MNEMONIC

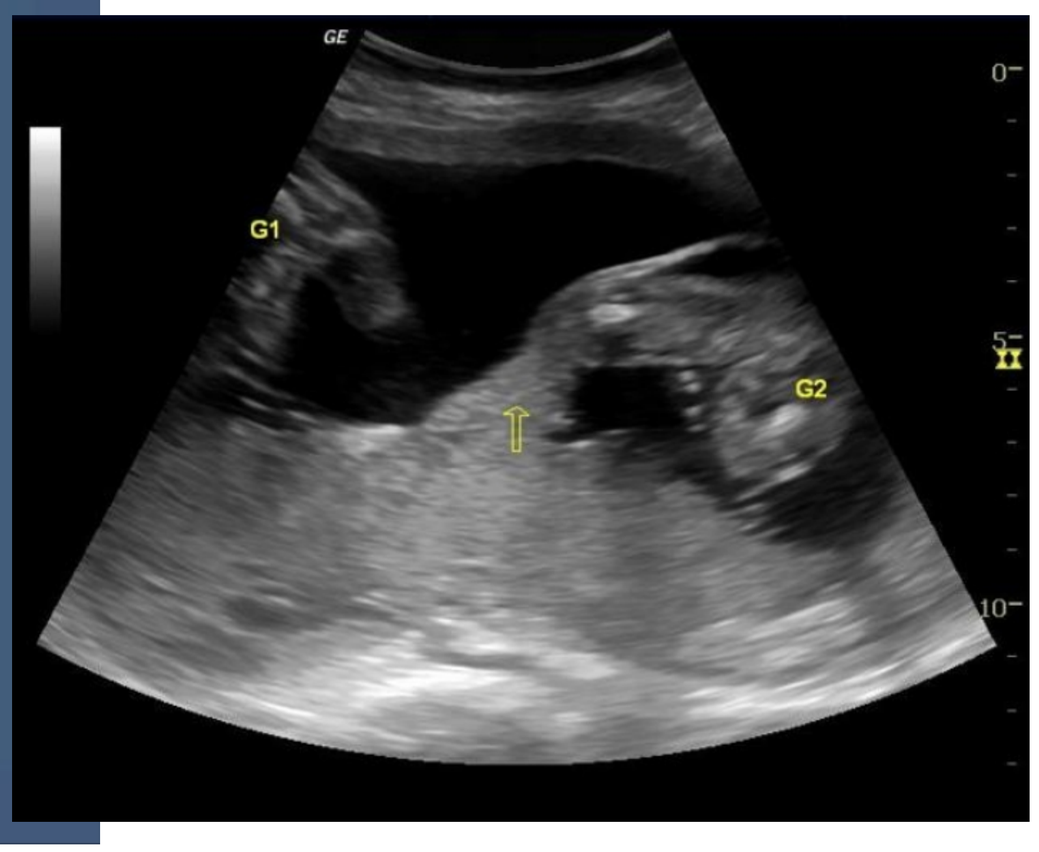
Dichorionic Diamniotic

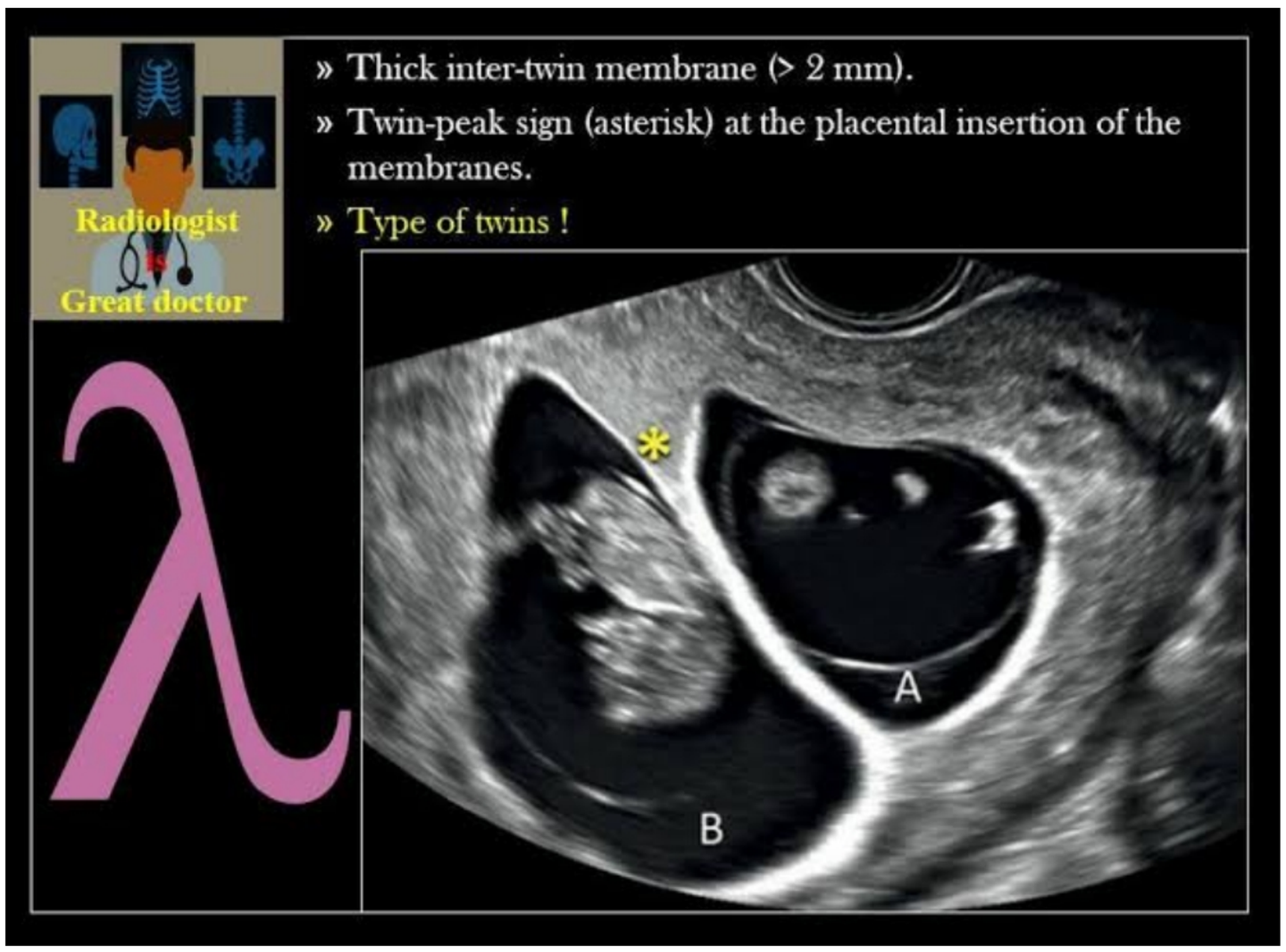
LaDDu

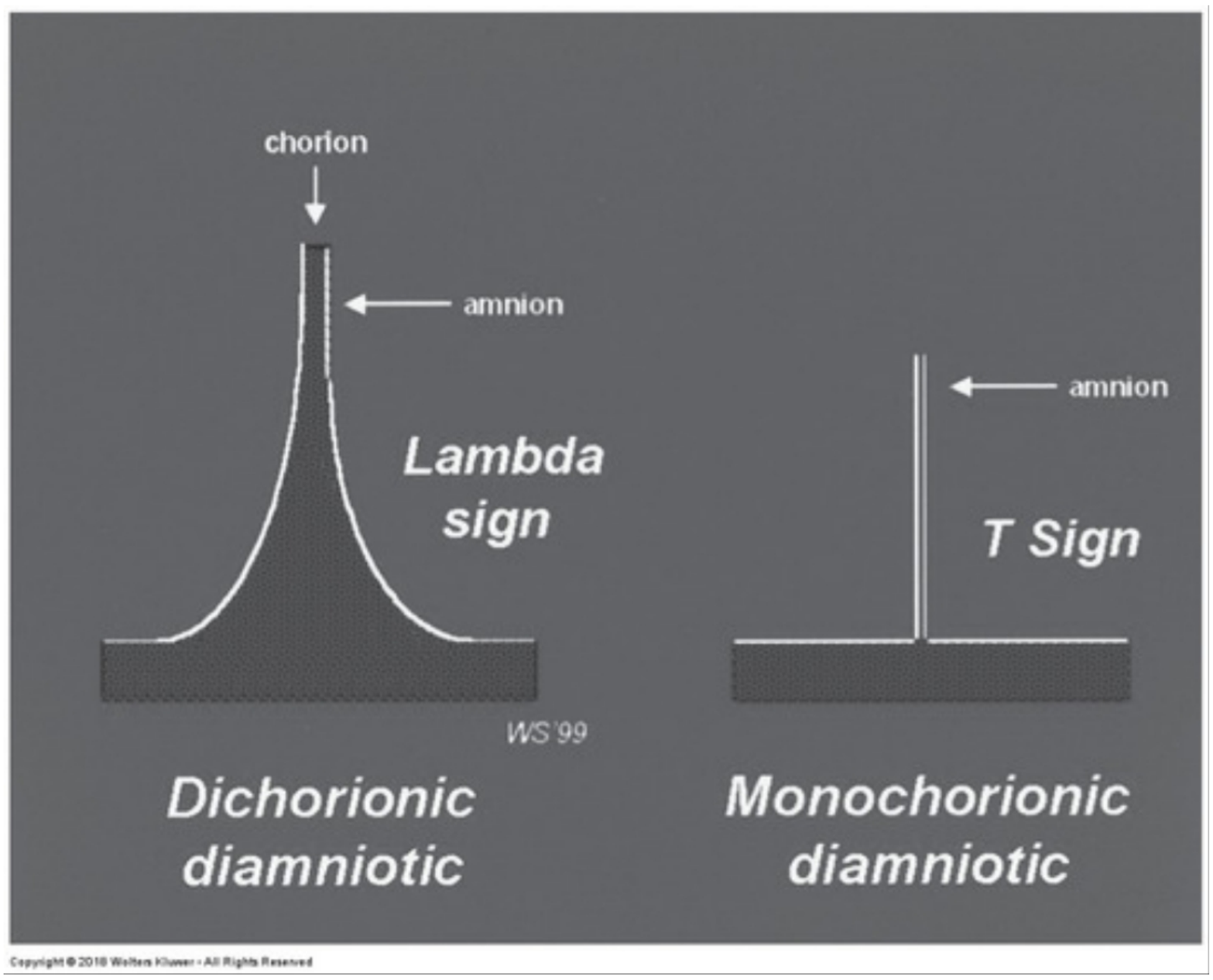
» L - Lambda sign (λ)

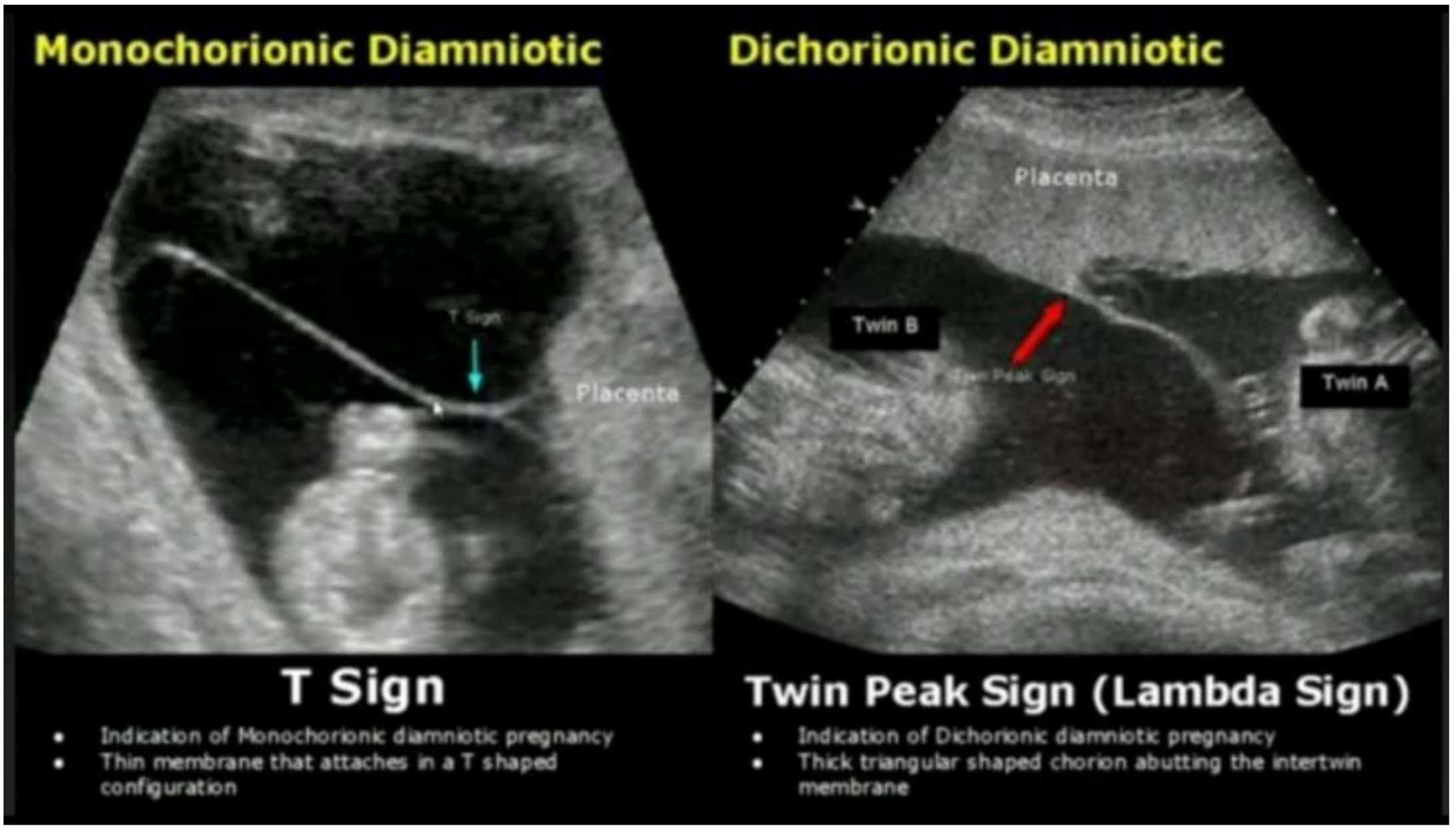
» D - Dichorionic

D - Diamniotic



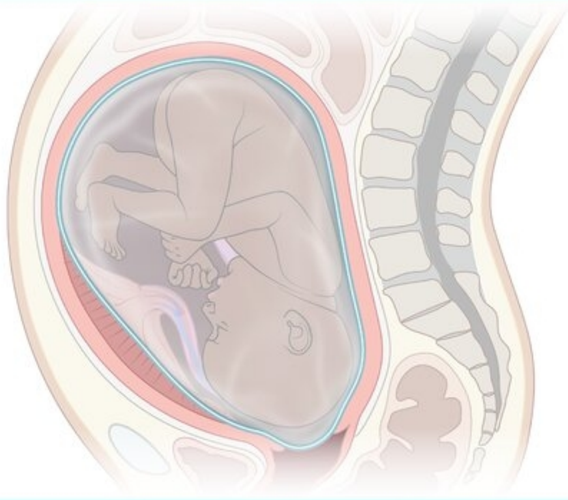
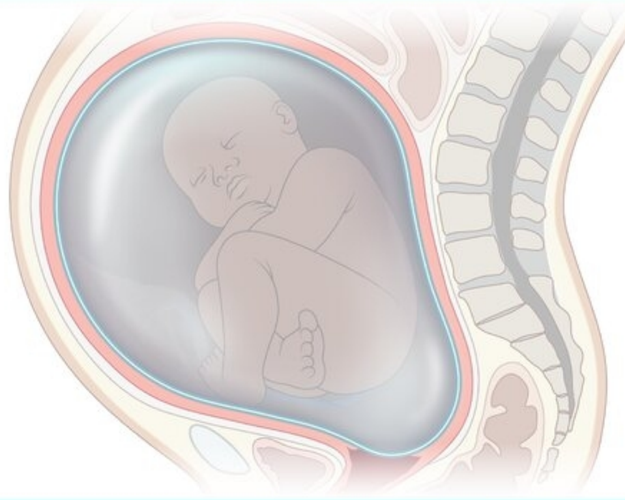


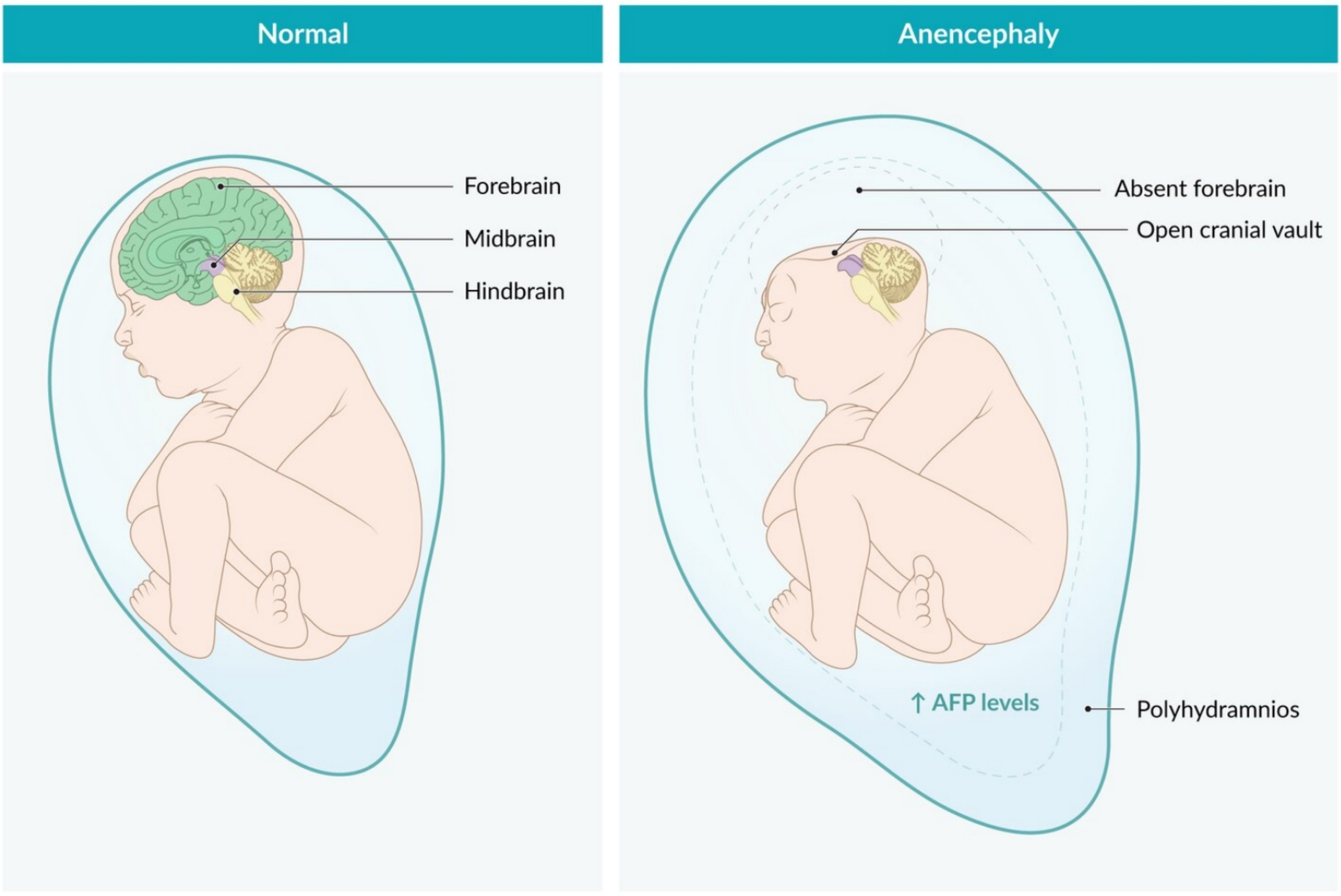




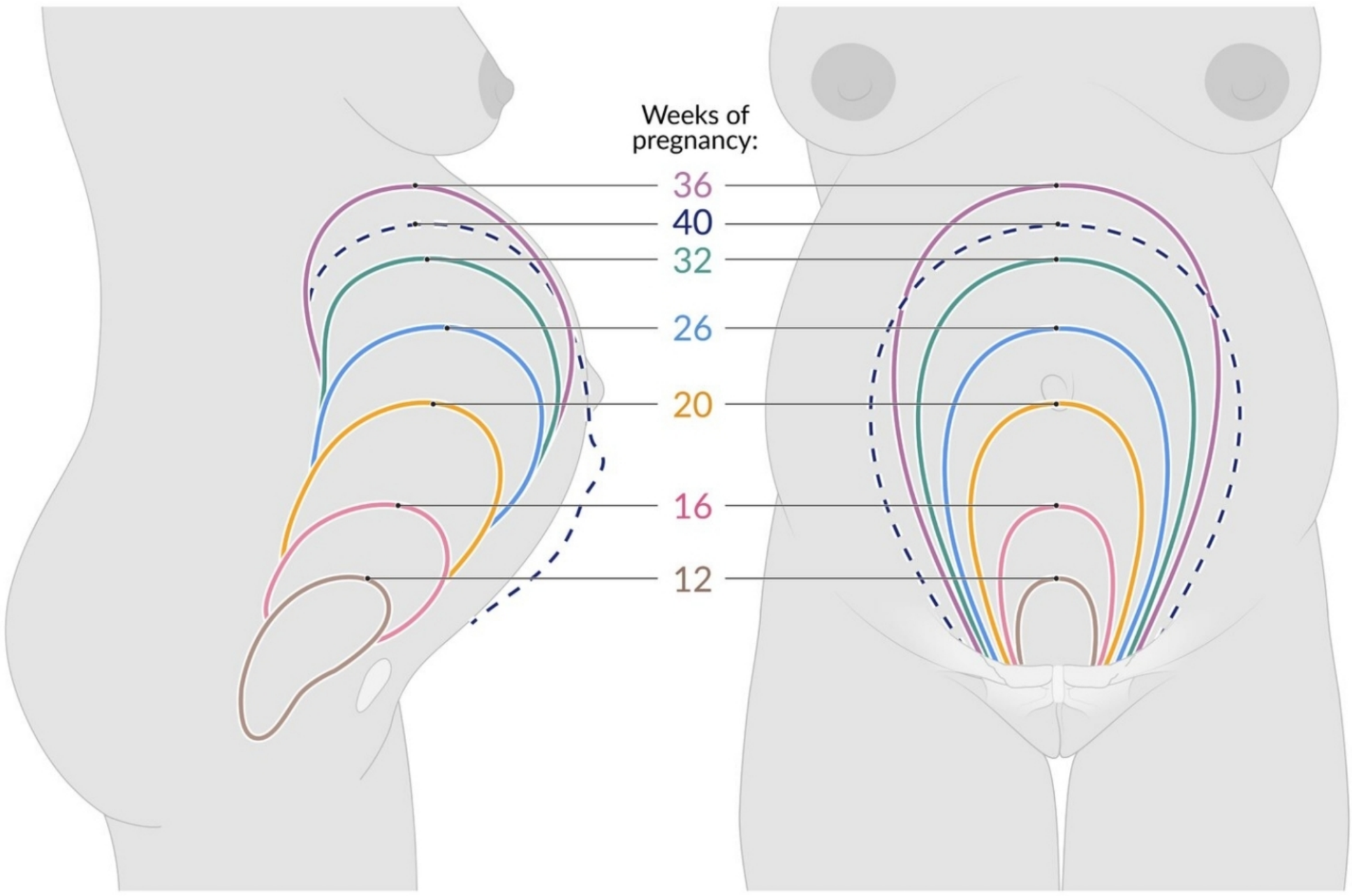
Probable signs [3][4]

Overview		
Signs	**Physical findings**	**Weeks of pregnancy**
Goodell	Cervical softening	First 4 weeks
Hegar	Softening of the lower segment of the uterus	Between 6–8 weeks
Ladin	Softening of the midline of the uterus	First 6 weeks
Chadwick	Bluish discoloration of vagina and cervix	Between 6–8 weeks
Telangiectasias and palmar erythema	Small blood vessels and redness of the palms	First 4 weeks
Chloasma	Hyperpigmentation of the face (forehead, cheeks, nose)	First 16 weeks

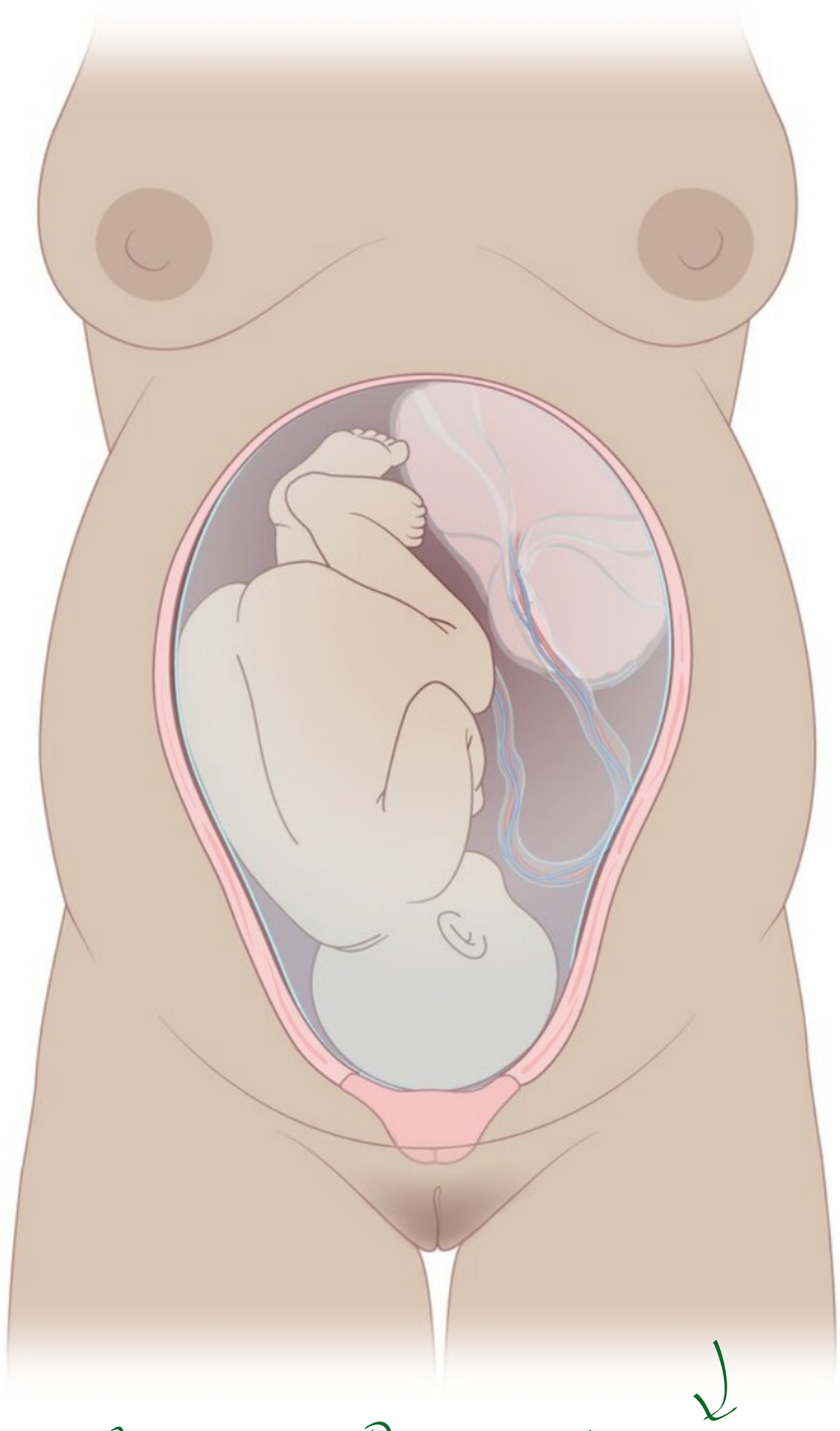
	Oligohydramnios	Polyhydramnios
		
Definition	• Amniotic fluid volume is less than expected for gestational age	• Amniotic fluid volume is more than expected for gestational age
Etiology	• Idiopathic (most common cause of mild oligohydramnios) • Fetal anomalies, e.g., urethral obstruction, bilateral renal agenesis, autosomal recessive polycystic kidney disease • Maternal conditions, e.g., placental insufficiency, late or postterm pregnancies (> 42 weeks of gestation), premature rupture of membranes	• Idiopathic (most common) • Fetal anomalies, e.g., gastrointestinal (esophageal atresia, duodenal atresia and stenosis), CNS (anencephaly, meningomyelocele), pulmonary (cystic lung malformations), twin-to-twin transfusion syndrome, fetal anemia • Maternal conditions, e.g., diabetes mellitus, Rh incompatibility
Diagnosis	• Decreased fundal height • Ultrasound: amniotic fluid index < 5 cm, fetal anomalies	• Increased fundal height • Ultrasound: amniotic fluid index ≥ 25 cm, fetal anomalies
Treatment	• Amniotransfusion • Treat underlying cause	• Amnioreduction • Treat underlying cause
Complications	• Intrauterine growth restriction • Birth complications • Potter sequence: pulmonary hypoplasia, craniofacial abnormalities, wrinkling of the skin, limb anomalies	• Fetal malposition • Umbilical cord prolapse • Premature birth


Normal
Forebrain
Midbrain
Hindbrain
Anencephaly
Absent forebrain
Open cranial vault
↑ AFP levels
Polyhydramnios

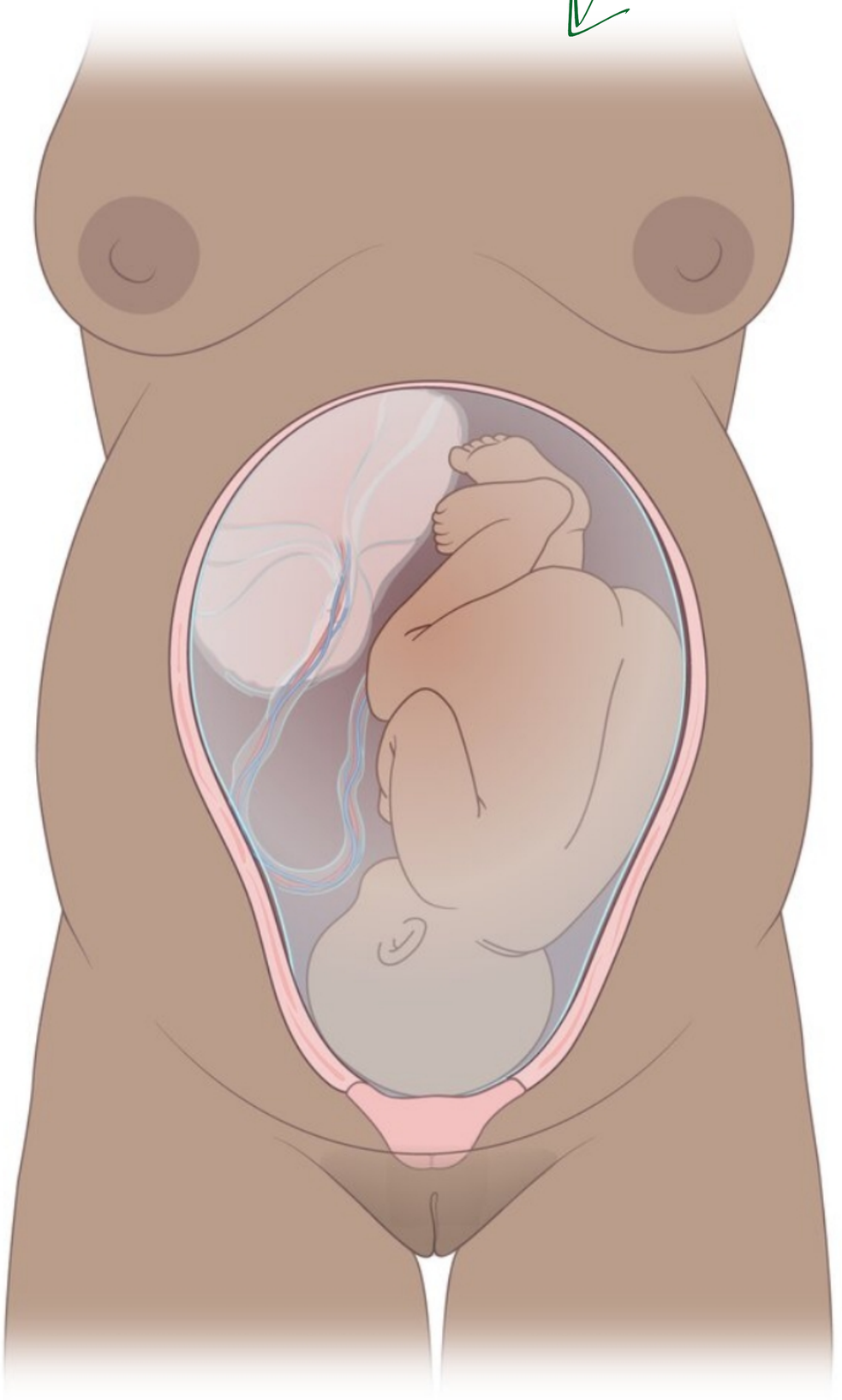
Fundal height and gestational age [11][13]	
Week of pregnancy	**Fundal height during pregnancy**
12th	Just above the symphysis
16th	Between the symphysis and navel
20– 24th	Navel
32nd	Between the navel and xiphoid
36th	Peak: at the costal arch
40th	Two finger widths below the costal arch



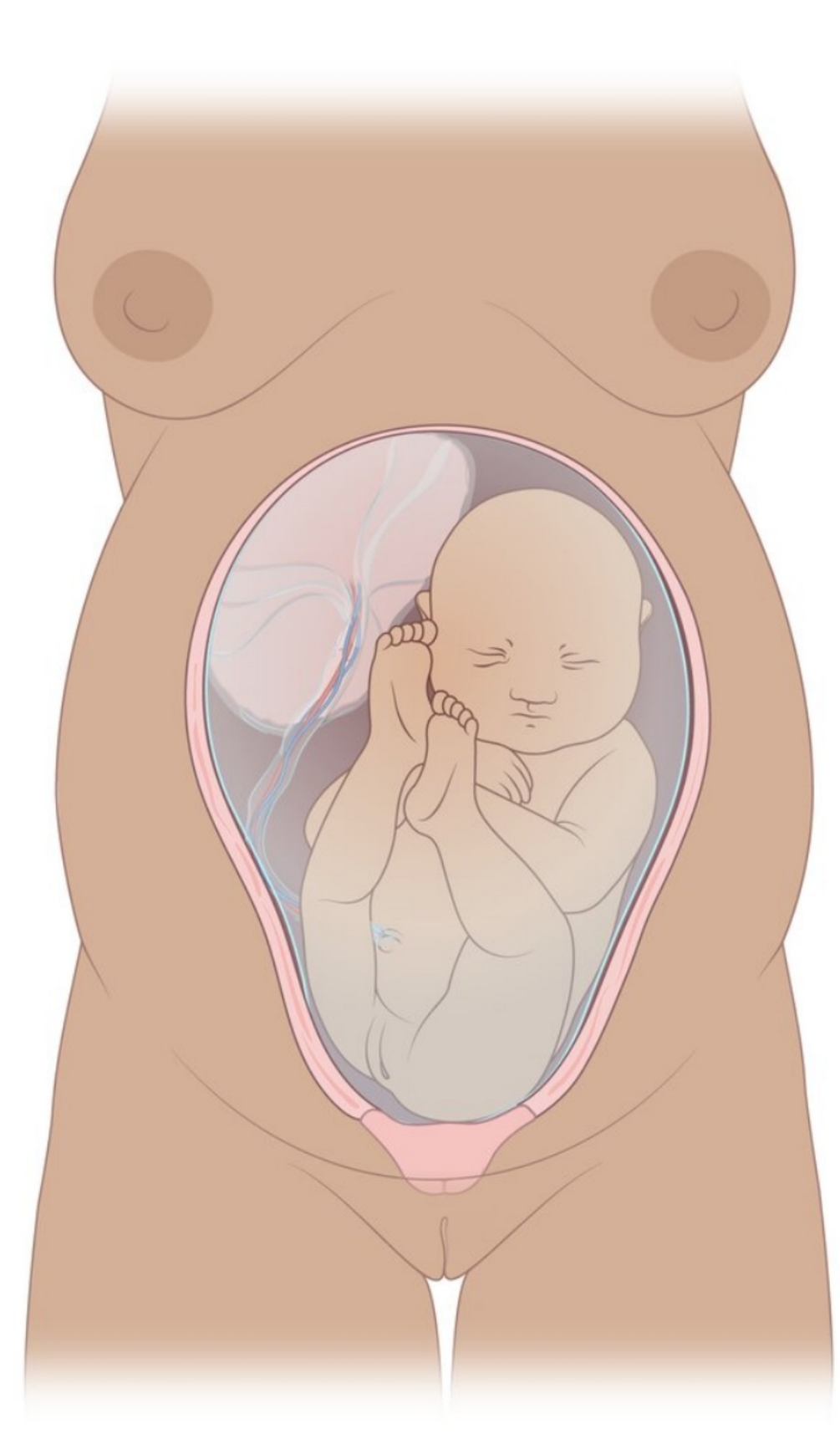
Transverse fetal lie



Cephalic Presentation
(Right occiput anterior)
Child's back faces towards
mother's right



Cephalic Presentation
(Left occiput anterior)



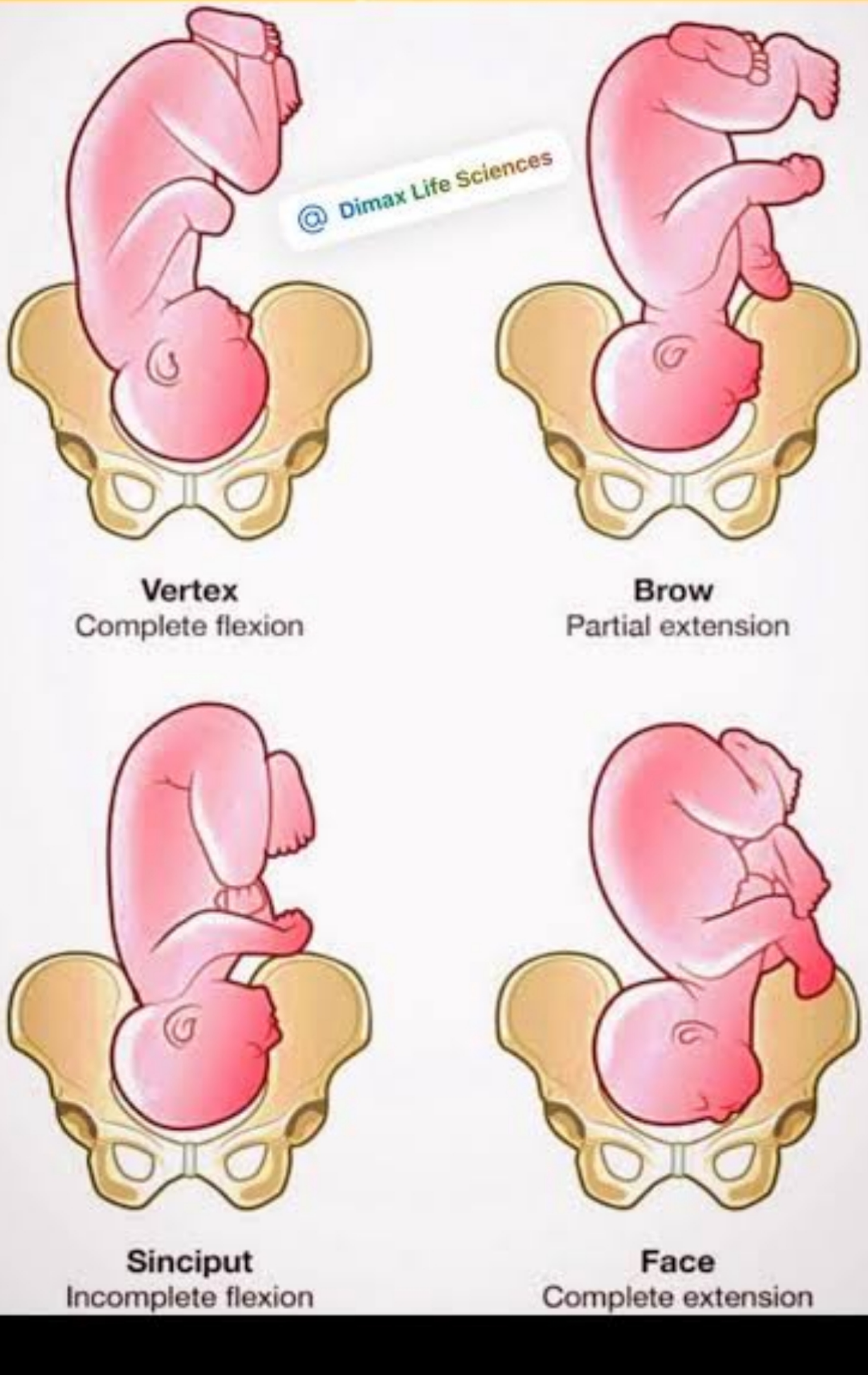
Frank Breech
(Flexed hips and extended knees)

Single Footling Breech
(one foot/leg is stretched to be delivered first)

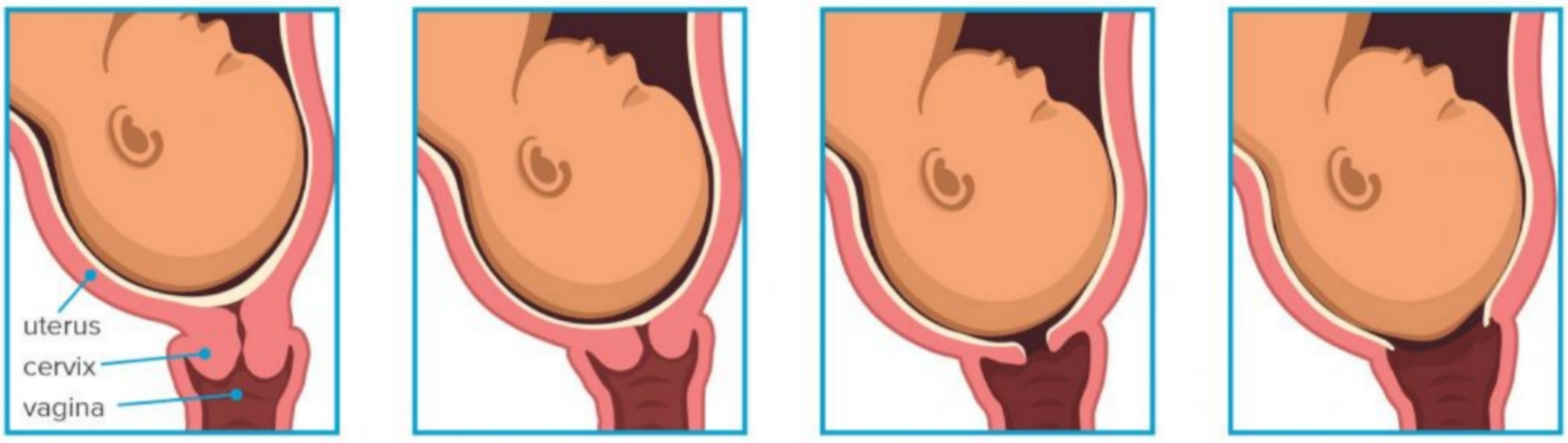
Comple Breech
(Both hips and knees are flexed with feet close to the buttocks)

Double Footling Breech
(both feet/legs are stretched to be delivered first)

Fetal attitude in cephalic presentation



Cervical Effacement

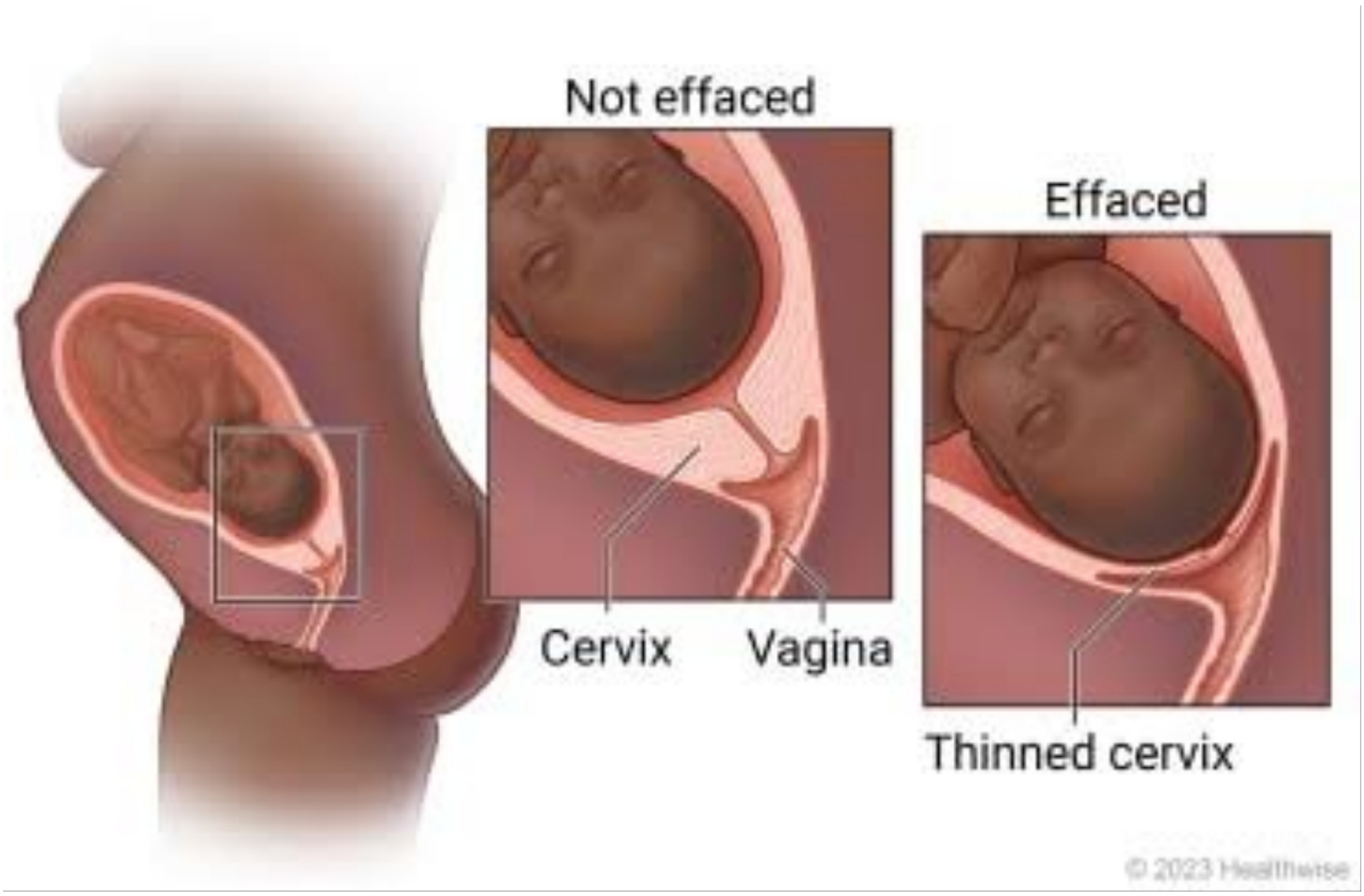


Cervix is not effaced or dilated

Cervix is 50% effaced and not dilated

Cervix is 100% effaced and dilated to 3 cm

Cervix is fully dilated to 10 cm

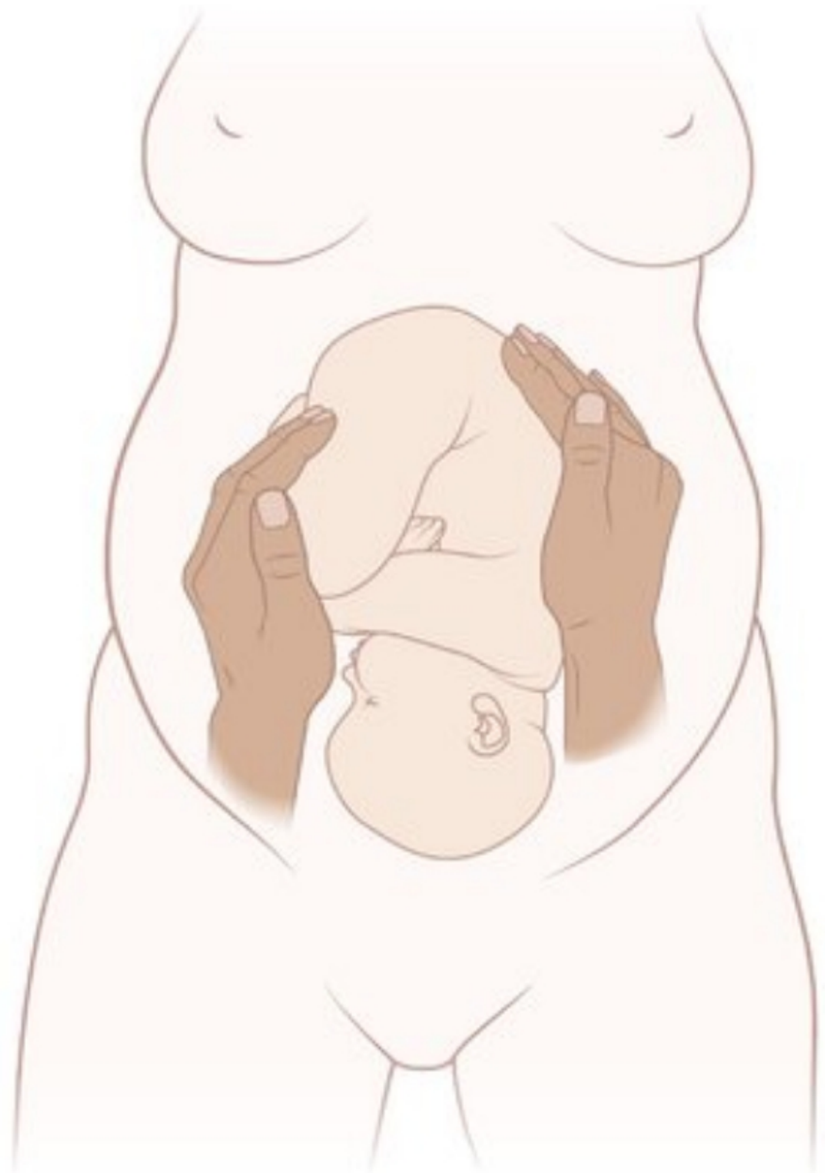


Leopold maneuvers [22][85]

- The Leopold maneuvers consist of four abdominal palpation maneuvers used to determine fetal lie, fetal presentation, and fetal position in utero.
  1. Use both hands to palpate the uterine fundus, fetal head, and buttocks to assess:
     - Fetal lie (longitudinal/oblique/transverse)
     - Fundal height
  2. Place each hand on either side of the maternal abdomen to determine the location of the fetal back.
  3. Grasp the lower maternal abdomen above the symphysis to determine the fetal presenting part and if it is engaged.
     - In cephalic presentation, the fetal head is felt as hard, round, and ballottable.
     - In breech presentation, the buttocks are felt as a soft, less movable structure.
  4. Facing the mother's feet, use both hands to determine:
     - The cephalic prominence
     - Fetal attitude (based on the degree of flexion of the fetus's head)
- If abnormal presentation (e.g., breech) is suspected or fetal position cannot be accurately determined, proceed to ultrasound. [86][87]

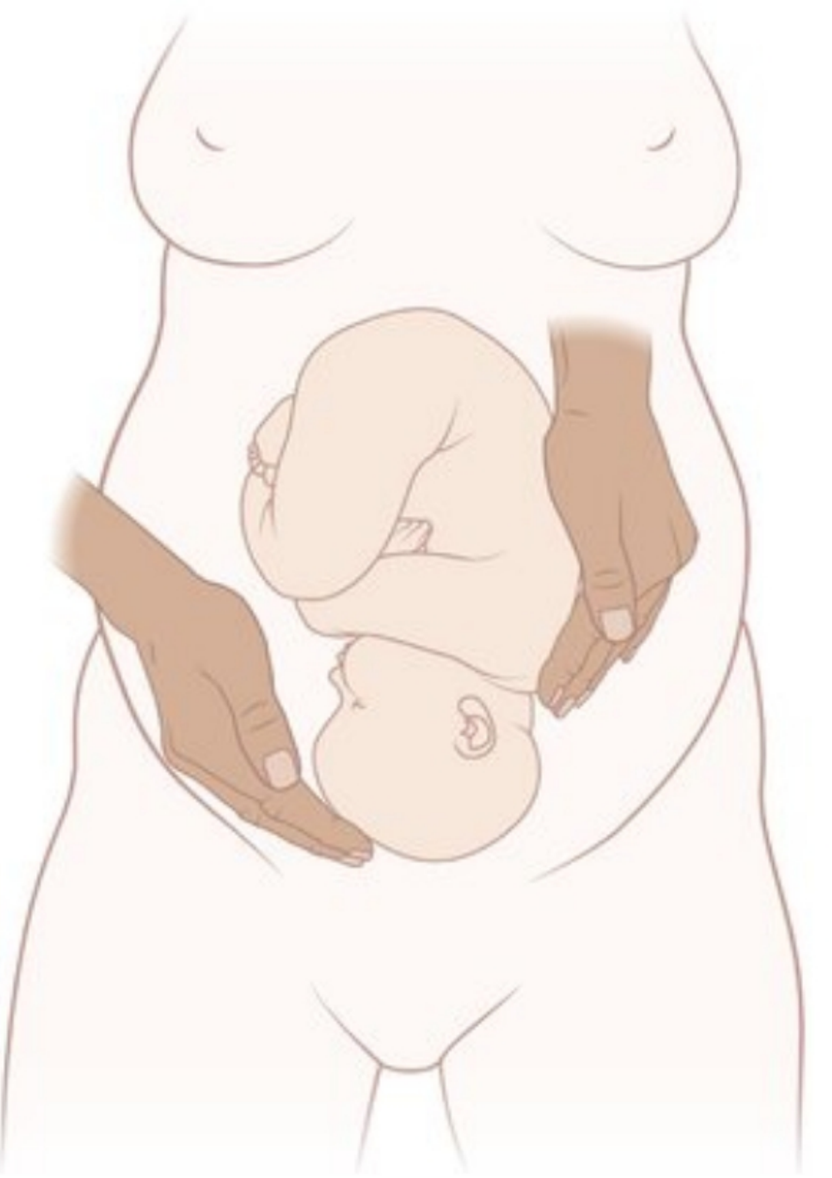
Leopold's maneuvers

First maneuver



Second maneuver

Third maneuver



Fourth maneuver

Breast Milk Advantages

B - Bonding b.w mother and baby

E - Economical

S - Safe / sterile

T - Temperature Ideal

M - Maternal health benefits
(Reduce breast and ovarian cancer,
postpartum weight loss,
natural contraception)

I - Immunity booster
Rich in antibodies esp
colostrum

L - Low Risk of chronic disease
in baby
Reduce risk of obesity, asthma,
allergies

K - Knowledge pamper
(Help in cognitive brain
development)

Disadvantages of Breast Milk

L - Latching problems (Difficulty with proper attachment or pain or poor suck feeding)

A - Availability of mother

T - Tiring for mother

C - Cultural or social stigma

H - Health Conditions

P - Pain (mastitis if)

A - Alcohol or Drug Restriction as it can pass through milk

I - Inconvenient in public

N - Nutritional deficiency in mother can affect milk

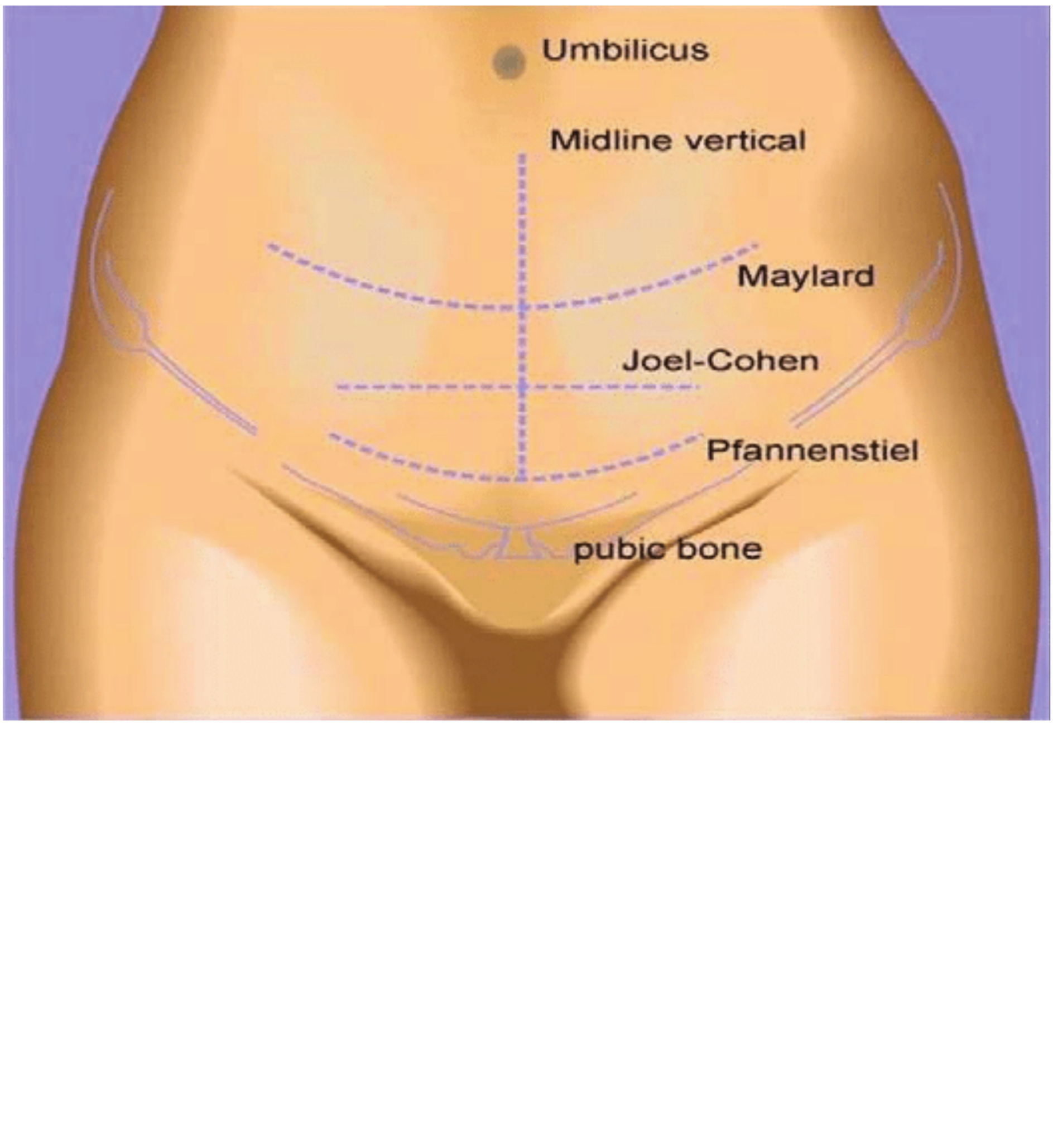
S - Supply Concerns

Bishop score

Bishop score [32]				
	Score			
	0 points	1 point	2 points	3 points
Cervical position	Posterior	Midline	Anterior	
Cervical consistency	Firm	Moderately firm	Soft (ripe)	
Cervical effacement	≤ 30%	31–50%	51–80%	> 80%
Cervical dilation	Closed	1–2 cm	3–4 cm	≥ 5 cm
Fetal station	-3 cm	-2 cm	-1/0 cm	+1/+2 cm

Simpson forceps	Kielland forceps	Piper forceps	Barton forceps
			


TYPES OF CESAREAN INCISION
Midline
Joel-Cohen
Pfannenstiel
shutterstock.com · 2625600771

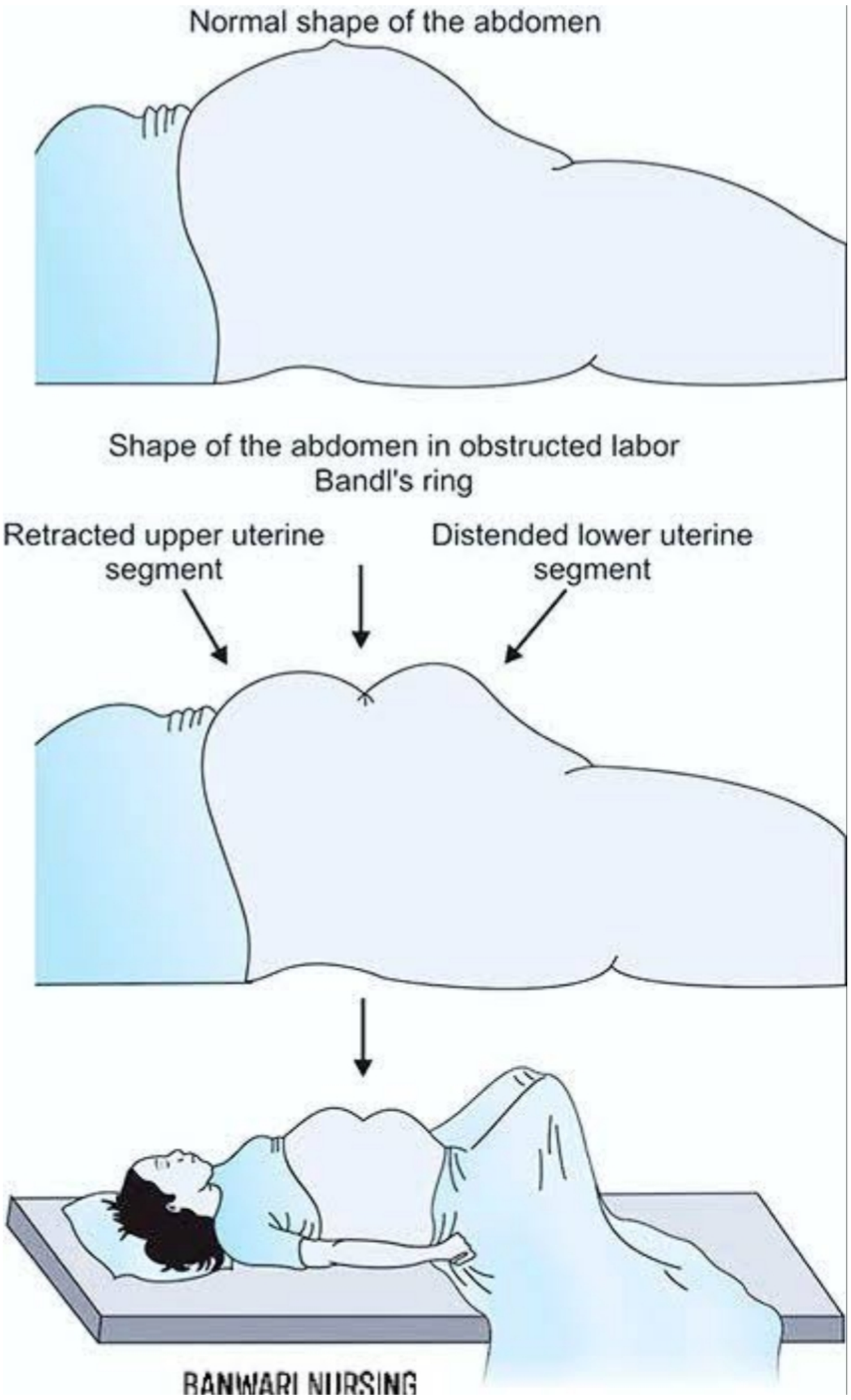

Umbilicus
Midline vertical
Maylard
Joel-Cohen
Pfannenstiel
pubic bone

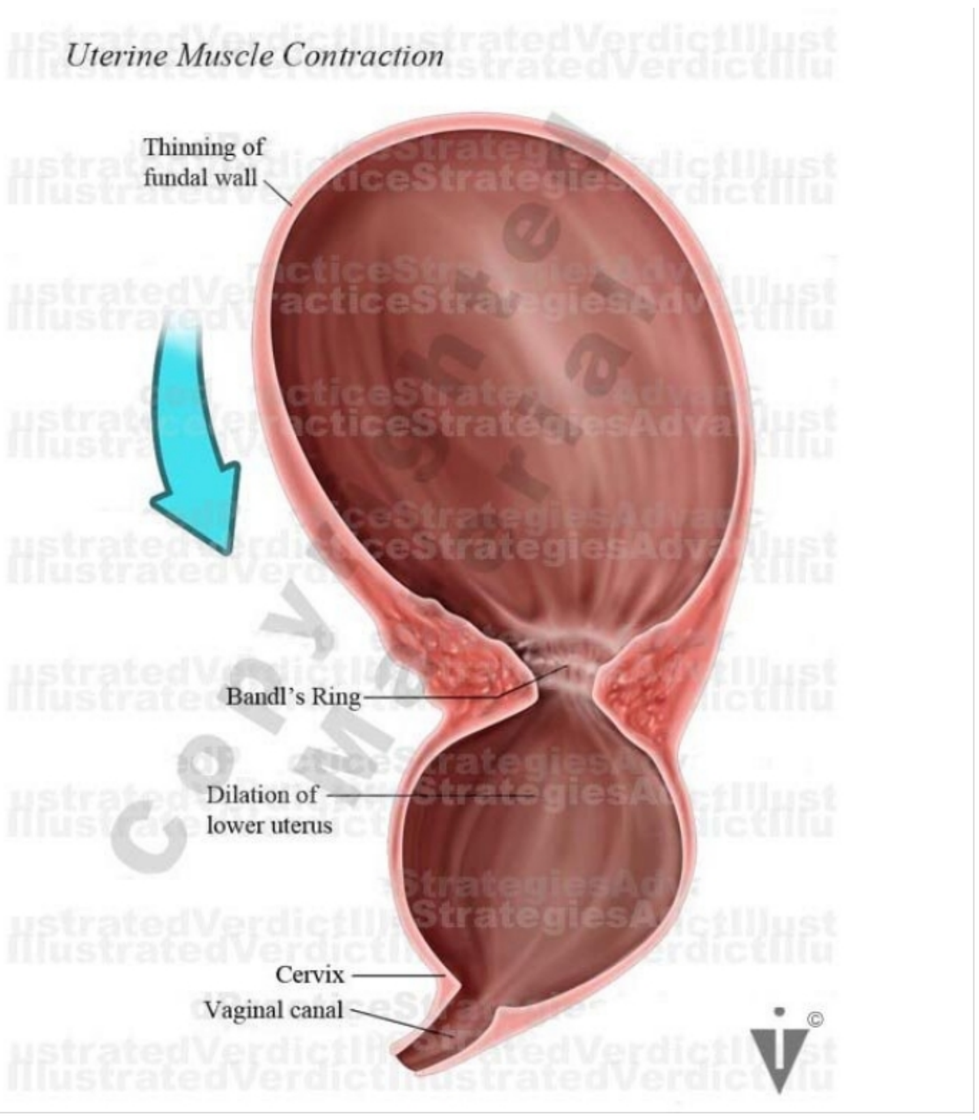
Apgar Scoring System

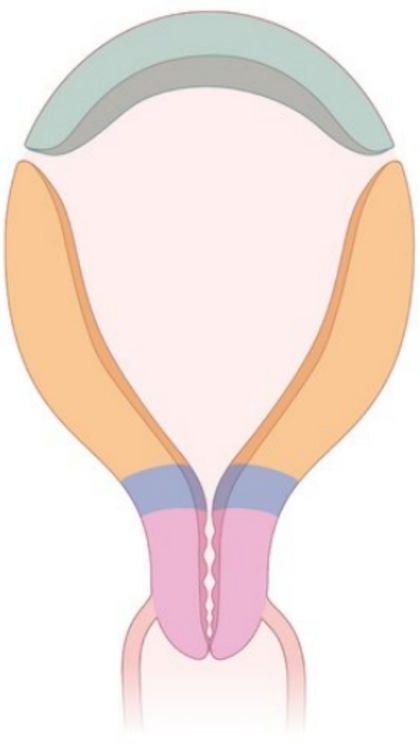
Indicator		0 Points	1 Point	2 Points
A	Activity (muscle tone)	Absent	Flexed limbs	Active
P	Pulse	Absent	< 100 BPM	> 100 BPM
G	Grimace (reflex irritability)	Floppy	Minimal response to stimulation	Prompt response to stimulation
A	Appearance (skin color)	Blue Pale	Pink body Blue extremeties	Pink
R	Respiration	Absent	Slow and irregular	Vigorous cry

HIE Help Center
hiehelpcenter.org

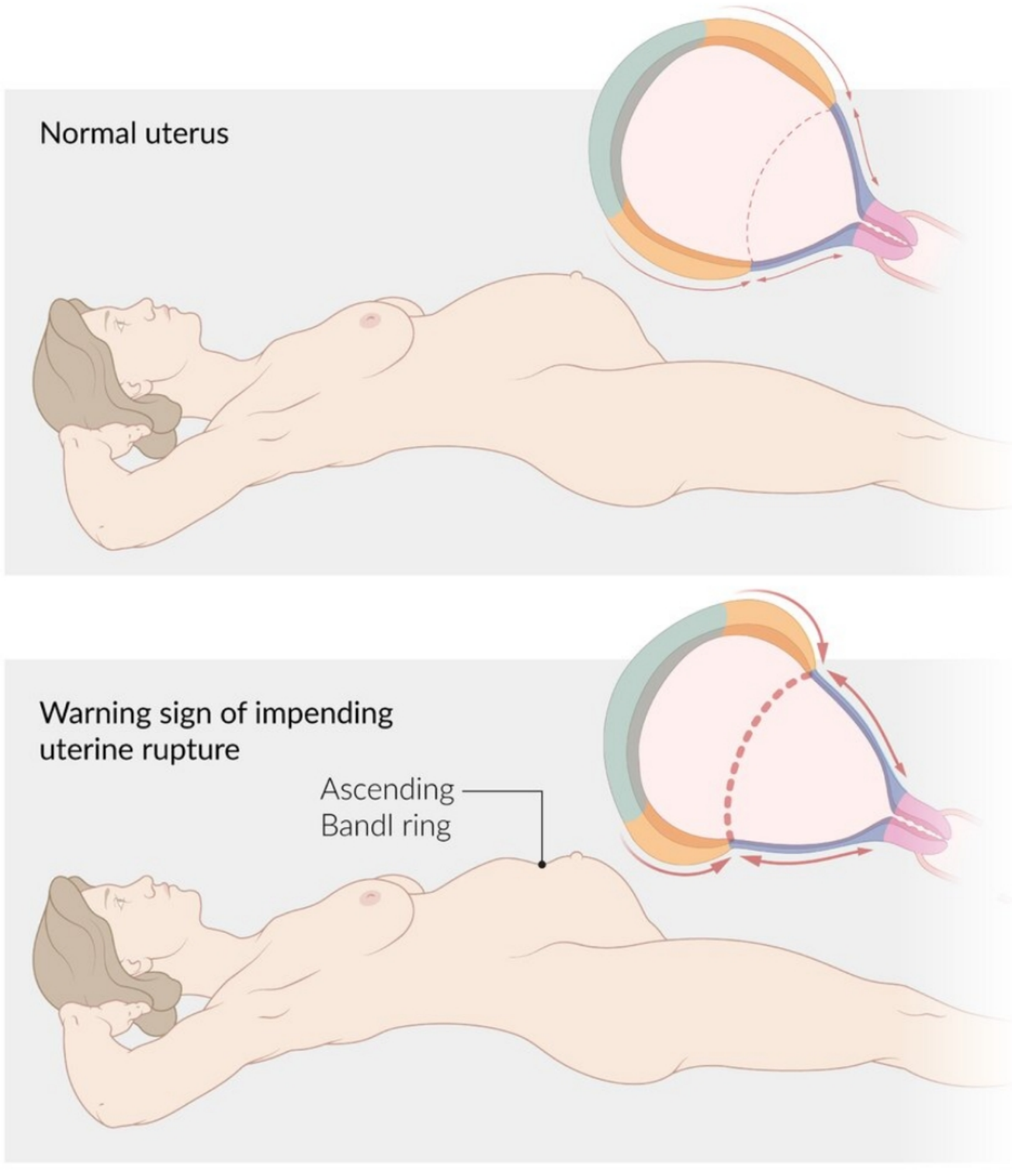
Bandl's Ring


Normal shape of the abdomen
Shape of the abdomen in obstructed labor
Bandl's ring
Retracted upper uterine segment
Distended lower uterine segment
BANWARI NURSING

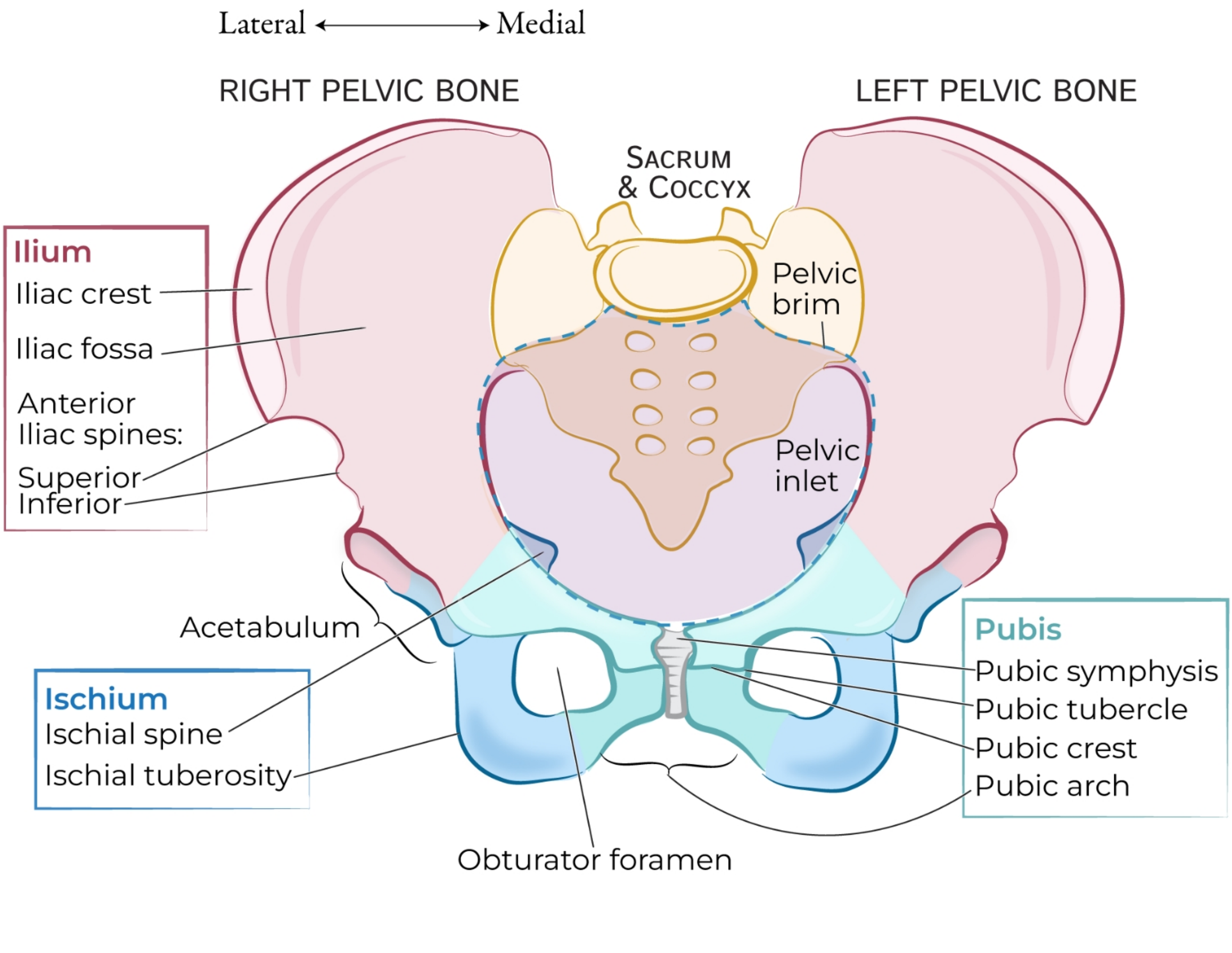

Uterine Muscle Contraction
Thinning of fundal wall
Bandl's Ring
Dilation of lower uterus
Cervix
Vaginal canal



Fundus
Body
Isthmus
Cervix



Pelvic Girdle: Anterior



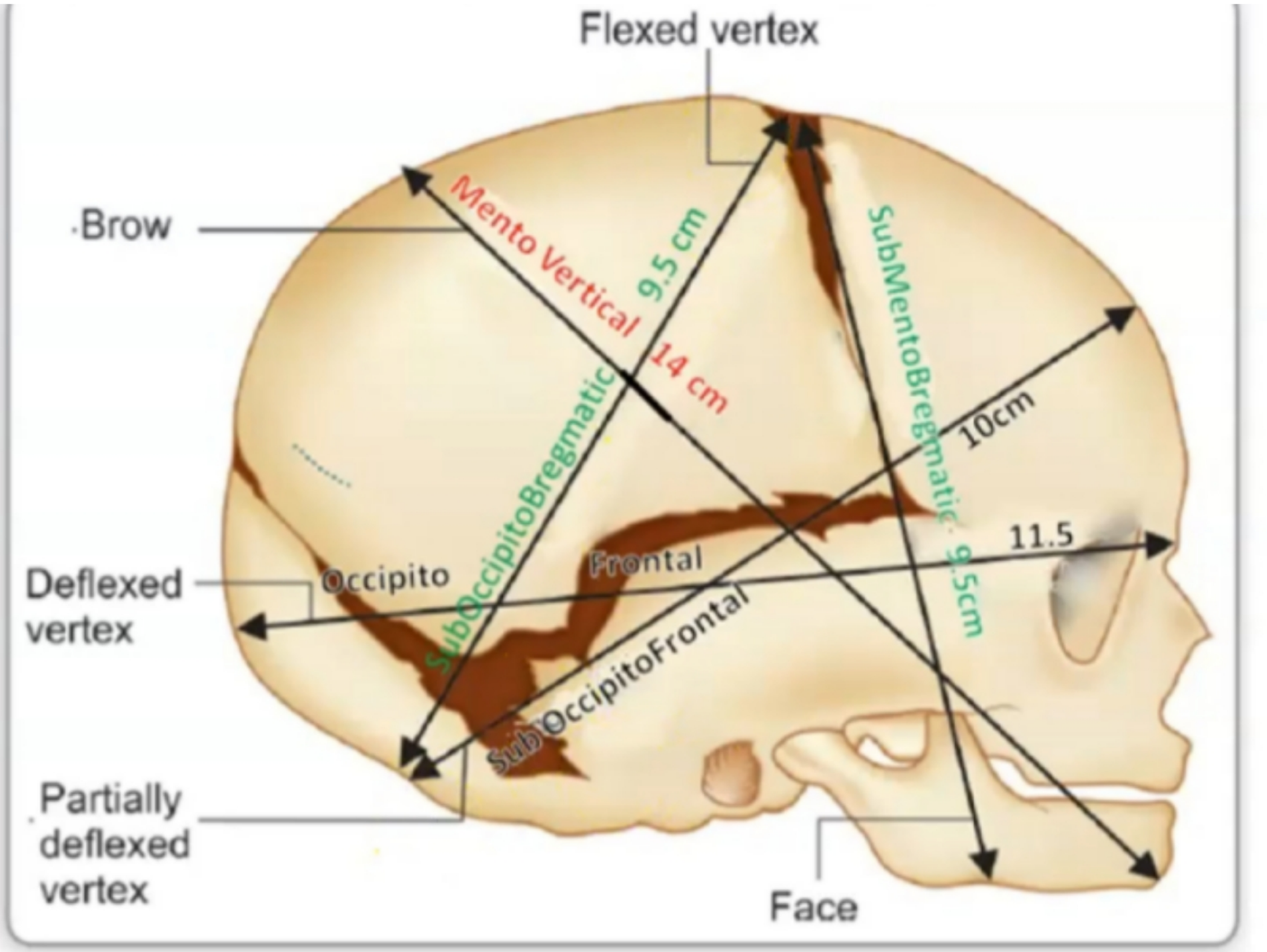
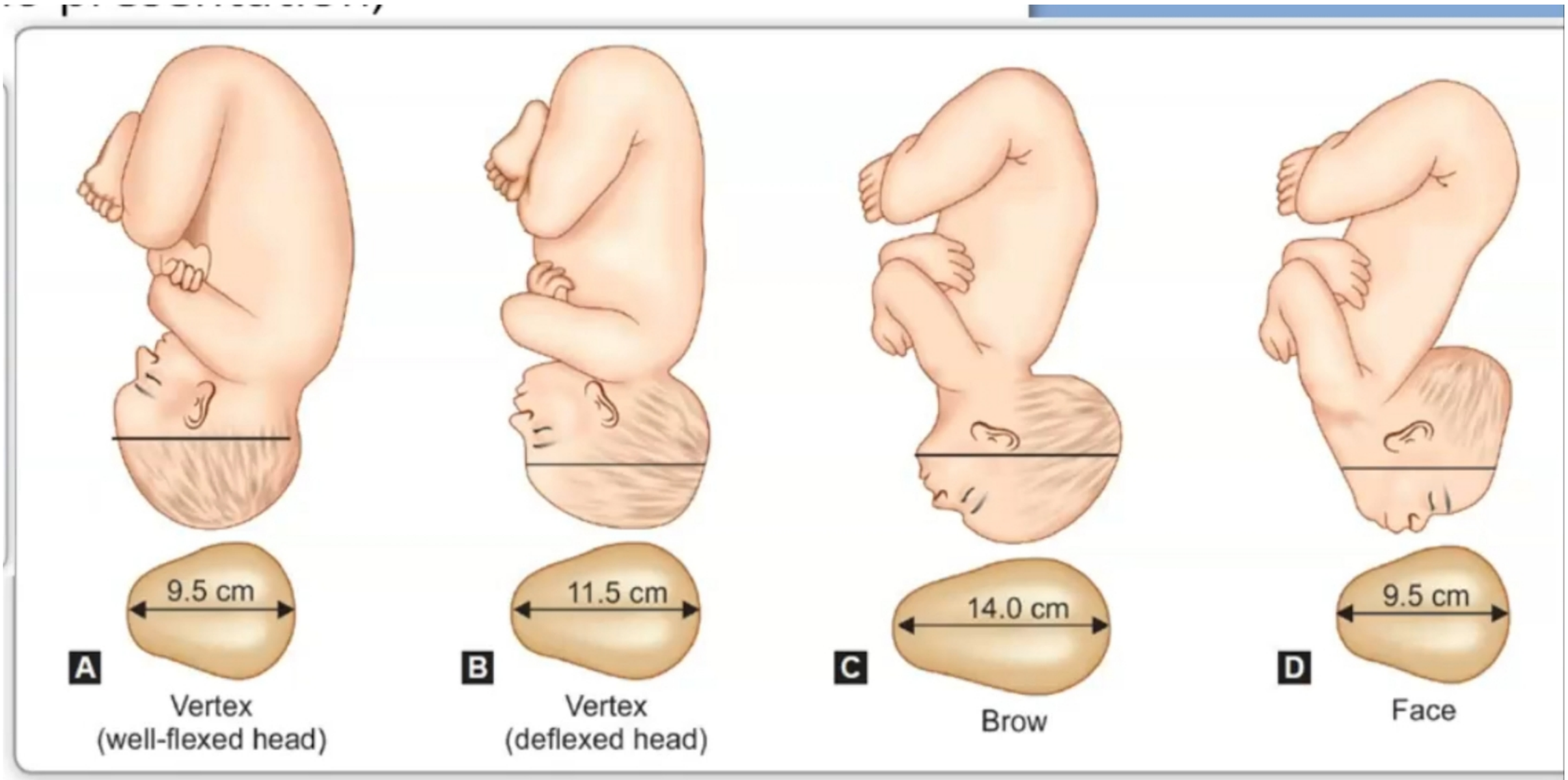
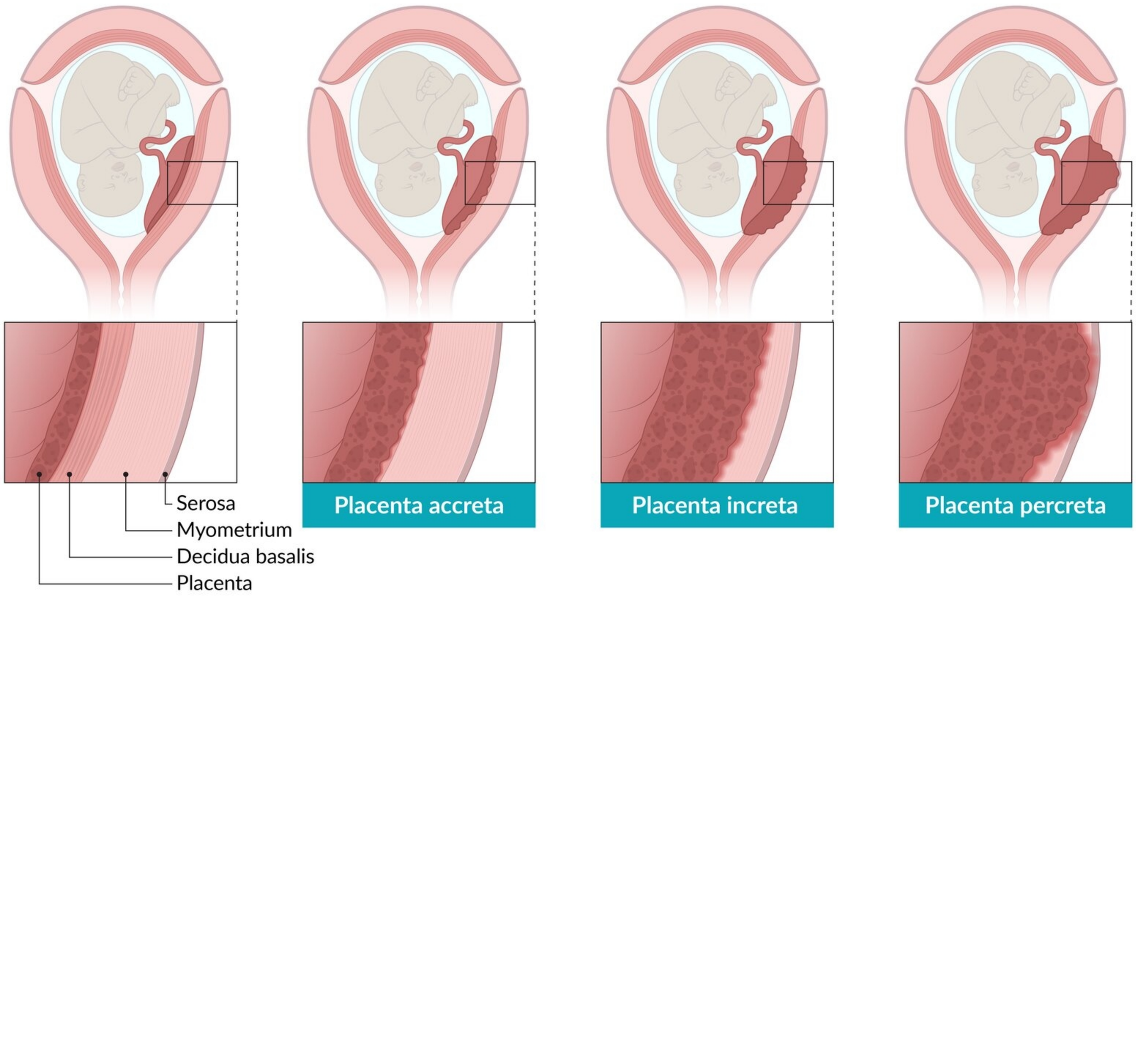


Fig. 9.3: The important landmarks of fetal skull



Figs 8.2A to D: Varieties of cephalic presentations in different attitude


Serosa
Myometrium
Decidua basalis
Placenta
Placenta accreta
Placenta increta
Placenta percreta

HTN

◀ High Risk Group Therapy (Special Cases):

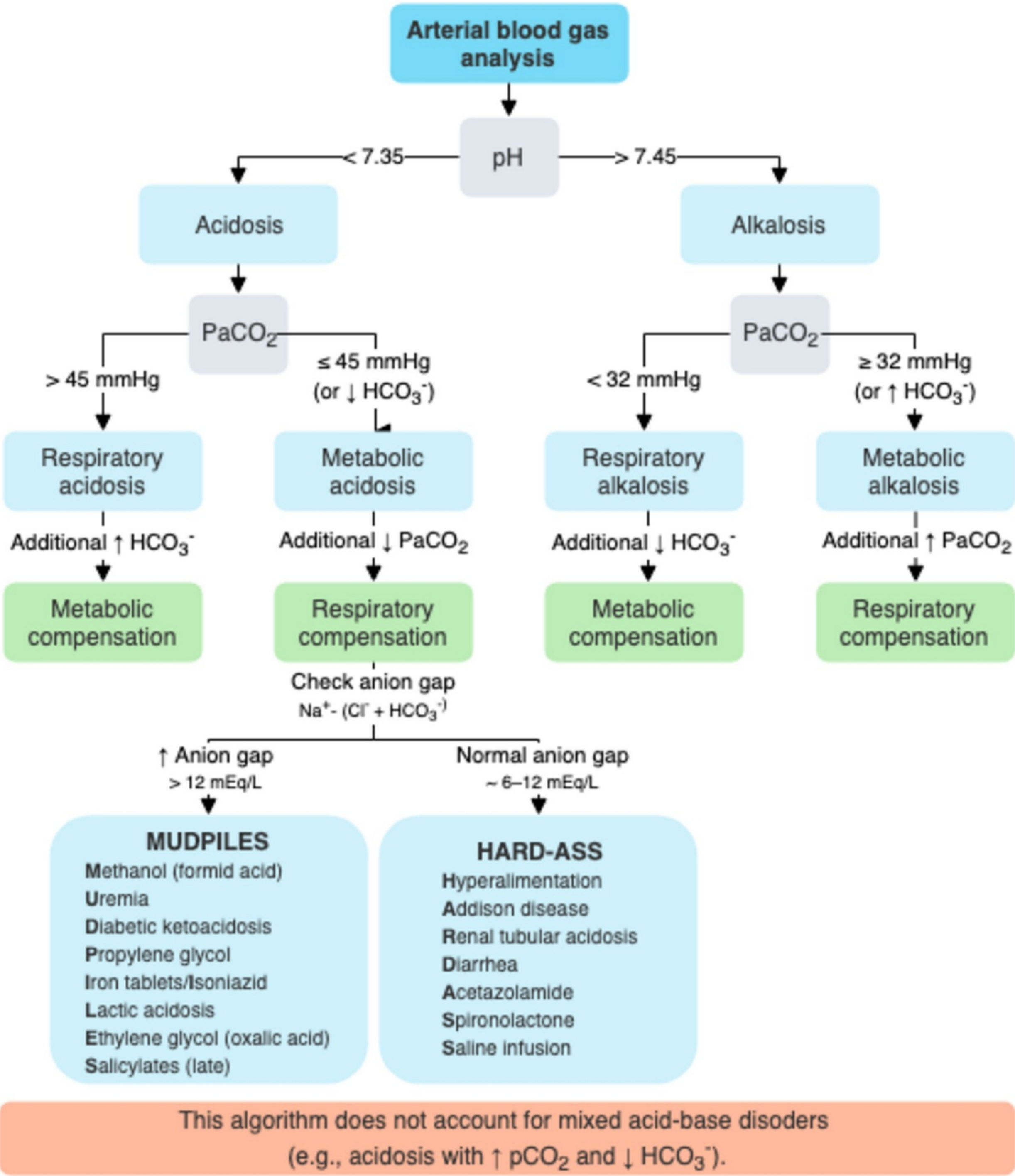
Condition	Preferred therapy
Congestive heart failure	Thiazide, ACEI, Aldosterone antagonist, BB
Post Myocardial Infarction	BB, ACEi
Diabetes mellitus with proteinuria	ACEi, ARBs, NO
Diabetes mellitus without Proteinuria	Thiazide, CCB, ARBs, ACEi
Chronic kidney disease	ACEi, ARB, Thiazide
Stroke	CCB+ACEi
Benign prostatic hyperplasia	α antagonists e.g. prazosin, terazosin, doxazosin
Pregnancy	Hydralazine (vasodilator), nifedipine (CCB), Aldomet (methyldopa), labetalol **Oral labetalol** is first-line therapy during pregnancy. Second Line agents are methyldopa and nifedipine. **In breastfeeding,** ACE inhibitors, beta-blockers and nifedipine are safe. Methyldopa should be avoided because of the risk of depression.

◀ Antihypertensive drug complications: 

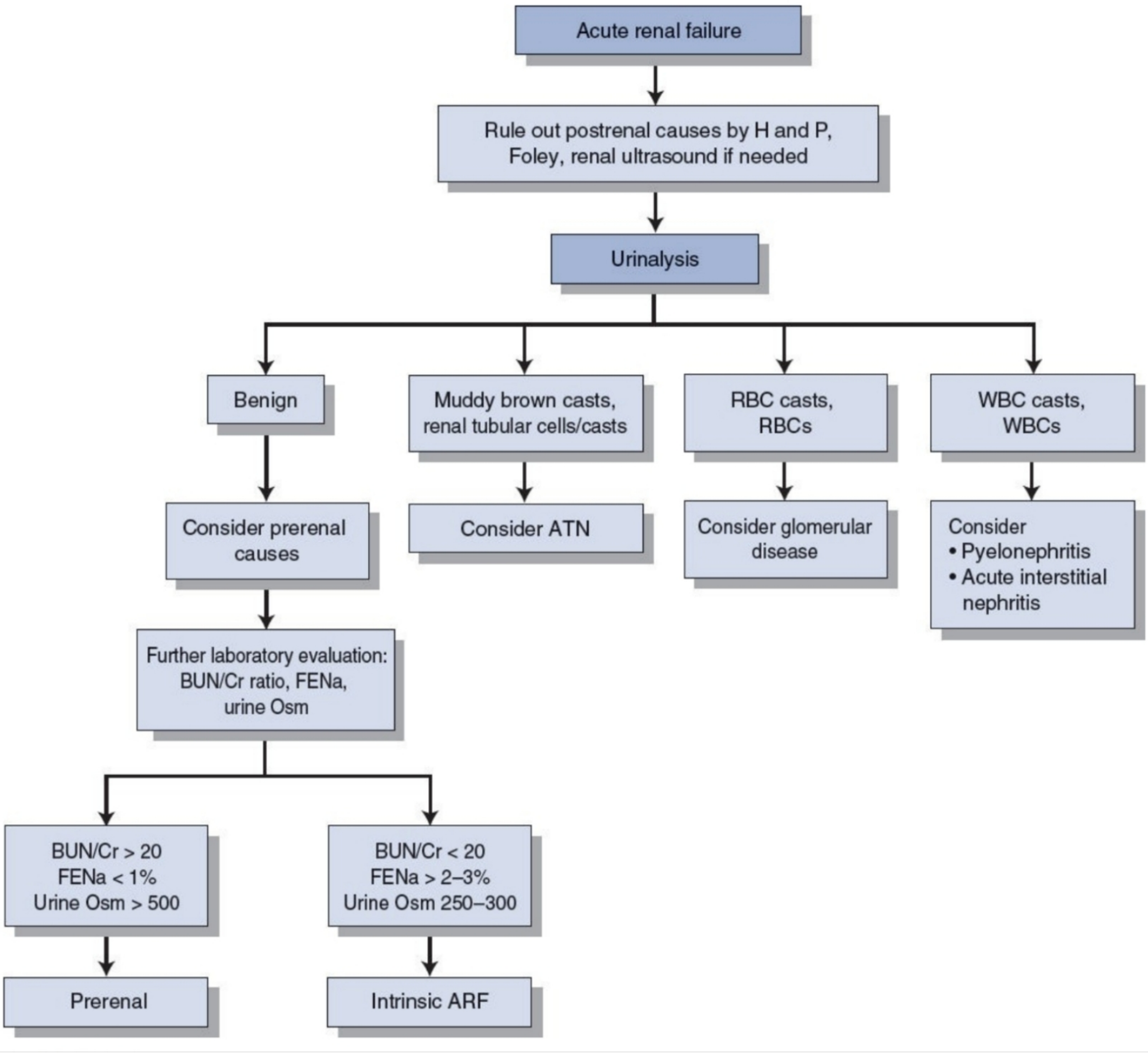
DRUG	Contraindication	ADR
Thiazide Diuretics	Gout	Hypokalemia
Beta-Adrenergic blockers	Asthma or COPD	Bradycardia
ACEI	Pregnancy, Renovascular disease	Hyperkalemia, Cough, Angioedema
Angiotensin II Receptor Blocker	Pregnancy	Hyperkalemia
Calcium Channel Blockers	AV-block, HF	Edema, Tachycardia, Bradycardia, Constipation, flushing, palpitations and fluid retention
Alpha-Adrenoceptor Antagonist	Urinary incontinence	1st dose postural hypotension,
Drugs with Central Sympatholytic Action	-	Drowsiness
Arteriolar Dilators	-	Tachycardia, Edema

➢ Primary disturbance:

Primary disorder	Respiratory acidosis	Respiratory alkalosis	Metabolic acidosis	Metabolic alkalosis
Problem	Hypoventilation	Hyperventilation	Gain of H^+ or loss of HCO_3^-	Gain of HCO_3^- or loss of H^+
pH	↓	↑	↓	↑
HCO_3-	↑	↓	↓↓	↑↑
$PaCO_2$[1]	↑↑	↓↓	↓	↑


Arterial blood gas analysis
pH
< 7.35
> 7.45
Acidosis
Alkalosis
PaCO2
PaCO2
> 45 mmHg
≤ 45 mmHg (or ↓ HCO3-)
< 32 mmHg
≥ 32 mmHg (or ↑ HCO3-)
Respiratory acidosis
Metabolic acidosis
Respiratory alkalosis
Metabolic alkalosis
Additional ↑ HCO3-
Additional ↓ PaCO2
Additional ↓ HCO3-
Additional ↑ PaCO2
Metabolic compensation
Respiratory compensation
Metabolic compensation
Respiratory compensation
Check anion gap
Na+ - (Cl- + HCO3-)
↑ Anion gap
> 12 mEq/L
Normal anion gap
~ 6–12 mEq/L
MUDPILES
Methanol (formid acid)
Uremia
Diabetic ketoacidosis
Propylene glycol
Iron tablets/Isoniazid
Lactic acidosis
Ethylene glycol (oxalic acid)
Salicylates (late)
HARD-ASS
Hyperalimentation
Addison disease
Renal tubular acidosis
Diarrhea
Acetazolamide
Spironolactone
Saline infusion
This algorithm does not account for mixed acid-base disoders
(e.g., acidosis with ↑ pCO2 and ↓ HCO3-).

Determination of the likely mechanism of acute kidney injury			
	Prerenal	**Intrinsic**	**Postrenal**
BUN:creatinine ratio	• > 20:1	• < 15:1	• Varies
FENa	• < 1%	• > 2–3%	
FEUrea	• < 35%	• > 50%	
Urine sodium concentration	• < 20 mEq/L	• > 40 mEq/L	
Urine osmolality	• > 500 mOsm/kg	• < 350 mOsm/kg	• < 350 mOsm/kg
Urine sediment	• Hyaline casts	• Renal tubular epithelial cells or granular, muddy brown, or pigmented casts (e.g., due to ATN) • RBC casts (e.g., due to glomerulonephritis) • Fatty casts (e.g., due to nephrotic syndrome) • WBC casts (e.g., due to allergic interstitial nephritis)	• Hematuria (stones, bladder cancer, clots) • Absent (neurogenic bladder)


Acute renal failure
Rule out postrenal causes by H and P, Foley, renal ultrasound if needed
Urinalysis
Benign
Muddy brown casts, renal tubular cells/casts
RBC casts, RBCs
WBC casts, WBCs
Consider prerenal causes
Consider ATN
Consider glomerular disease
Consider
• Pyelonephritis
• Acute interstitial nephritis
Further laboratory evaluation: BUN/Cr ratio, FENa, urine Osm
BUN/Cr > 20
FENa < 1%
Urine Osm > 500
BUN/Cr < 20
FENa > 2–3%
Urine Osm 250–300
Prerenal
Intrinsic ARF

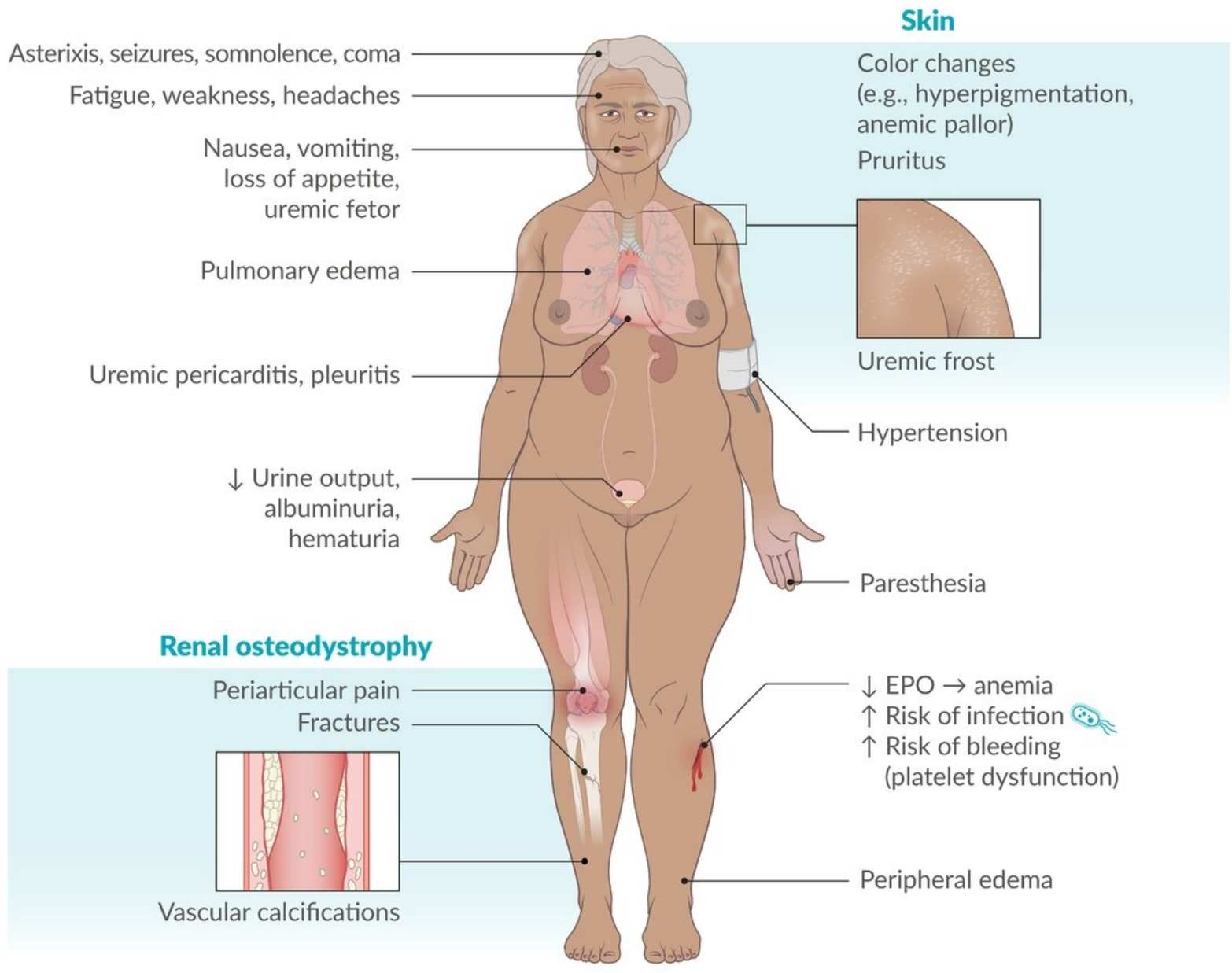
Chronic kidney disease

Definition
Abnormality of kidney structure or function
> 3 months as eGFR < 60 mL/min/1.73 m^2

Etiology
- Diabetic nephropathy
- Hypertensive nephropathy
- Glomerulonephritis
- Idiopathic

Risk factors
- Diabetes, hypertension, obesity
- Age > 60 years of age
- Recreational drug use
- AKI
- Family history of CKD
- African American race or Hispanic ethnicity

Laboratory findings
- ↑ Creatinine and BUN, ↓ eGFR
- Hyperkalemia
- Metabolic acidosis
- ↓ Hb, normal MCV



Uremic frost - Scalp and forehead of a patient with a markedly elevated blood urea nitrogen level (approx. 100 mg/dL) The widely disseminated tiny white spots are crystallized urea resulting from the evaporation of sweat that contains high amounts of urea. This finding is a sign of severe azotemia, which is rare in settings where renal replacement therapy is available.

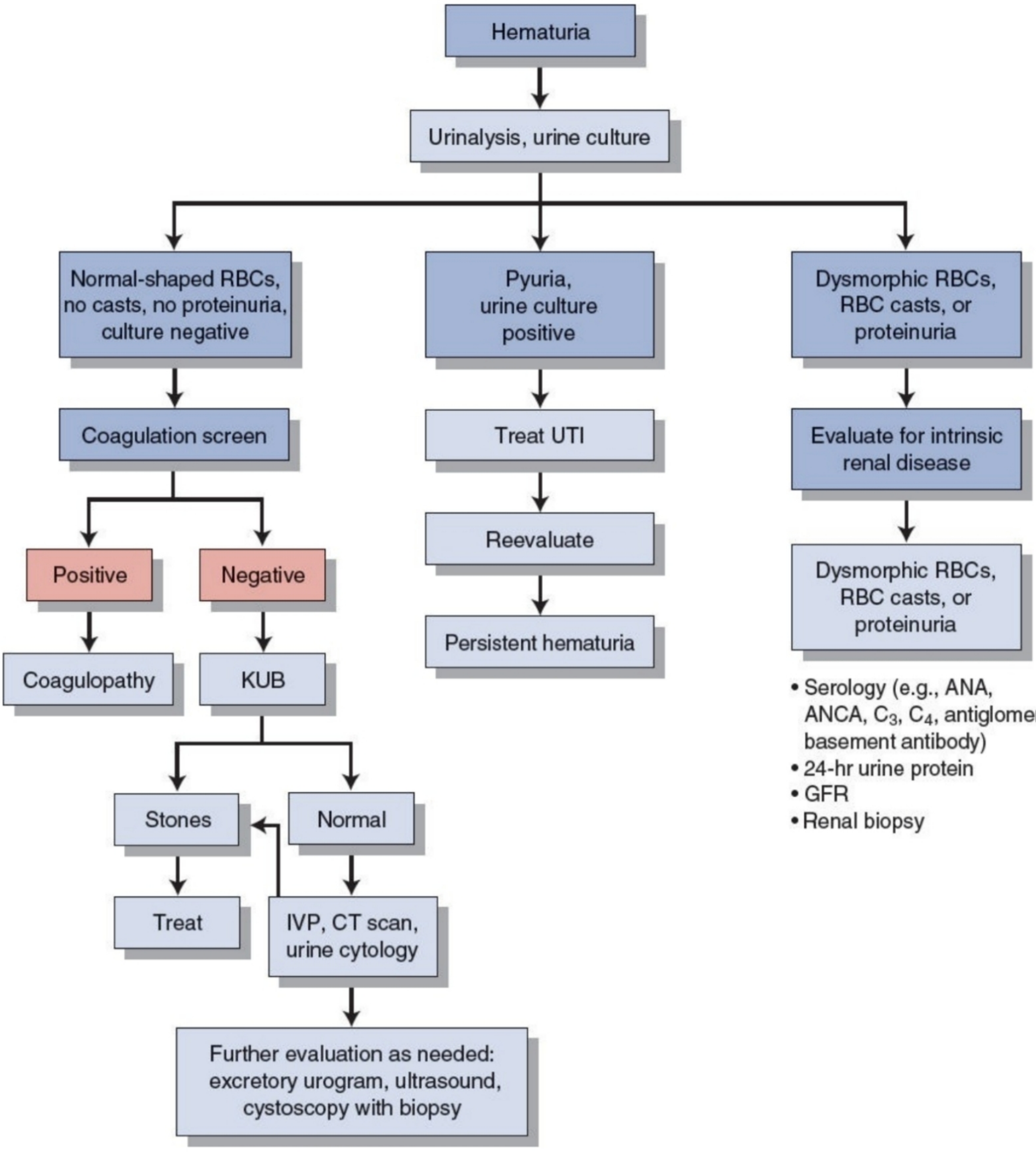


FIGURE 7.3 **Evaluation of hematuria.**

Terminology of glomerular diseases

- **Primary**: a kidney disease specifically affecting the glomeruli (e.g., minimal change glomerulonephritis)
- **Secondary**: a disease affecting the glomeruli in the context of a systemic disease (e.g., lupus nephritis in SLE) or a disease affecting another organ (e.g., diabetic nephropathy)
- **Diffuse**: > 50% of glomeruli affected (e.g., diffuse proliferative glomerulonephritis)
- **Focal**: < 50% of glomeruli affected (e.g., focal segmental glomerulosclerosis)
- **Global**: entire glomerulus is affected
- **Segmental**: only part of the glomerulus is affected
- **Proliferative**: an increased number of cells in the glomerulus
- **Membranous**: thickening of the glomerular basement membrane (e.g., membranous nephropathy)
- **Sclerosing**: scarring of the glomerulus
- **Necrotizing**: cell death within the glomerulus
- **Crescentic**: accumulation of cells such as macrophages, fibroblasts, and epithelial cells in Bowman space

---

Dynamic phases of IV fluid treatment [2][3][8]

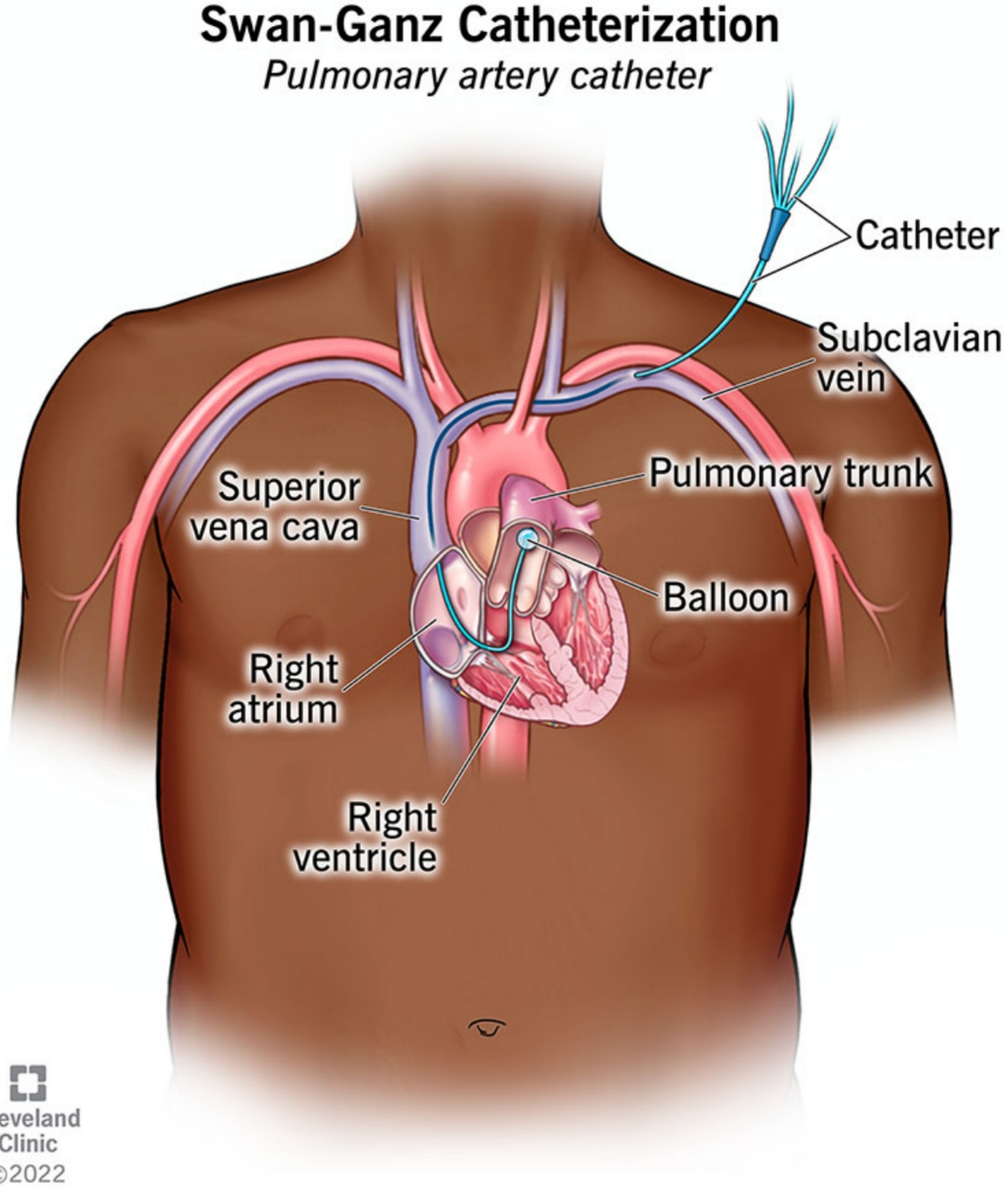
- **Patient rescue phase** (minutes): life-saving intervention for patients with severe shock (i.e., fluid resuscitation)
- **Organ rescue phase** (hours): maintenance of tissue perfusion in patients with or at risk of hemodynamic instability (i.e., IV fluid challenges , titration of maintenance fluids to maintain tissue perfusion)
- **Organ support phase** (days): management of IV fluids in stable patients (i.e., maintenance fluid therapy, replacement of ongoing losses) and prevention of unnecessary fluid accumulation (i.e., switch to oral/enteral hydration)
- **Organ recovery phase** (days to weeks): reduction of IV fluids and evacuation of fluid overload

IV fluid management strategies

Overview of fluid management strategies [2][8][19]		
Clinical scenario	**Fluid management strategy**	**Goal**
Hypovolemic shock	Immediate hemodynamic support with aggressive IV fluid resuscitation	Patient rescue
Hypovolemia or dehydration without shock	Judicious fluid replacement (e.g., with IV fluid challenge)	Organ rescue
Ongoing fluid loss greater than oral intake	Replacement of ongoing fluid loss	Organ support
Hypernatremia	Correction of free water deficit	
Inability to meet daily fluid requirements enterally	Maintenance fluid therapy	
Recovering patients	De-escalation of IV fluid therapy	Organ recovery

Swan-Ganz Catheterization

Pulmonary artery catheter



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I.V Fluids

◄ Type of I.V fluid at the time of infusion

Type of I.V fluid at the time of infusion	What happens after administration of IV?	Example
Hypotonic	Water will move from ECF into ICF	• Distilled Water • 0.45% NaCl(1/2NS) • 0.33% NaCl(1/3NS)
Isotonic	It will remain in the ECF	• NS (0.9% NaCl) • Ringers Lactate 2/3 • DW-1/3 NS • 5% Dextrose in Water(D5W)
Hypertonic	Water will move from ICF to ECF	• 3% NaCl • 10%-50% Dextrose • D5W-1/2 NS • D5NS (5%Dextrose in NS) • Amino acid solution

◄ General rules of IV fluids

- Before giving any type of I.V fluids you have to think of:
  - type of solution
  - amount
  - rate
  - duration
  - You have to reassess the patient to see if you should continue with what you gave or not. etc.
- When your aim to **resuscitate** the patient, then you **look at the blood pressure**. On the other hand, if you want **maintain** the fluid level then you have to **assess the input and output**
- An NPO 40 year old female having a normal blood pressure waiting for a lab call should be given D5 1⁄2 NS + 20 KCl + 1 cc/kg/hr. K is added to hypotonic fluids

◄ Intravenous solutions (crystalloids vs colloids)

Crystalloids

- Intravenous solutions that contain solutes that readily cross the capillary membrane (electrolytes).
- Examples: Dextrose & electrolyte solutions

Colloids[1] (PRBC)

- Intravenous solutions (IV 300mL) that DO NOT readily cross the capillary membrane **and will stay intravascular** (non-electrolytes large molecules)
- Examples: Blood, albumin[2], plasma.

- **Pure Water CAN'T be given IV, it's a hypotonic fluid thus it will go inside the RBC causing them to swell and burst (hemolysis) and damage the lining of the capillary wall (thrombophlebitis).**

Syndrome of inappropriate ADH

◀ Causes

- **Tumours**
- **Central nervous system disorders:** stroke, trauma, infection, psychosis, porphyria
- **Pulmonary disorders:** pneumonia, tuberculosis, obstructive lung disease
- **Drugs:** anticonvulsants, psychotropics, antidepressants, cytotoxics, oral hypoglycaemic agents, opiates
- **Idiopathic**

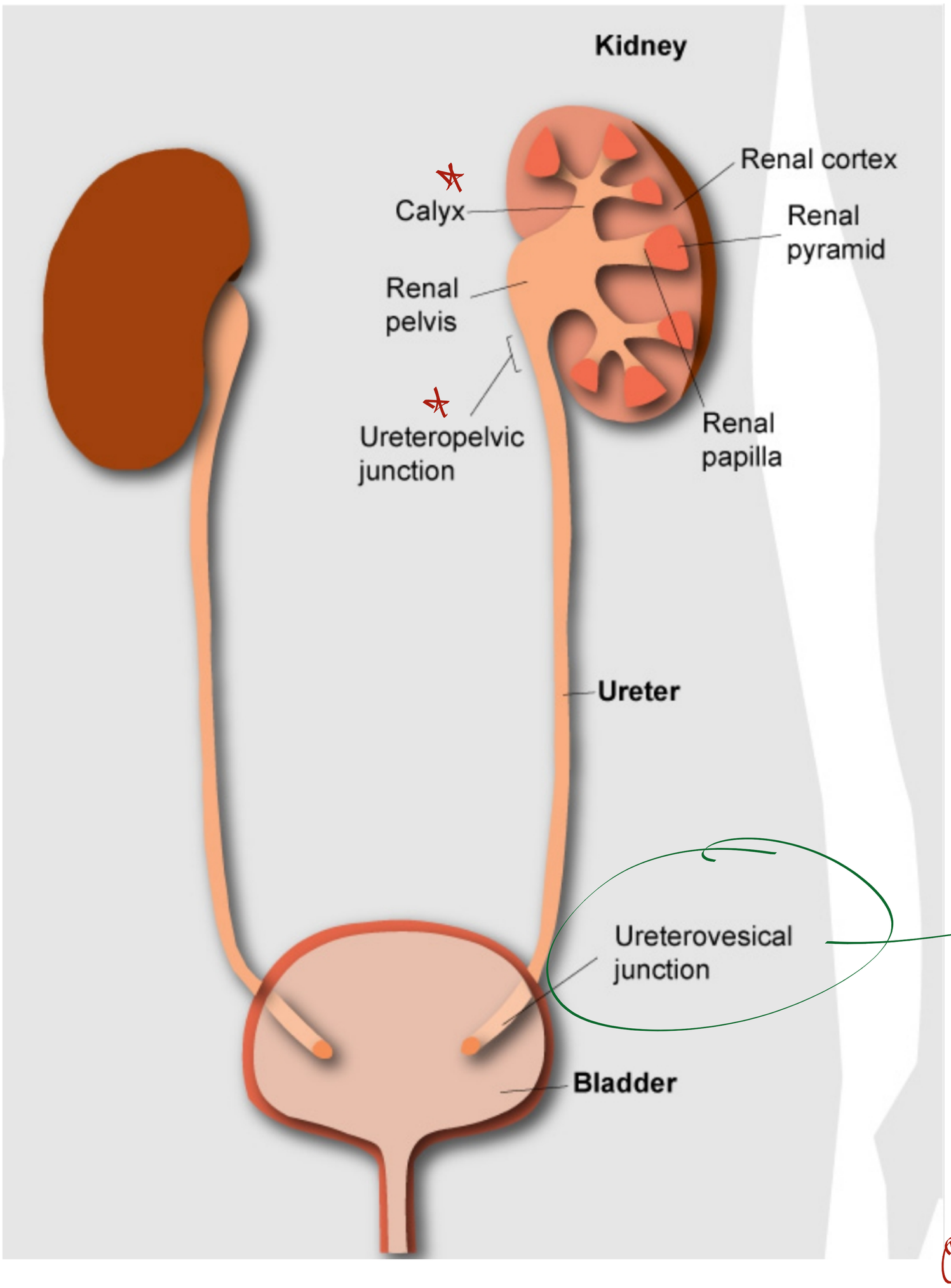
◀ Diagnosis

1. **Low plasma sodium concentration (typically < 130 mmol/L)**
2. **Low plasma osmolality (< 275 mOsmol/kg)**
3. Urine osmolality not minimally low (typically > 100 mOsmol/kg)
4. Urine sodium concentration not minimally low (> 30 mmol/L)
5. Low–normal plasma urea, creatinine, uric acid
6. **Clinical euvolaemia**
7. Absence of adrenal, thyroid, pituitary or renal insufficiency
8. No recent use of diuretics
9. Exclusion of other causes of hyponatraemia
10. Appropriate clinical context

Calciphylaxis

* In CKD, hyperphosphatemia may cause calcium and phosphate to precipitate, which causes vascular calcifications that may result in necrotic skin lesions. This is called calciphylaxis and is often irreversible

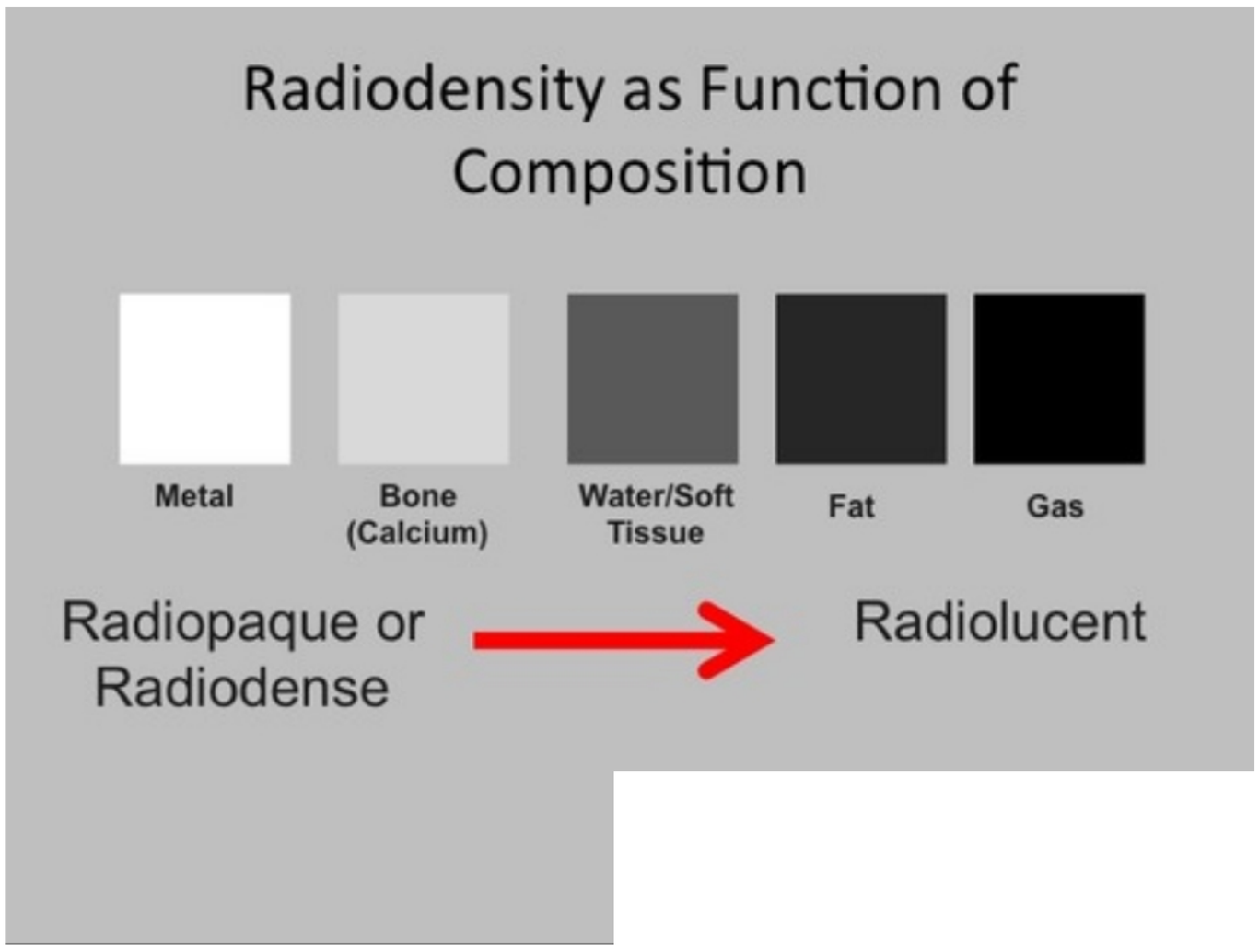
Nephrolithiasis

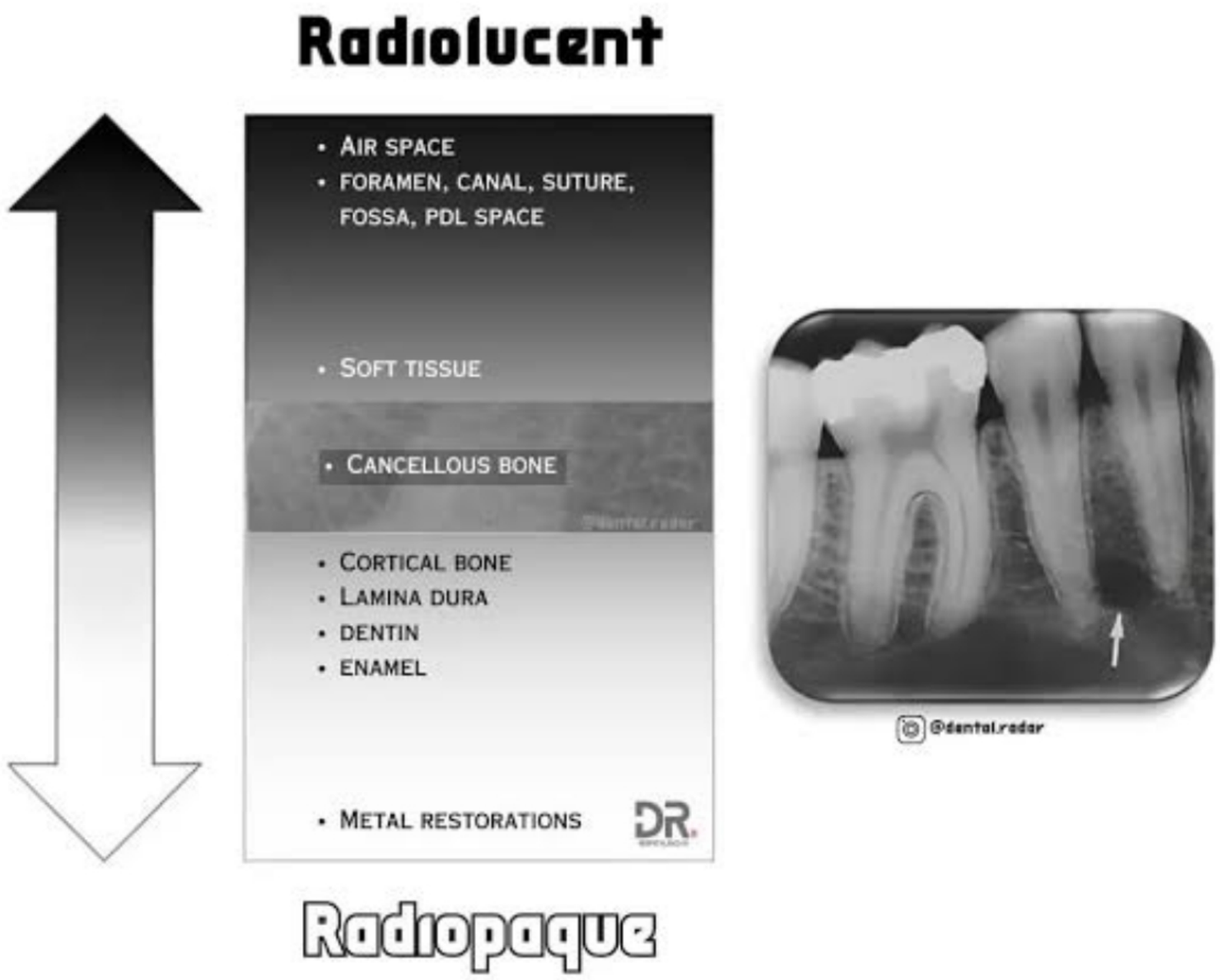


most common site of stone impaction

Other sites:
- Calyx of kidney
- Uretero pelvic junction
- Intersection of ureter and iliac vessels (near the pelvic brim)

Term	Radiopaque	Radiolucent
Definition	Substance or area that appears white on X-ray images	Substance or area that appears black or transparent on X-ray images
Appearance	Dense and blocks X-rays	Less dense and allows X-rays to pass through
Examples	Metal objects, bones, and some types of tissue	Air, gas, and some types of soft tissue
Clinical Use	Helps identify fractures, foreign objects, or tumors	Identifies air-filled structures or abnormalities
Interpretation	Indicates areas of high density or opacity	Highlights areas of low density or transparency





KIDNEY STONES

RADIOPAQUE

CALCIUM STONES (MOST COMMON)

~ CaC_2O_4 PRECIPITATES to FORM BLACK/DARK BROWN STONES SHAPED like ENVELOPE/DUMBBELL

~ Ca^{2+} + PO_4^{3-} forms DIRTY WHITE STONES SHAPED like WEDGE-PRISMS

STRUVITE STONES

~ $Mg + NH_4^+ + PO_4^{3-}$ from ACTIVITY of UREASE-POSITIVE BACTERIA

~ JAGGED CRYSTALS (LESS DENSE than CALCIUM STONES)

RADIOLUCENT

URIC ACID STONES

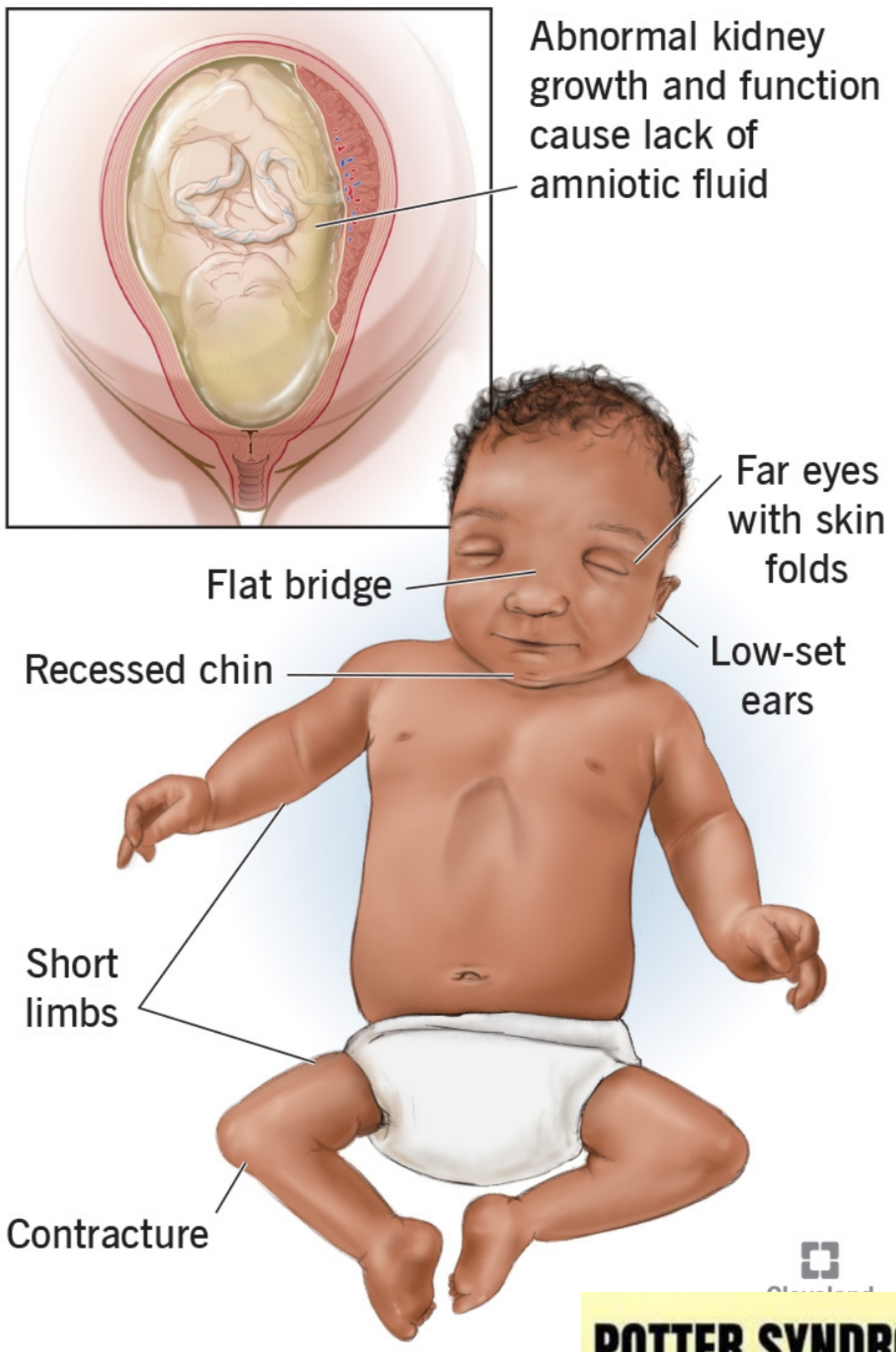
~ FORMED by MONOSODIUM URATE

~ RED-COLORED, RHOMBOID-SHAPED

CYSTEINE STONES

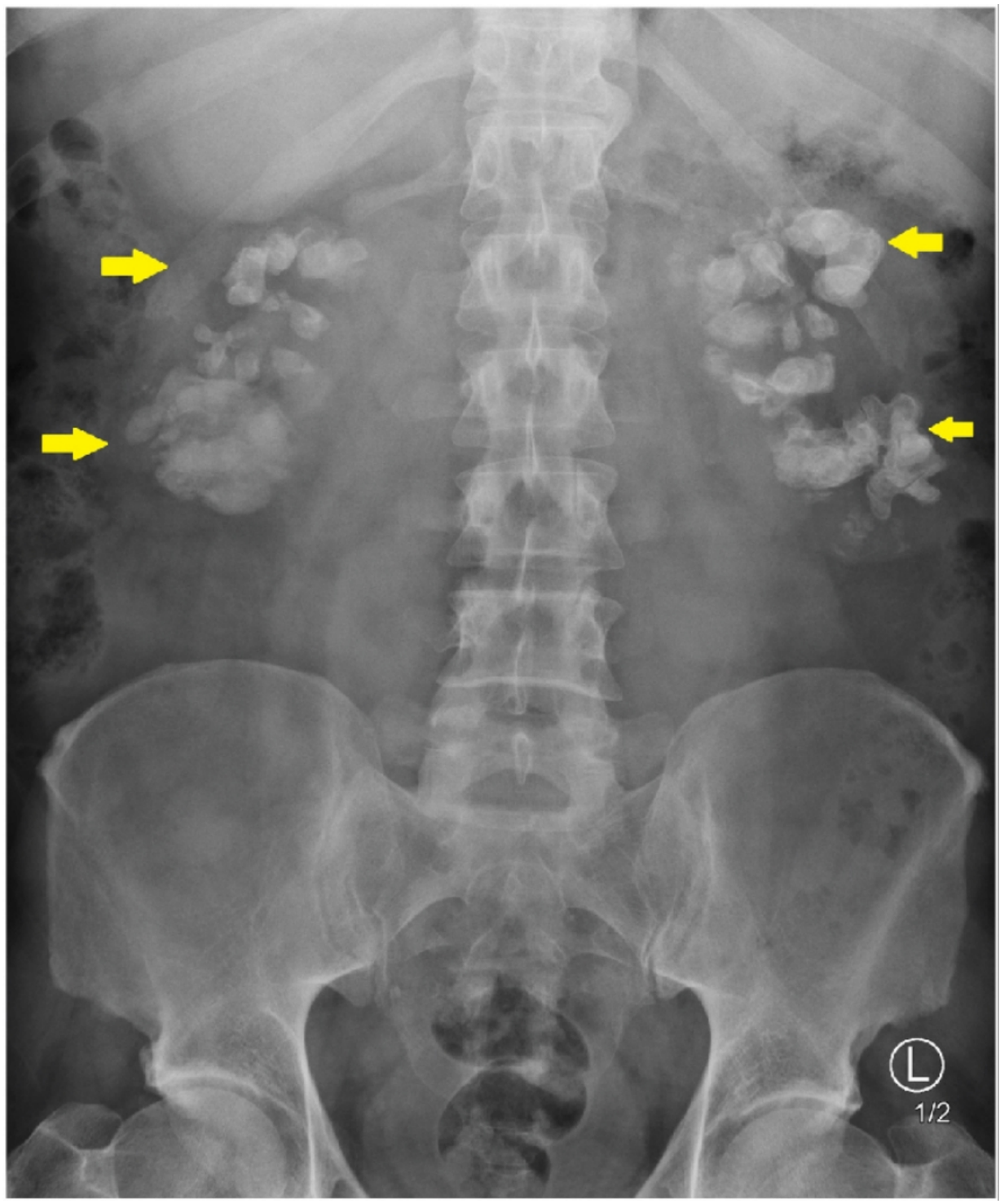
~ COMPOSED of A.A. CYSTEINE

~ YELLOW or LIGHT PINK HEXAGONS

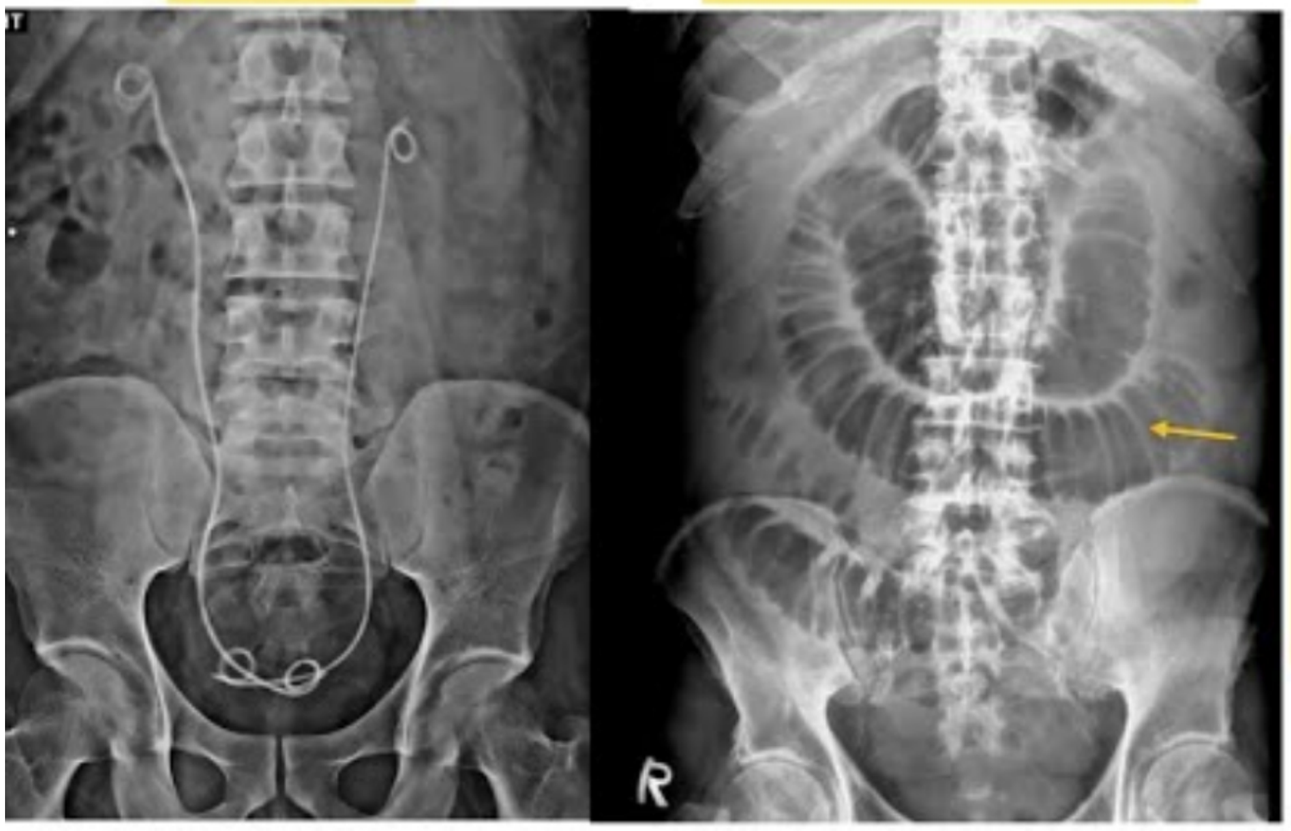

Potter Syndrome
Abnormal kidney growth and function cause lack of amniotic fluid
Far eyes with skin folds
Flat bridge
Low-set ears
Recessed chin
Short limbs
Contracture


POTTER SYNDROME signs and symptoms
POTTER
P -Pulmonary hypoplasia
O -Oligohydramnios
T -Twisted skin (wrinkly skin)
T -Twisted face (Potter face)
E -Extremities defects
R -Renal agenesis

X-Ray KUB



KUB Abdomen



Difference Between Abdomen & KUB

CTG

Contractions

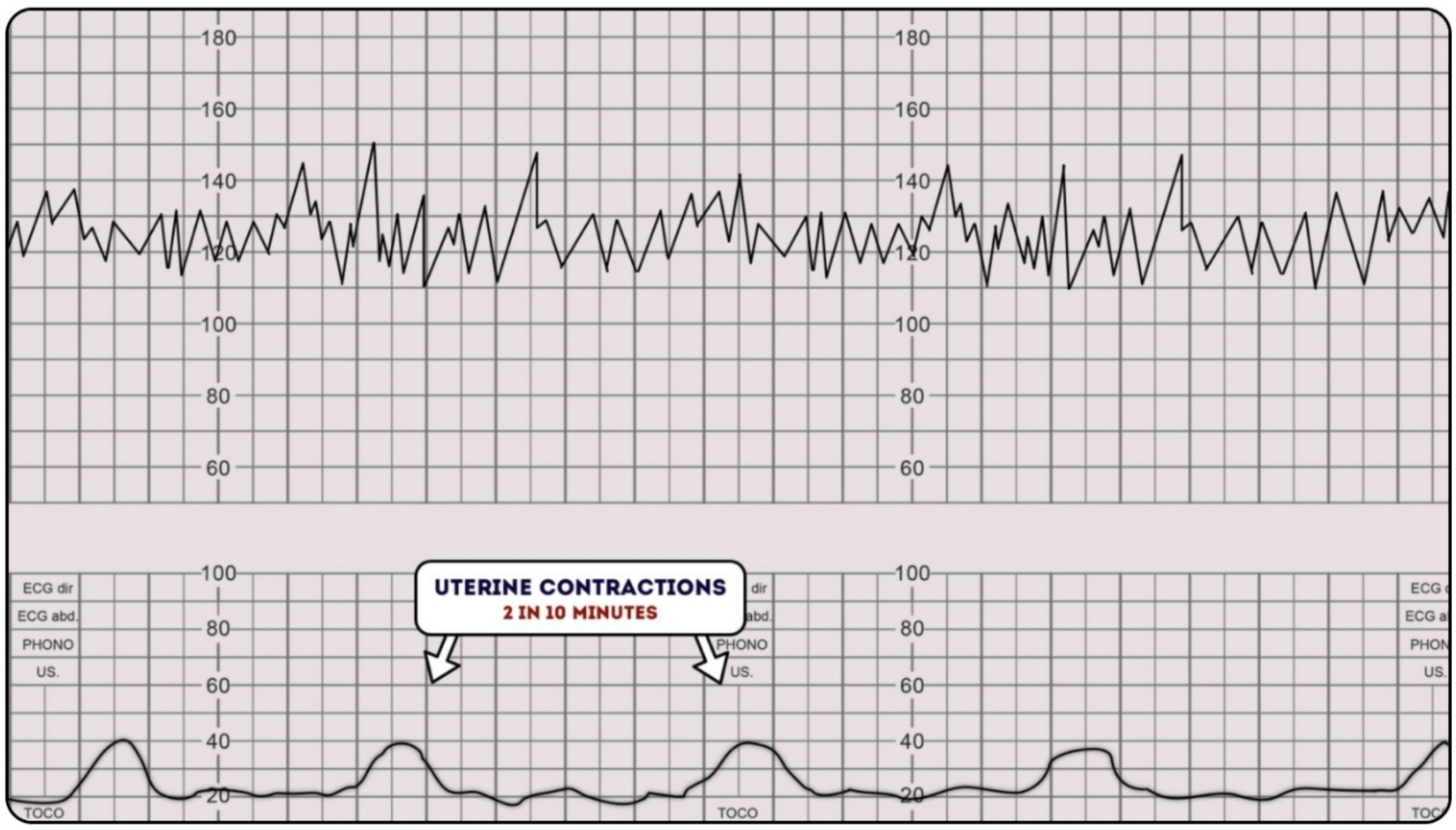
Next, you need to record the **number** of **contractions** present in a **10 minute period**.

Each **big square** on the example CTG chart below is **equal to one minute**, so look at how many contractions occurred within 10 big squares.

Individual contractions are seen as **peaks** on the part of the CTG monitoring uterine activity.

Assess contractions for the following:

- **Duration:** How long do the contractions last?
- **Intensity:** How strong are the contractions (assessed using palpation)?
- In the below example, there are 2 contractions in a 10 minute period (this is often referred to as *"2 in 10"*).



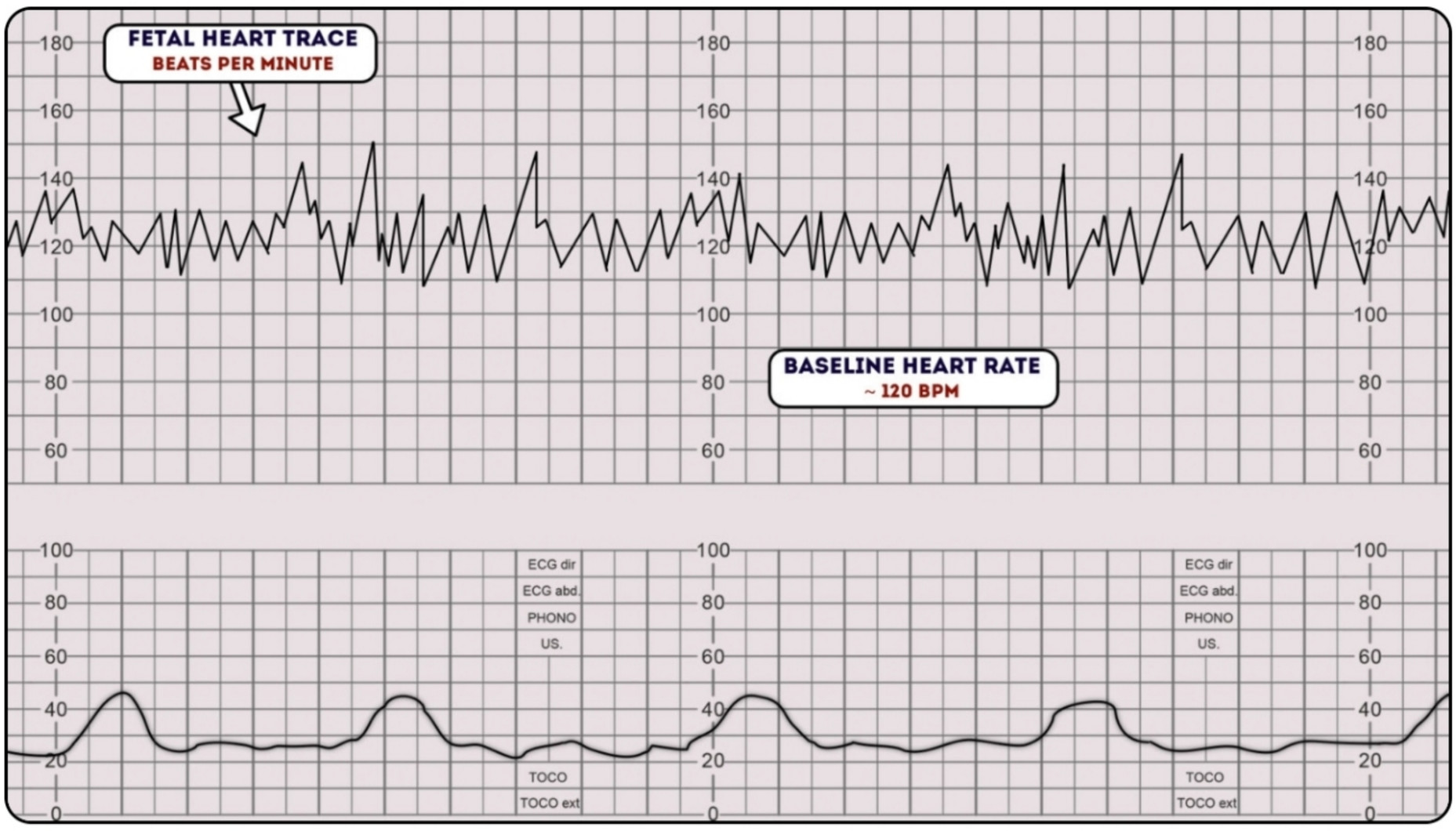
Uterine contractions (CTG)

Baseline rate of the fetal heart

The **baseline rate** is the **average heart rate** of the **fetus** within a **10-minute window**.

Look at the CTG and assess what the average heart rate has been over the last 10 minutes, ignoring any accelerations or decelerations.

A **normal fetal heart rate** is between **110-160 bpm**.



CTG: Baseline heart rate

Variability categorisation

Variability can be **categorised** as either **reassuring**, **non-reassuring** or **abnormal**. [3]

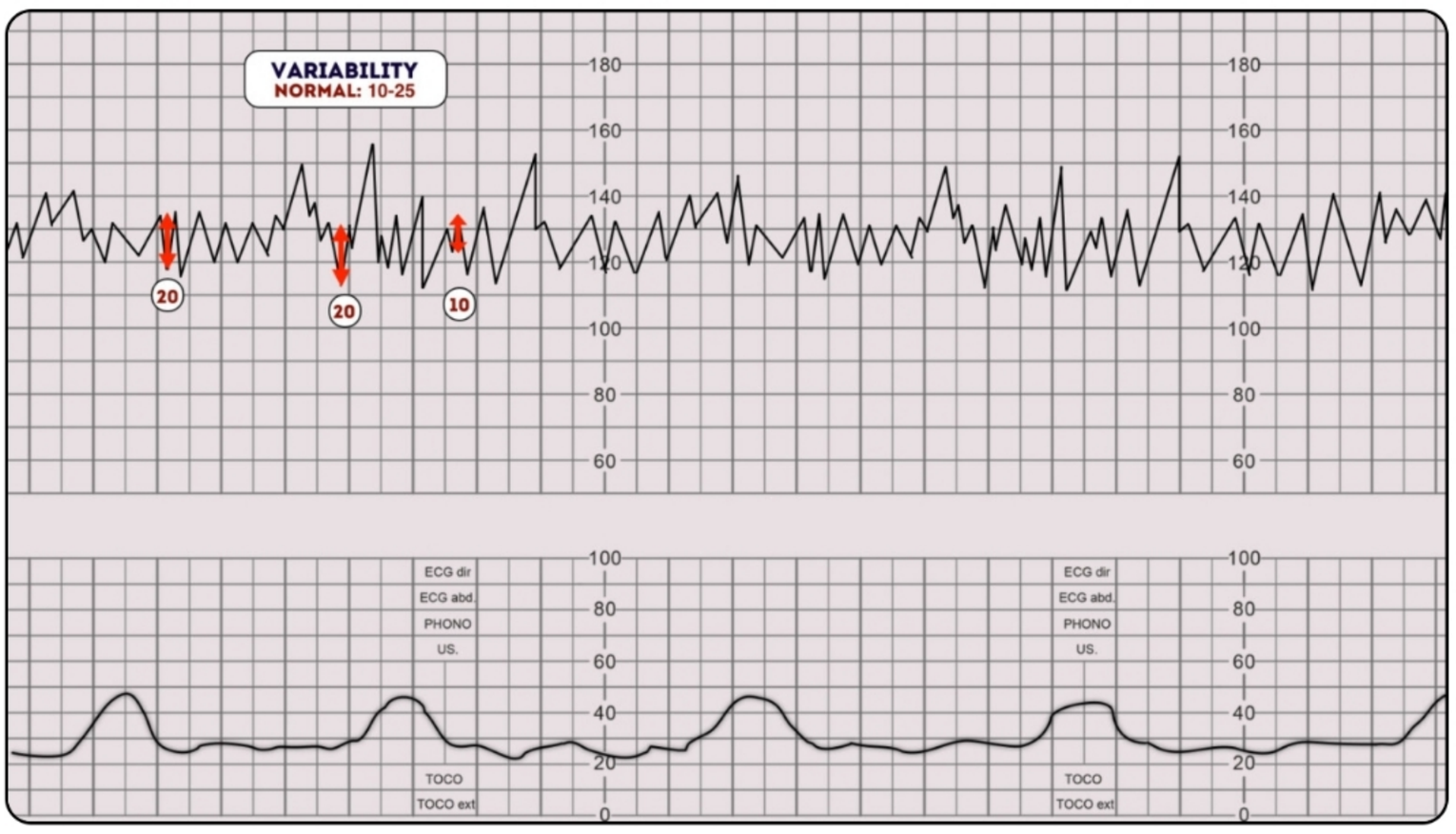
Reassuring: 5 – 25 bpm

Non-reassuring:

- less than 5 bpm for between 30-50 minutes
- more than 25 bpm for 15-25 minutes

Abnormal:

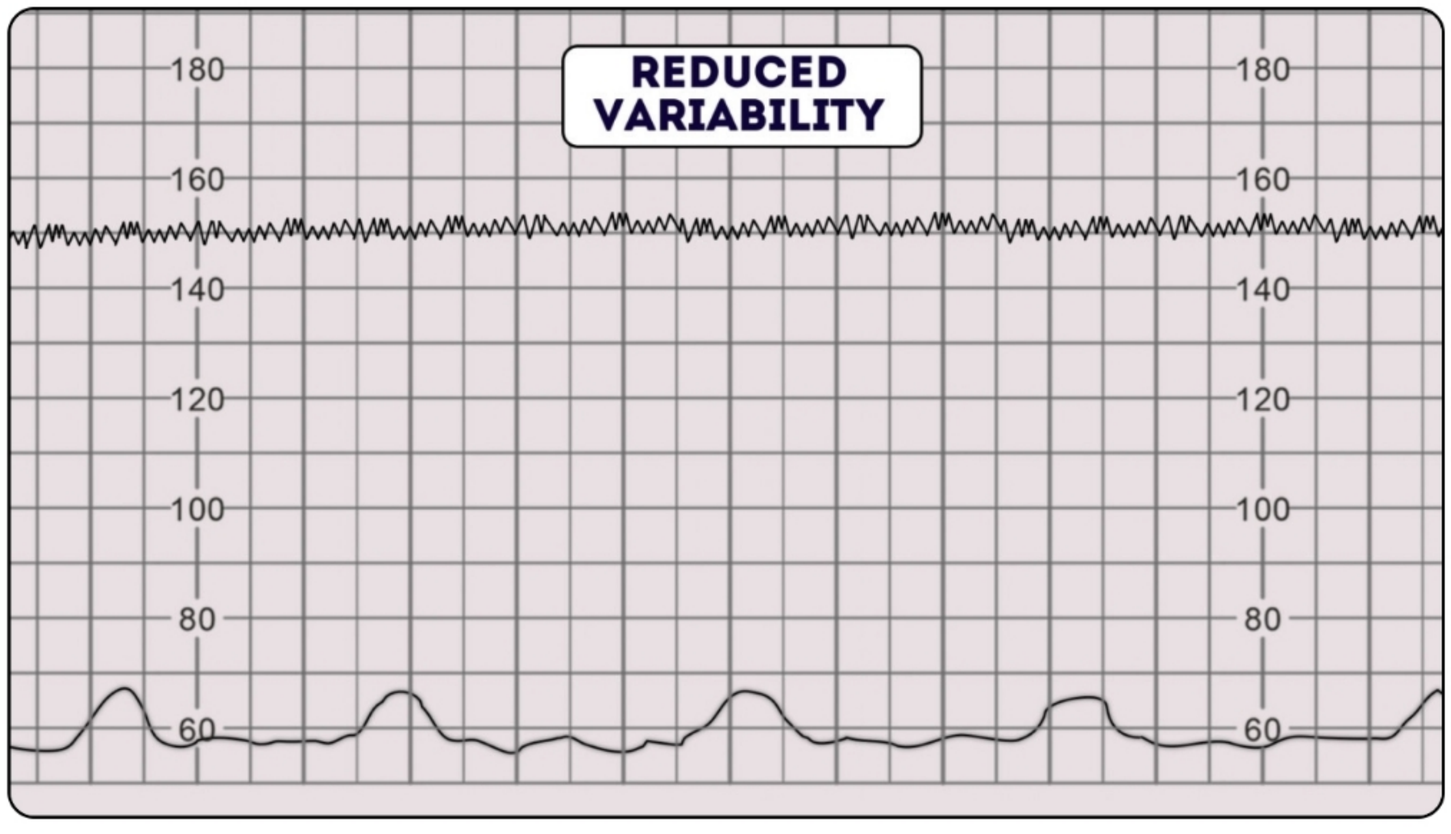
- less than 5 bpm for more than 50 minutes
- more than 25 bpm for more than 25 minutes
- sinusoidal



CTG: Variability

Reduced variability can be **caused** by any of the following:[2]

- Fetal sleeping: this should last no longer than 40 minutes (this is the most common cause)
- Fetal acidosis (due to hypoxia): more likely if late decelerations are also present
- Fetal tachycardia
- Drugs: opiates, benzodiazepines, methyldopa and magnesium sulphate
- Prematurity: variability is reduced at earlier gestation (<28 weeks)
- Congenital heart abnormalities



CTG: Reduced variability

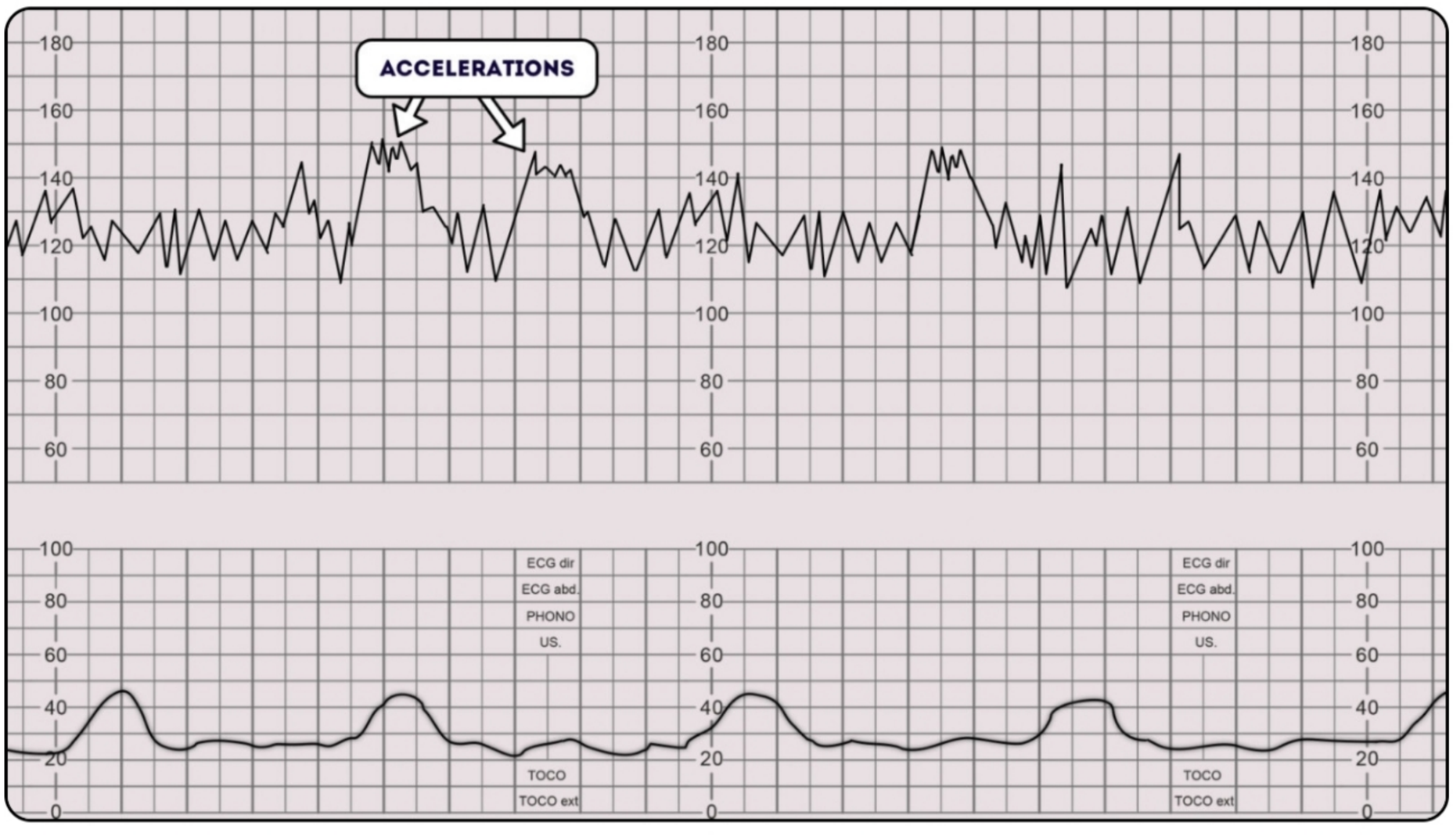
Accelerations

Accelerations are an **abrupt increase** in the **baseline fetal heart** rate of **greater than 15 bpm** for **greater than 15 seconds**.[1]

The presence of accelerations is **reassuring**.

Accelerations occurring **alongside uterine contractions** is a sign of a **healthy fetus**.

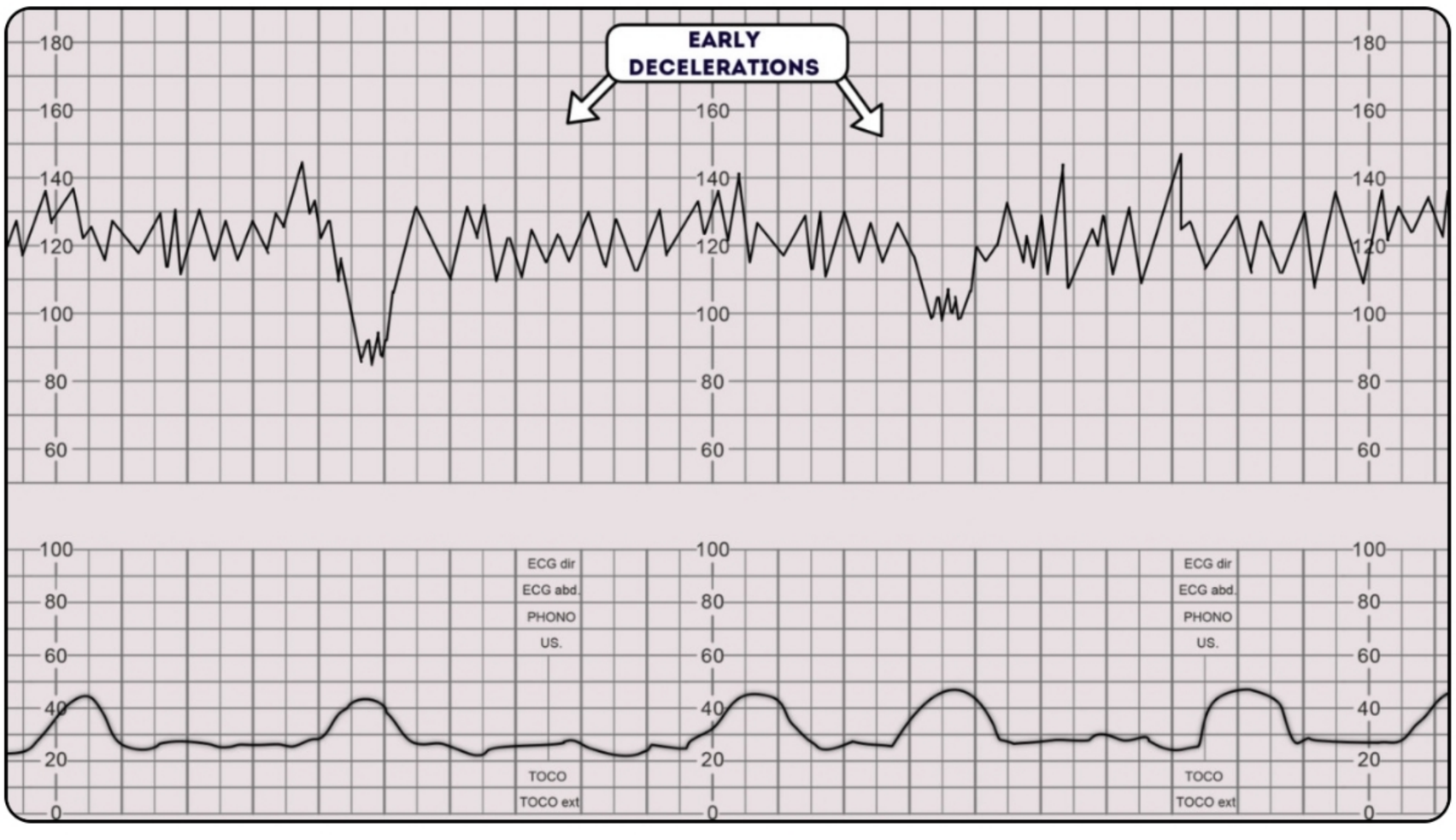
The **absence** of **accelerations** with an otherwise **normal CTG** is of **uncertain significance**.



CTG: Accelerations

Early deceleration

Early decelerations start when the **uterine contraction begins** and **recover** when **uterine contraction stops**. This is due to increased fetal intracranial pressure causing increased vagal tone. It therefore quickly resolves once the uterine contraction ends and intracranial pressure reduces. This type of deceleration is, therefore, considered to be physiological and not pathological.[3]



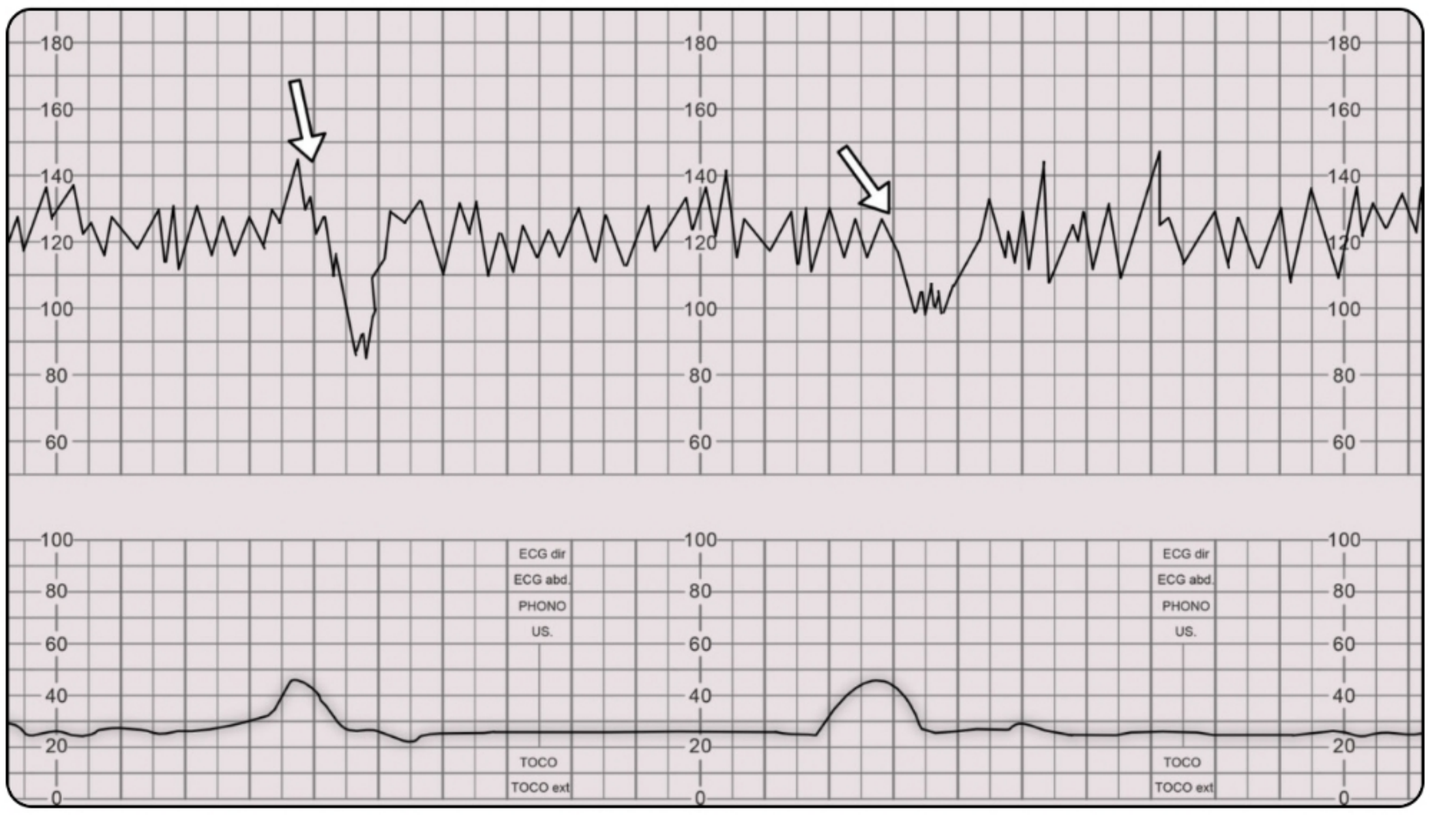
CTG: Early decelerations

Late deceleration

Late decelerations begin at the **peak of the uterine contraction** and **recover after the contraction ends**. This type of deceleration indicates there is insufficient blood flow to the uterus and placenta. As a result, blood flow to the fetus is significantly reduced causing fetal hypoxia and acidosis.

Causes of **reduced uteroplacental blood flow** include:[1]

- Maternal hypotension
- Pre-eclampsia
- Uterine hyperstimulation



CTG: Late decelerations

Variable deceleration

Variable decelerations are observed as a **rapid fall in baseline fetal heart rate** with a **variable recovery phase**.

They are variable in their duration and may not have any relationship to uterine contractions.

They are most often seen during labour and in patients' with reduced amniotic fluid volume.

All fetuses experience stress during the labour process, as a result of uterine contractions reducing fetal perfusion. Whilst fetal stress is to be expected during labour, the challenge is to pick up pathological fetal distress.

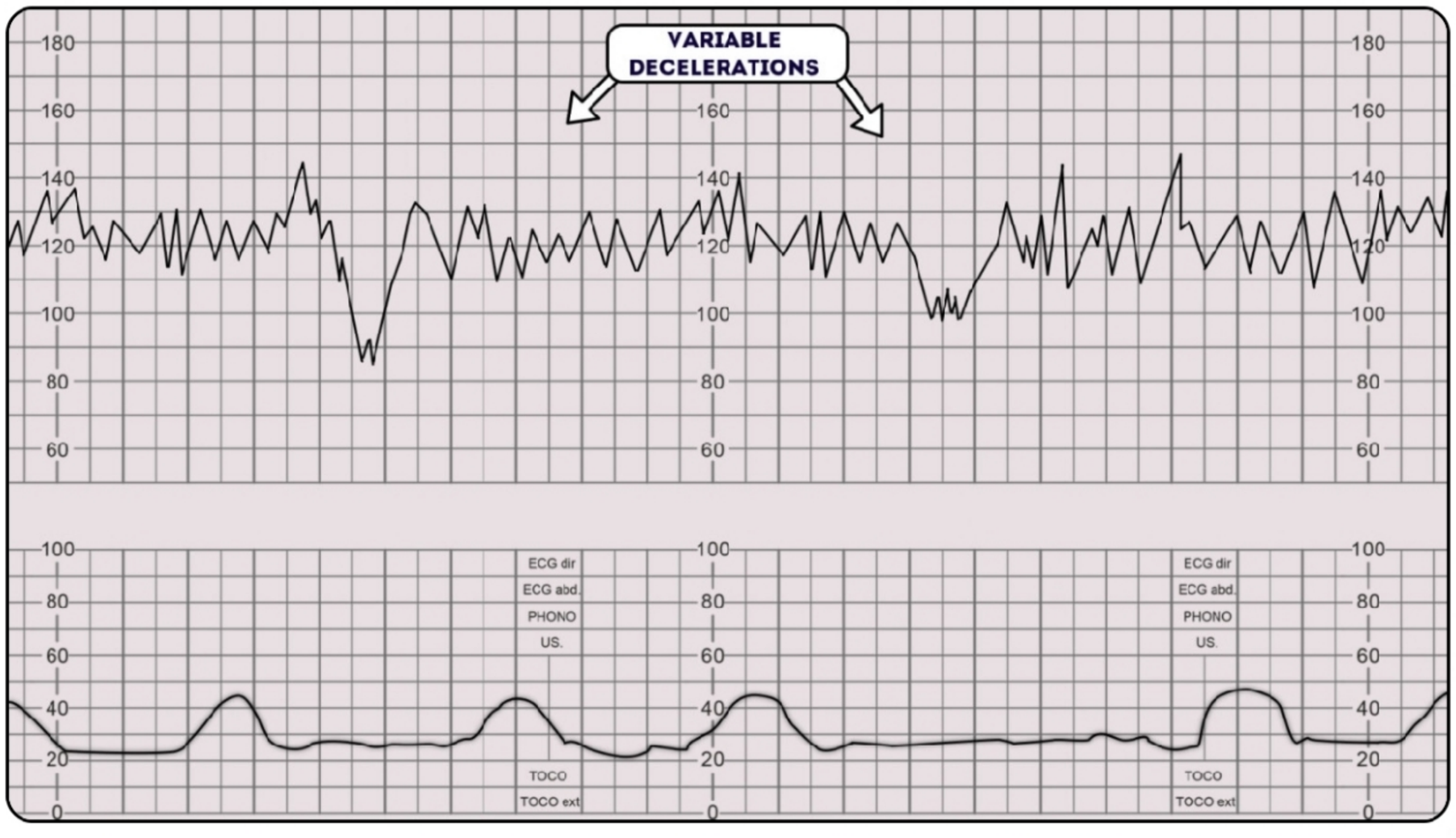
Variable decelerations are usually caused by **umbilical cord compression**. The **mechanism** is as follows:[1]

1. The umbilical vein is often occluded first causing an acceleration of the fetal heart rate in response.

2. Then the umbilical artery is occluded causing a subsequent rapid deceleration.

3. When pressure on the cord is reduced another acceleration occurs and then the baseline rate returns.

The **accelerations before** and **after** a **variable deceleration** are known as the **shoulders of deceleration**. Their presence indicates the fetus is not yet hypoxic and is adapting to the reduced blood flow. Variable decelerations can sometimes resolve if the mother changes position. The presence of persistent variable decelerations indicates the need for close monitoring. Variable decelerations without the shoulders are more worrying, as it suggests the fetus is becoming hypoxic.

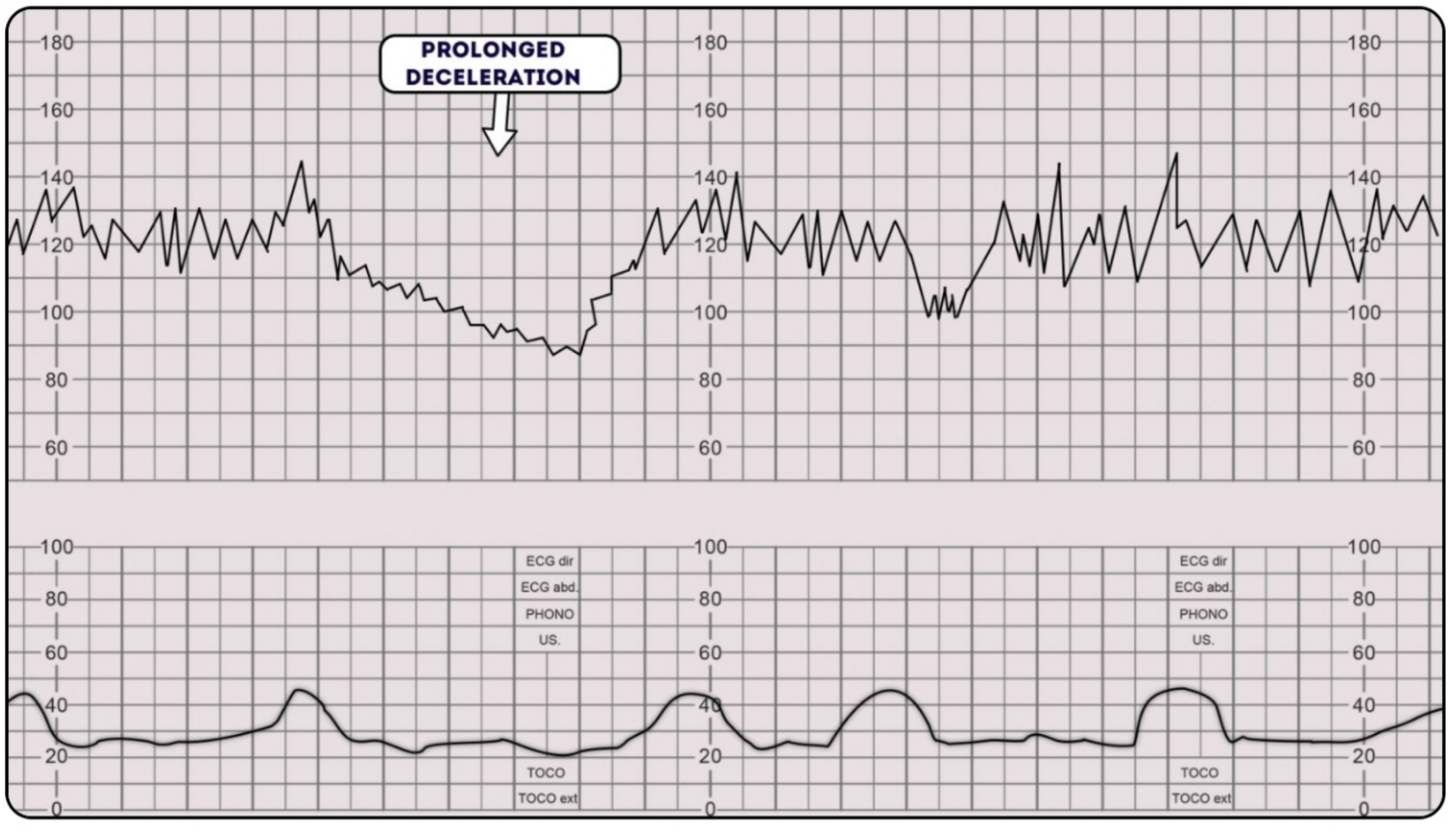


CTG: Variable decelerations

Prolonged deceleration

A **prolonged deceleration** is defined as a deceleration that lasts **more than 2 minutes**:

- If it lasts **between 2-3 minutes** it is classed as **non-reassuring**.
- If it lasts **longer than 3 minutes** it is immediately classed as **abnormal**.



CTG: Prolonged deceleration

Sinusoidal pattern

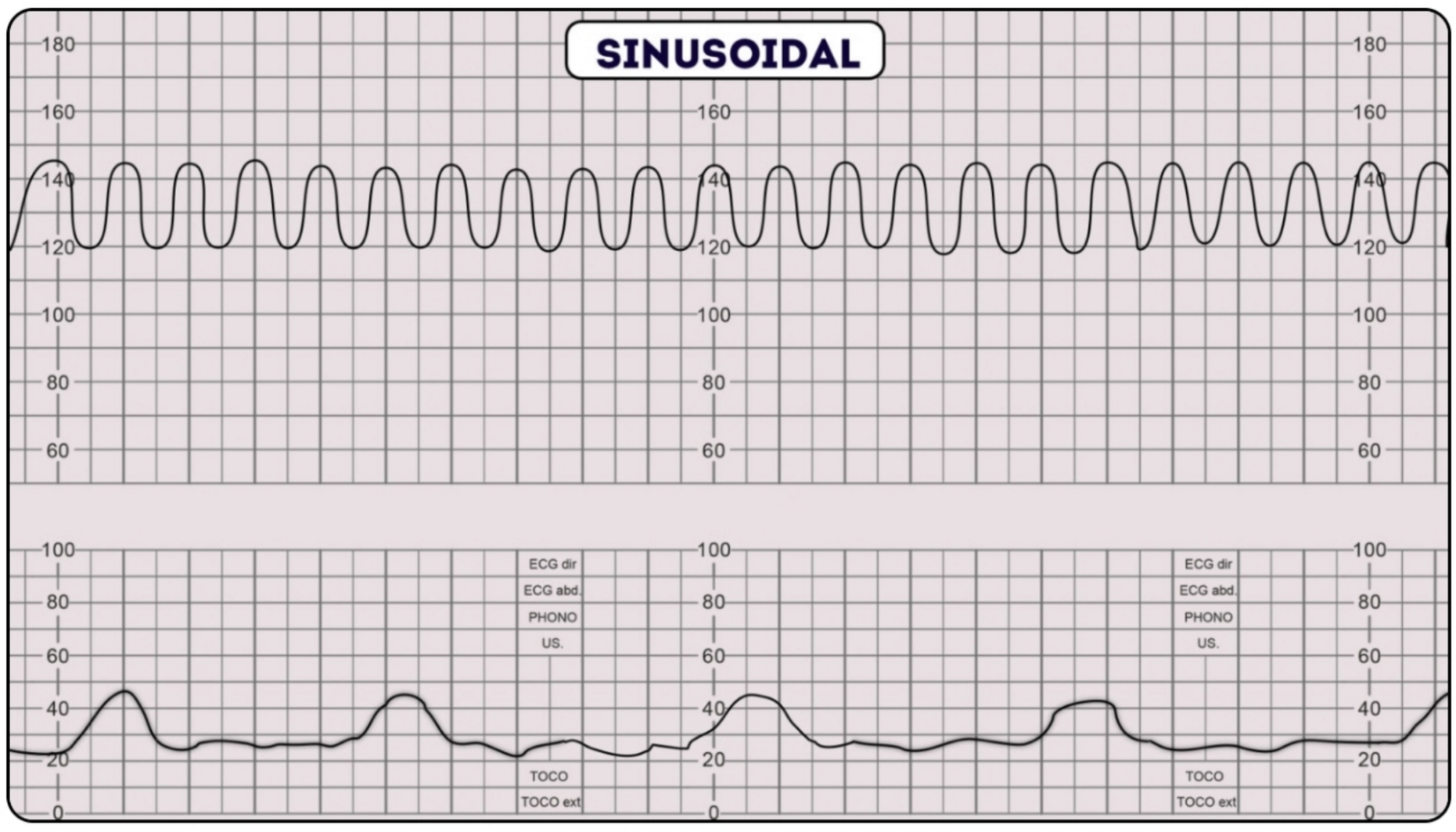
A sinusoidal CTG pattern is **rare**, however, if present it is **very concerning** as it is associated with high rates of **fetal morbidity** and **mortality**.[1]

A **sinusoidal CTG pattern** has the following **characteristics**:

- A **smooth**, **regular**, **wave-like** pattern
- Frequency of around **2-5 cycles** a **minute**
- **Stable baseline rate** around 120-160bpm
- **No** beat to beat variability

A **sinusoidal pattern** usually **indicates** one or more of the following:

- Severe fetal hypoxia
- Severe fetal anaemia
- Fetal/maternal haemorrhage



CTG: Sinusoidal pattern

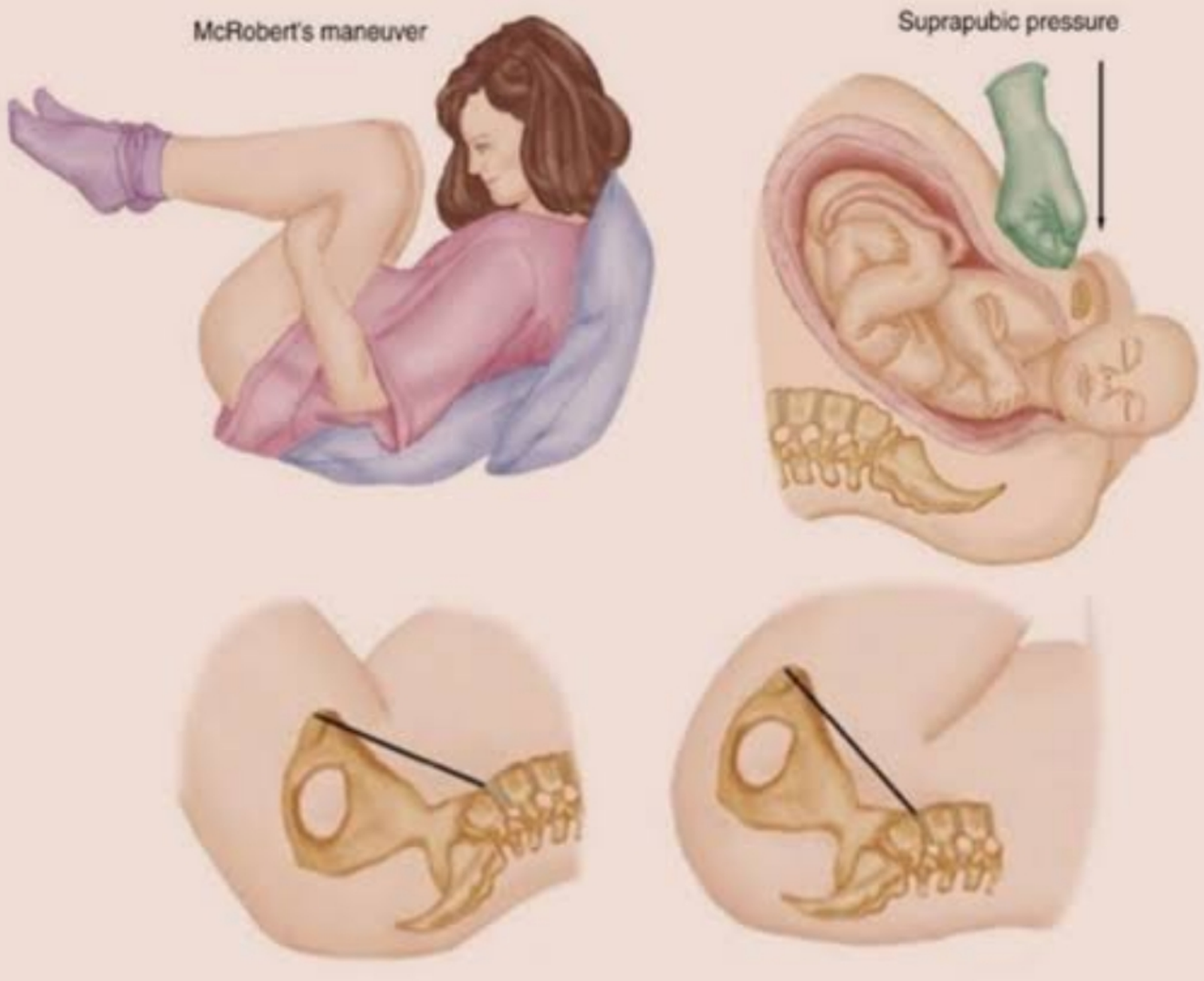
Management of urgent hypertensive pregnancy disorders

- Initiate antihypertensives for urgent blood pressure control in pregnancy.
- Administer magnesium sulfate for seizure prophylaxis.
- Assess for indications for immediate delivery regardless of gestational age.
  - If present: urgent delivery after maternal hemodynamic stabilization
  - If absent:
    - ≥ 34 weeks' gestation: Deliver.
    - 24–34 weeks' gestation: Administer corticosteroids for fetal lung maturity followed by expedited delivery.
    - Before fetal viability: Continuation of pregnancy is not recommended because of the significant risk of maternal life-threatening complications. [18]

Administer antihypertensives within 30–60 minutes of diagnosis of an urgent hypertensive pregnancy disorder, if feasible. [2]

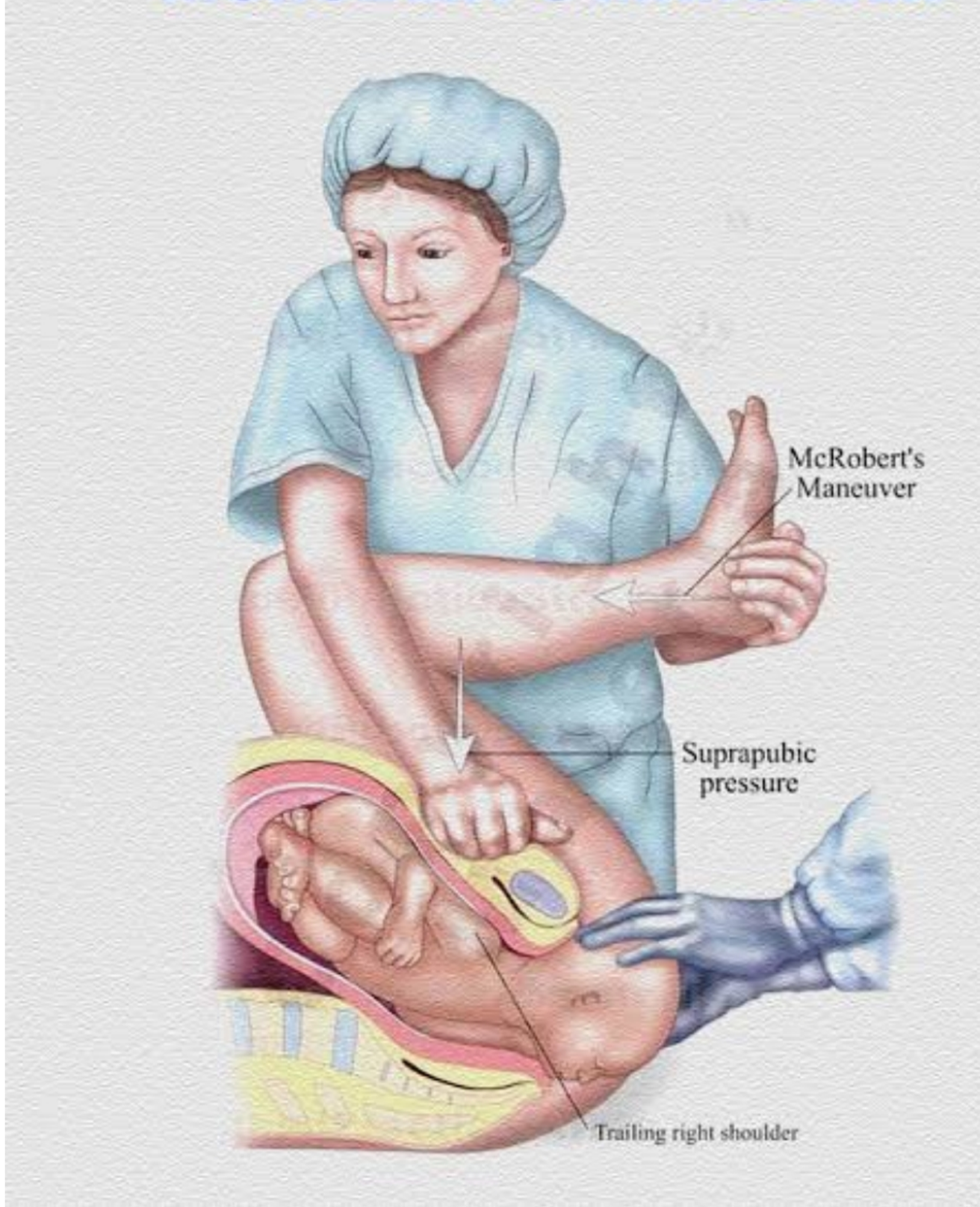
Delivery is the only cure for preeclampsia, eclampsia, and HELLP syndrome. [18]

McROBERTS MANEUVER



The McRoberts maneuver is a medical technique used to assist in difficult deliveries, particularly when the baby's shoulder is stuck (shoulder dystocia).

MCROBERT'S MANUEVER

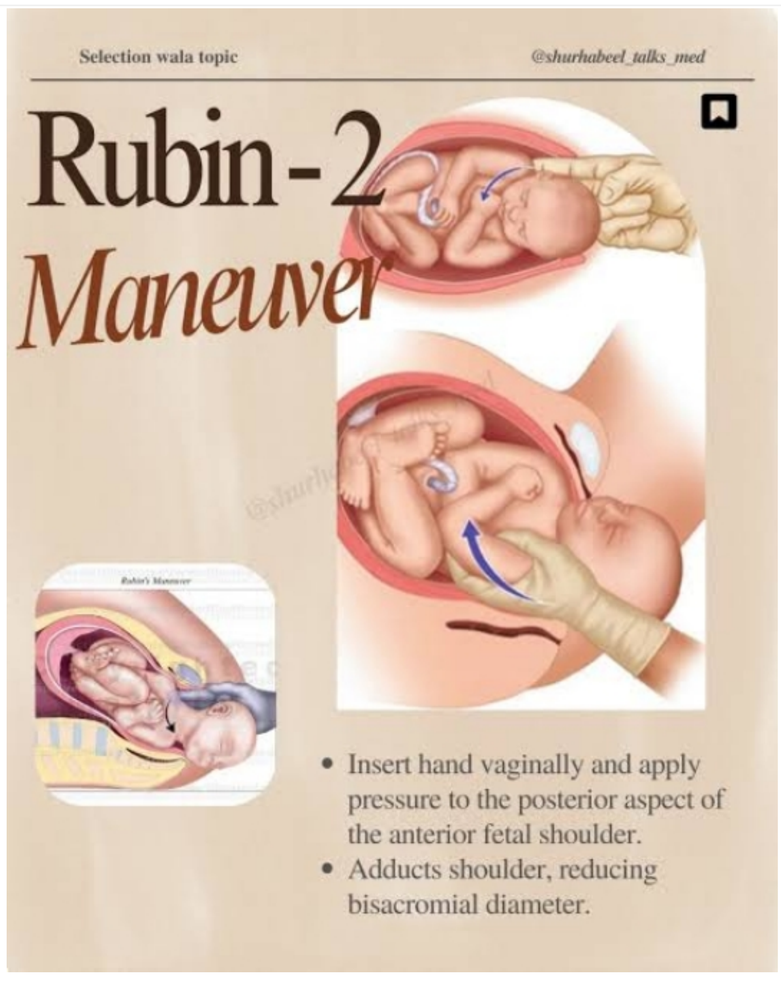


Rubin-2 *Maneuver*

- Insert hand vaginally and apply pressure to the posterior aspect of the anterior fetal shoulder.
- Adducts shoulder, reducing bisacromial diameter.

Shoulder dystocia maneuvers [11][15][19]

- **Initial maneuvers**
  - McRoberts maneuver
    - The patient should stop bearing down and lie supine with the buttocks on the edge of the bed.
    - Abduct, externally rotate, and hyperflex the maternal hips (with the maternal legs pulled towards the head).
  - McRoberts maneuver with suprapubic pressure (Rubin I maneuver) [17]
  - Manual delivery of the posterior fetal arm
- **Secondary maneuvers**
  - Rubin II maneuver: Manually rotate the fetal shoulder girdle by applying pressure to the posterior part of the anterior shoulder.
  - Woods corkscrew maneuver: Manually rotate the fetal shoulder girdle by applying pressure to the anterior part of the posterior shoulder.
  - Gaskin maneuver (all fours positions)
    - The patient moves into hands and knees position.
    - Rubin and Woods maneuvers can be repeated.



- **Maneuvers of last resort**
  - Intentional clavicular fracture: Fracture the clavicle by pulling the midportion of the clavicle upward or outward.
  - Zavanelli maneuver: The fetal head is pushed back into the pelvis to alleviate pressure on the umbilical cord while the patient is transported to the OR for cesarean delivery.
  - Symphysiotomy: surgical separation of the anterior fibers of the symphyseal ligament, only performed if all other maneuvers are unsuccessful and cesarean delivery is not available

Internal maneuvers (i.e., maneuvers requiring direct manipulation of fetal parts) may require episiotomy and can be performed together with the McRoberts maneuver. [19]

Avoid excessive downward or lateral traction on the fetal head and neck, as this can result in brachial plexus injuries. [18]

Woods Screw Maneuver

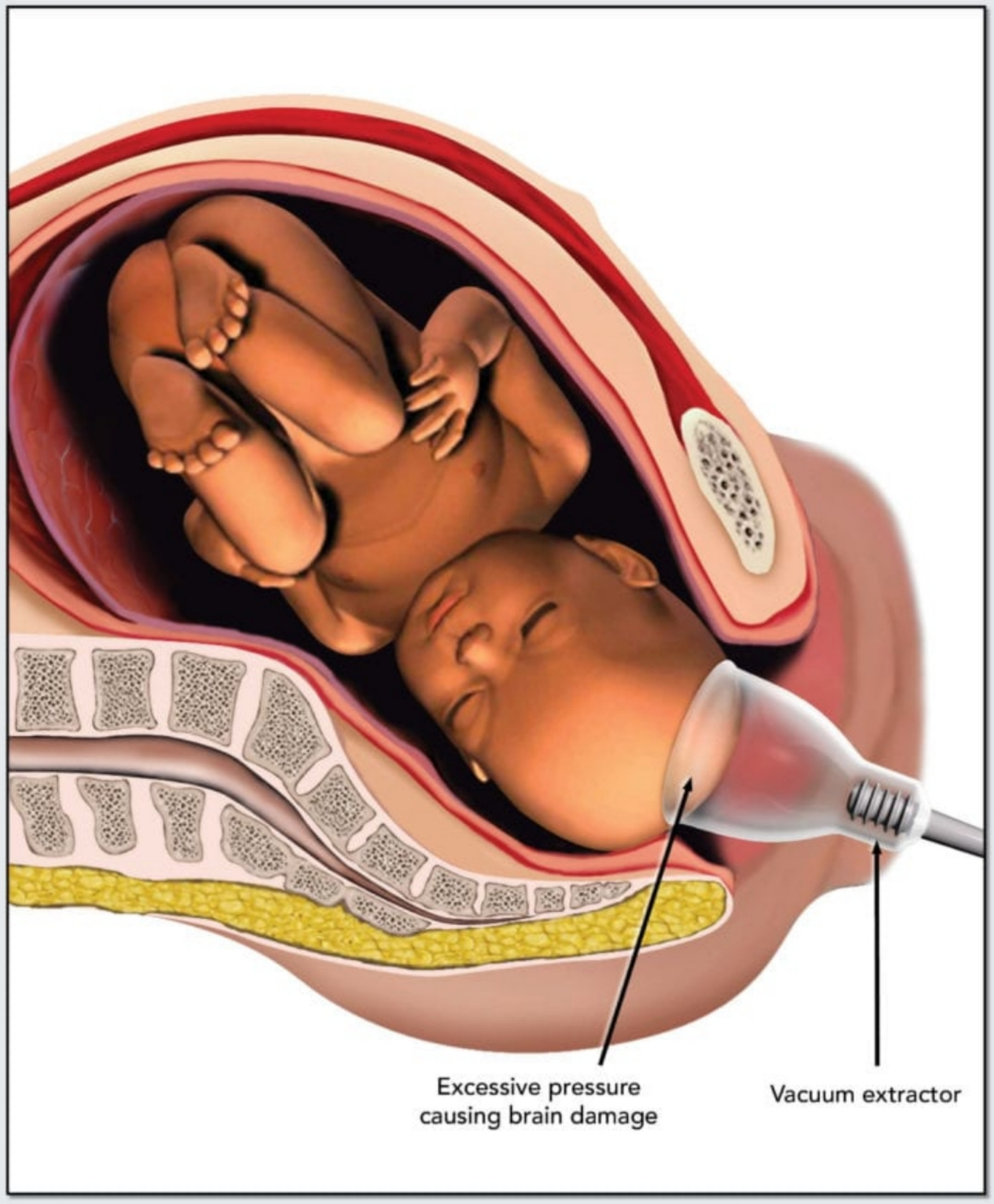
TRUSTED

- The Woods screw maneuver, also known as the Wood's corkscrew maneuver, is an obstetric technique used to manage shoulder dystocia during delivery, aiming to dislodge the impacted shoulder and facilitate delivery. This maneuver is a component of a larger set of maneuvers employed when the baby's shoulders become stuck behind the mother's pubic bone during delivery
- Goal-
  To facilitate Shoulder delivery
- Technique- Rotating the baby's shoulders within the birth canal using a rotational, corkscrew-like motion

Nursing Highlights Channel

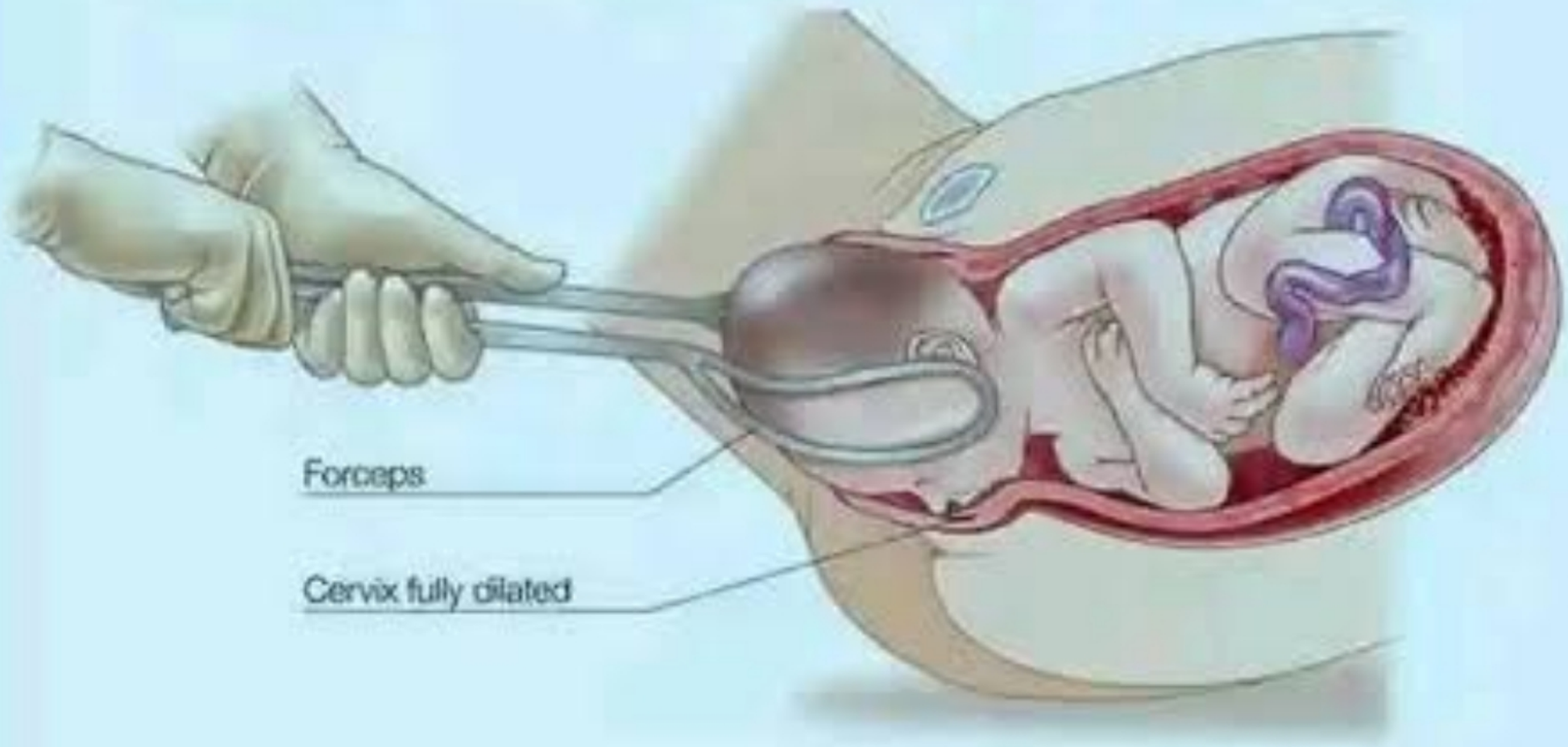
LIKE & SUBSCRIBE

BABY IN BIRTH CANAL WITH VACUUM EXTRACTOR



Attempt to deliver baby with the use of vacuum extractor

FORCEP ASSISTED DELIVERY



VACCUM ASSISTED DELIVERY

Uterine Rupture

* Uterine rupture caused by gross uterine distension or uterine scarring

Signs of imminent uterine rupture [7]

- Severe abdominal pain
- Increased contractions followed by hyperactive labor
- Bandl ring: muscular ring that can be seen above the belly button due to the powerful contractions of the upper uterine segment

Signs of uterine rupture [3][7][8][9]

- Fetal distress (earliest and most sensitive sign)
- **Severe abdominal pain**
- Referred pain in the shoulder may be present.
- Sudden pause in contractions
- Light to moderate **vaginal bleeding**
- Hemodynamic instability (as a result of abdominal bleeding)
- **Loss of fetal station** (a specific but uncommon sign)
- **Palpable fetal parts** through the rupture (a specific but uncommon sign)

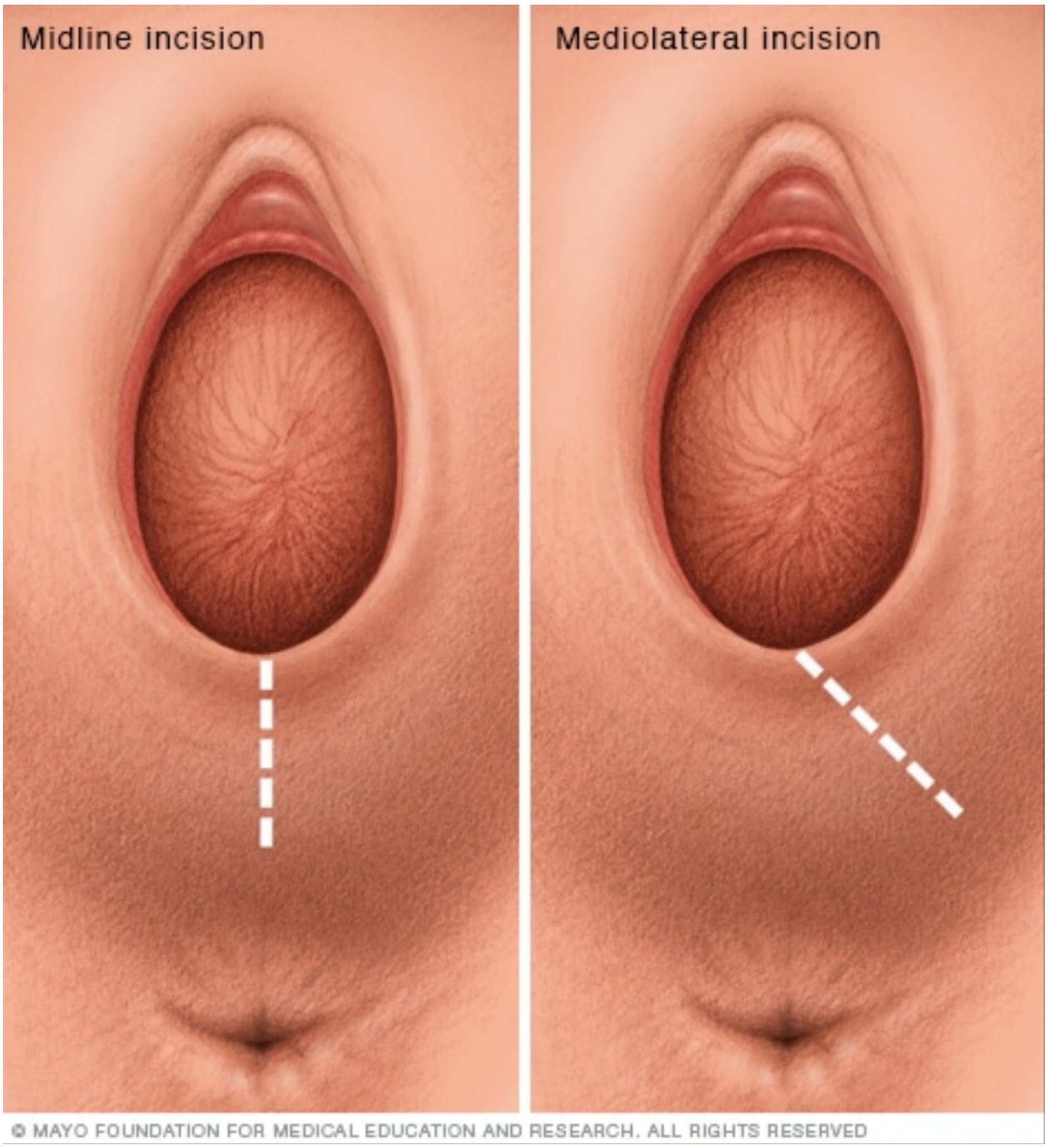
Uterine rupture generally occurs during active labor. However, a third of uterine ruptures occur prior to the onset of labor. [8][10]

Treatment

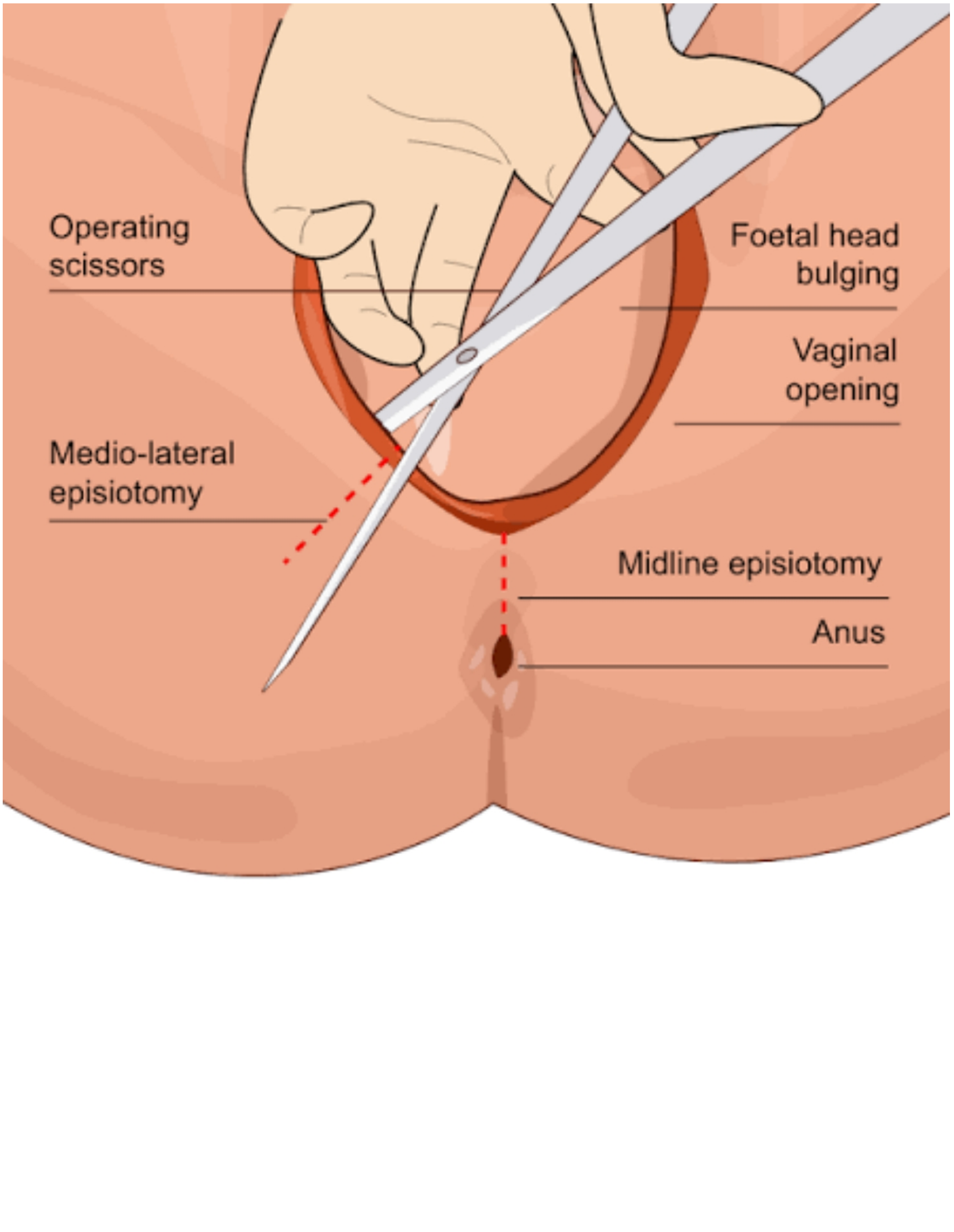
- ABCDE approach and immediate hemodynamic support
- Immediate OB-GYN consultation for emergency cesarean delivery [8][12]
- Avoid uterotonic agents. [8]
- Consider hysterectomy for refractory hemorrhage.

All patients with uterine rupture or imminent rupture require immediate laparotomy with emergency cesarean delivery within 30 minutes. [8]

Episiotomy



Episiotomy


Operating scissors
Foetal head bulging
Vaginal opening
Medio-lateral episiotomy
Midline episiotomy
Anus

Symptoms of Cephalohematoma

The most common symptom of cephalohematoma is an unnatural bulge appearing on the infant's head. There will be no cut or bruise, but you may notice the bulge gets harder over time.