to prevent scarring.
Topical preparations:
(1) Benzoyl peroxide; keratolytic agent, encourages skin peeling, and ↓ number of *P. acnes* (S/E: irritation and bleaching of clothes).
(2) Vitamin A derivatives; tretinoin, may take 3–4 months to work.
(3) Azelaic acid.

Antibiotics:
(1) Topical: clindamycin, erythromycin.
(2) Systemic: tetracycline only in >16 years. (S/E: discolours teeth and may soften bones in children.)

A gradual ↑ in *P. acne* resistance to many antibiotics has been documented; growing need to use either appropriate antibiotics or change the therapeutic strategy in favour of other regimens.
Isotretinoin (Roaccutane P.O.): vitamin A derivative, 4–6-month course only by specialist prescription for severe acne (S/E: teratogenic; females require OCP, hyperlipidaemia).
Antiandrogens: in females only; OCP or cyproterone acetate.
UVB: adjunctive therapy, but rarely used.
Advice: improvement may not be seen for at least a couple of months, use non-greasy cosmetics, wash face daily, moderate exposure to sunshine is beneficial.

Acne vulgaris continued

C: **Physical:** facial scarring (atrophic/keloid), hyperpigmentation of scars, 2° infection and fistulae.
Psychosocial: lack of self-confidence.

P: Generally improves spontaneously over months/years. Persists into adulthood in 22% of women and 3% of men.

D: Abnormalities of the female genital tract not present at birth.

A: **Labial adhesions:** adherence of the labia minora in the midline; may give the appearance of absence of the vagina. A thin pale semi-translucent membrane covers the vaginal os. Trauma causes denudation of the epithelial layer of the labia minora mucosa and leads to fibrous tissue formation; therefore sealing of the labia minora. Trauma can involve inflammatory conditions (vulvitis, vulvovaginitis), sexual abuse, or straddle injuries.
Vulvovaginitis: pruritus, vulval pain, vulval erythema, vaginal discharge or bleeding. Usually associated with poor perineal hygeine, constipation, and atopic dermatitis caused by local irritants (bubble bath, soaps, shampoo) or by occlusive clothing causing irritation. May be caused by trauma 2° to abuse; therefore this should be considered if other concerns are present.

A/R: Vulvovaginitis is often misdiagnosed as a UTI due to its similar presentation.

E: **Labial adhesions:** peak age: 3 months to 6 years, incidence: 1–2%.
Vulvovaginitis: very common in $<$ 5-year-olds.

H: **Labial adhesions:** usually asymptomatic and noted on routine examination. Some patients may leak urine when they stand after voiding.
Vulvovaginitis: history should include toilet-training, type of nappy used, bad odour or dark discharge, scratching, history of eczema, allergic rhinitis, or diarrhoea, tendency of child to insert objects, and any possible indication of abuse.

E: **General:** should be by a skilled clinician, in a well-lit room with a relaxed and distracted child (mother reading book).
Labial adhesions: the edges of the labia minora are sealed along the midline, beginning at the posterior fourchette and extending anteriorly towards the clitoris.
Vulvovaginitis: commonly, only vulvitis will be detected, although vaginal discharge and bleeding may also be present.

P: See **A**.

I: Exclude other vaginal disorders such as imperforate hymen or septate vagina prior to treatment.
Microbiology: vaginal swab if discharge present, MSU.
Radiology: indirect cystourethrogram may show urinary retention behind the fused labia, bladder distention $+/-$ hydronephrosis in labial adhesions.

M: **Labial adhesions:** oestrogen cream dissolves the adhesions in 90% of cases. Once adhesions have been lysed vasoline is used as prophylaxis for 1–2 months.
Vulvovaginitis:
- Treat any underlying infection with appropriate antibiotics.
- Education of adequate perineal hygiene and removal of potential irritants.

C: **Labial adhesions:** without adequate treatment 20–40% will develop UTI.

P: **Labial adhesions:** recurrence is common, therefore good follow-up is required.
Vulvovaginitis: outcome good with improved perineal hygiene.