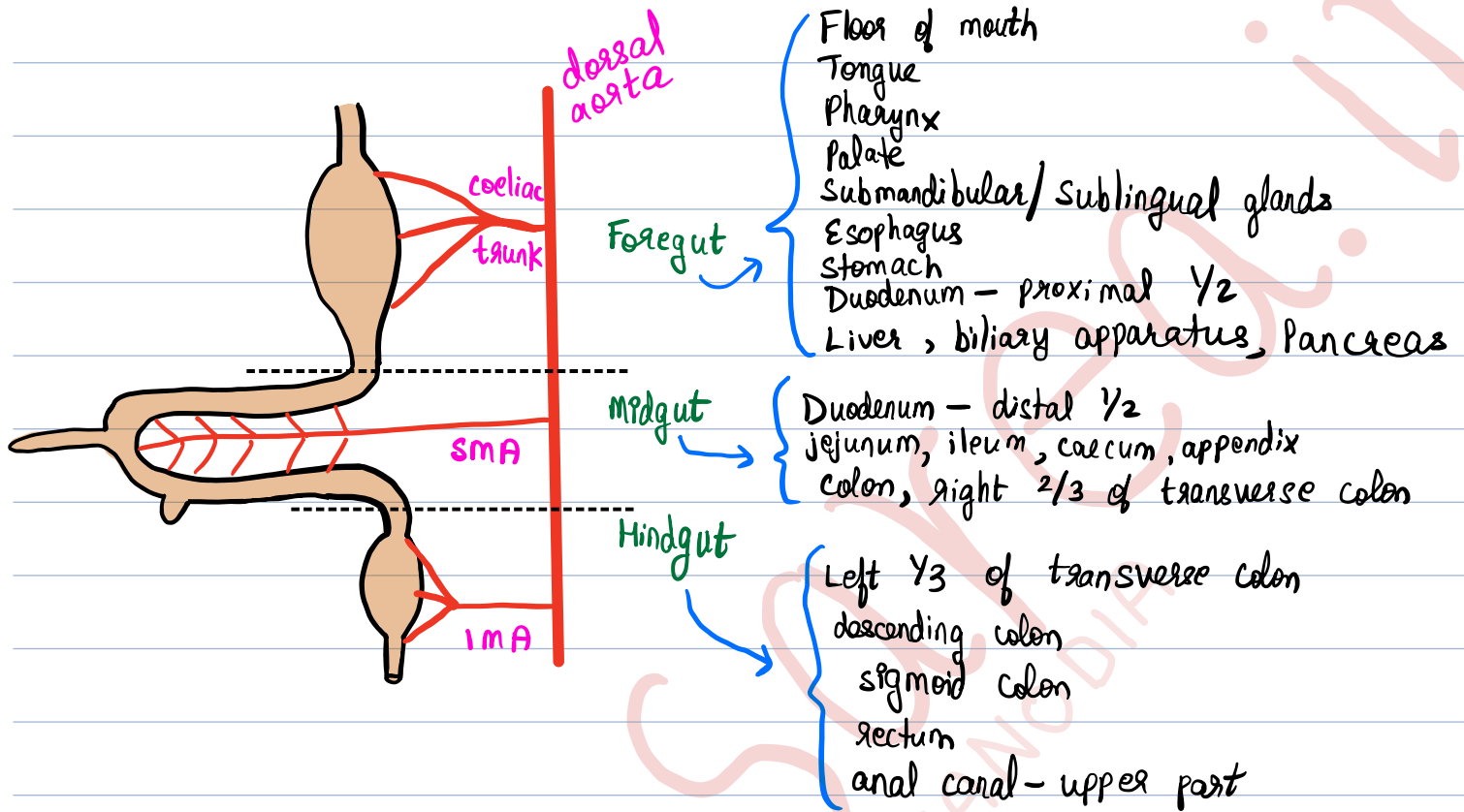


Derivatives of Gut Tubes:

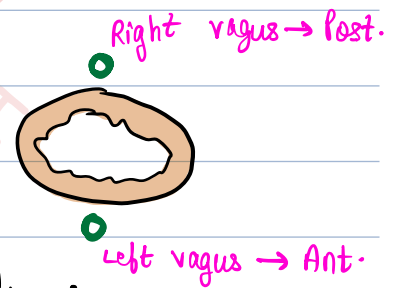
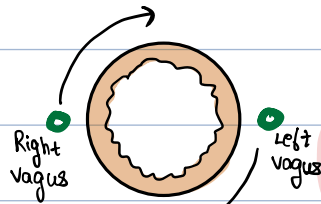
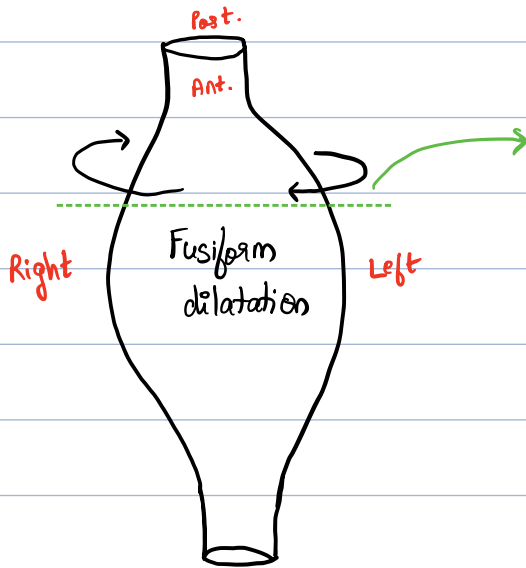
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Development of Stomach:

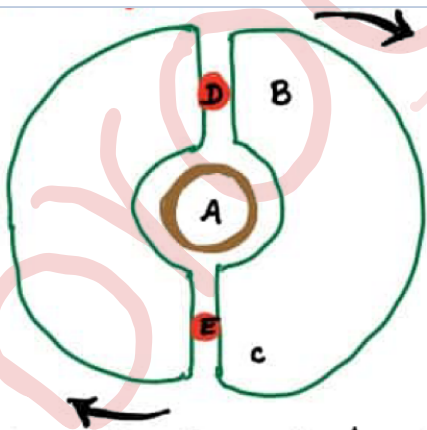
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- Distal part of foregut forms a fusiform dilatation which forms the stomach.
- 4-5th week of IUL

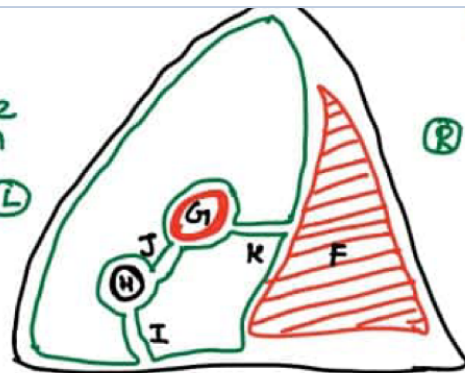


3 factors governing the rotation of stomach to adult position:

- rapid enlargement of liver
- omental bursa shifts to left
- rotation by active growth process



clockwise
rotation
90°



A-stomach, B-ventral mesogastrium, C-Dorsal mesogastrium
D-Liver, E-spleen, F-Liver, G-Stomach, H-spleen, I-lesser omentum
J-gastrosplenic ligament, K-lesser omentum

Development of stomach:

Stomach → Rotates 2 times (clock wise direction)

- 1st Rotation - clockwise direction
- vertical axis

Anterior border becomes → (R) border

lesser curvature

Posterior border becomes → (L) border

Greater curvature

∴ (L) vagus on (L) border → superficial → Ant. vagal trunk
(R) vagus → Deep plane (posterior) → Post vagal trunk

- 2nd Rotation - clockwise direction
- Anteroposterior axis

Cardiac end → (L) side

pyloric end → (R) side

→ Gastric glands develop during 3rd month

→ Oxyntic & zymogen cells " " 4th "

→ Rennin begins to function by 5th month

Mesogastrium:

- derivatives of ventral mesogastrium

lesser omentum → Derived from dorsal part of ventral mesogastrium

Hepatogastric
Hepato duodenal

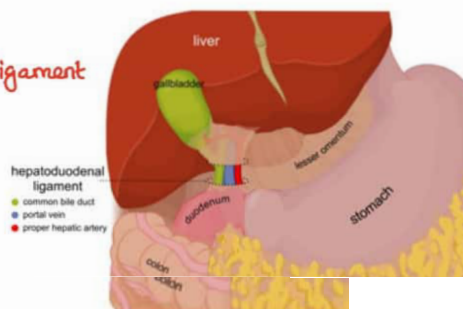
Falciform ligament → Derived from ventral part of ventral mesogastrium

coronary ligament

Triangular ligament

Derivatives of dorsal mesogastrium:

Gastrosplenic ligament
Leinorenal ligament
Greater omentum
Gastrophrenic ligament



Hypertrophic pyloric stenosis:

- muscularis externus in pyloric region
- Hypertrophy

Pyloric stenosis

- Palpable mass at (R) costal margin
- projectile Non bilious vomiting immediately after feeding



Development of Duodenum:

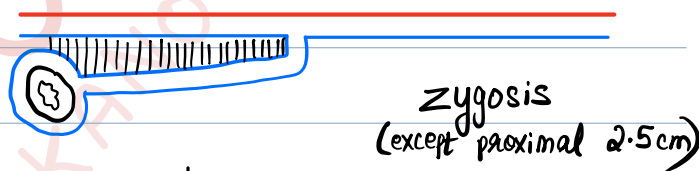
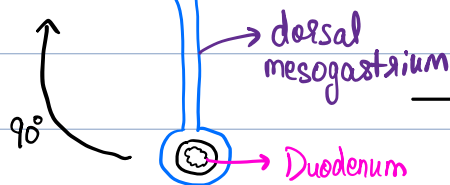
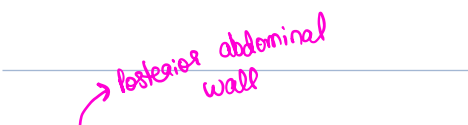
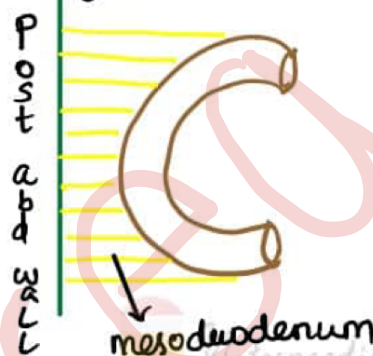
Development of duodenum:

- upto 2nd part of duodenum → foregut → celiac trunk (Campulla of Vater)
- Rest of duodenum → midgut → sup. mesenteric artery
- due to rotation of stomach - duodenum rotates to right
- mesoduodenum - extends from duodenum to posterior Abdomen wall

↓
It disappears later (zygosis)

↓
Duodenum becomes retroperitoneal
[except the proximal part - intra parietal]
- proximal part of duodenum → Hepato duodenal part of lesser omentum

* proximal 2.5 cm



↓
becomes retroperitoneal



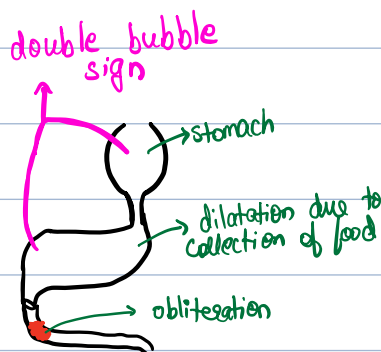
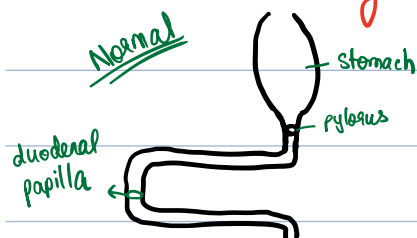
→ lumen of duodenum is obliterated in 8th week & recanalized in 3rd month

Duodenal Atresia: lumen of duodenum is obliterated

due to - failure of recanalization

- annular pancreas

Double bubble Sign in USG:



Development of Pancreas:

Development: pancreas develops from 2 separate buds - ventral & dorsal pancreatic buds

∴ it has 2 ducts.

- smaller ventral pancreatic bud arises in common with hepatic bud for liver
- larger dorsal pancreatic bud arises more proximally, directly from duodenum.
- After rotation of gut, ventral pancreatic bud passes along with bile duct into the position dorsal to dorsal pancreatic bud
- the two buds fuse & their ducts anastomose in such a way that duct of ventral pancreas forms proximal part of main pancreatic duct
- duct of dorsal pancreatic bud forms distal part of main pancreatic duct.
- proximal part of dorsal pancreatic duct forms accessory pancreatic duct.

Congenital anomalies:

Annular pancreas: ring of pancreatic tissue encircles 2nd part of duodenum

Accessory pancreatic tissue: may be found in - stomach wall

- duodenum
- meckel's diverticulum
- hilum of spleen
- small intestine
- greater omentum

→ yellowish, lobulated appearance.

Pancreatic divisum:

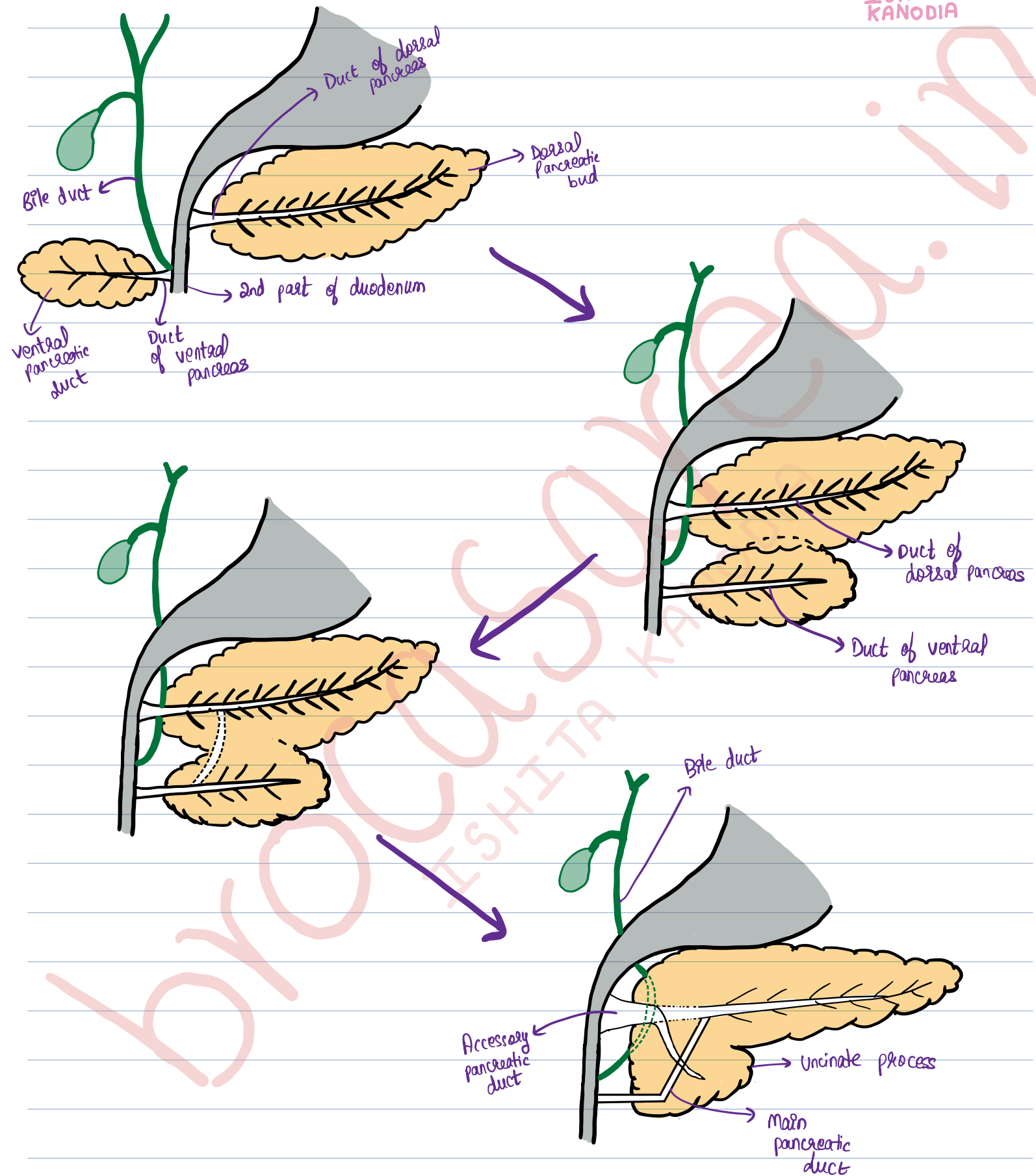
m/c anomaly in development of pancreas
ventral & dorsal duct remain separated

Dorsal duct $\xrightarrow{\text{opens}}$ into minor duodenal papilla

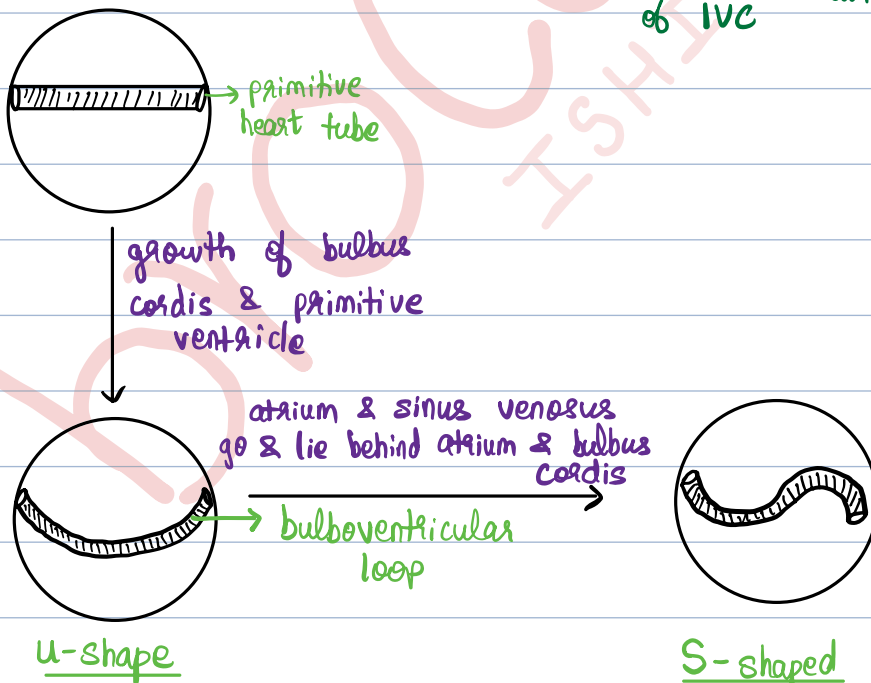
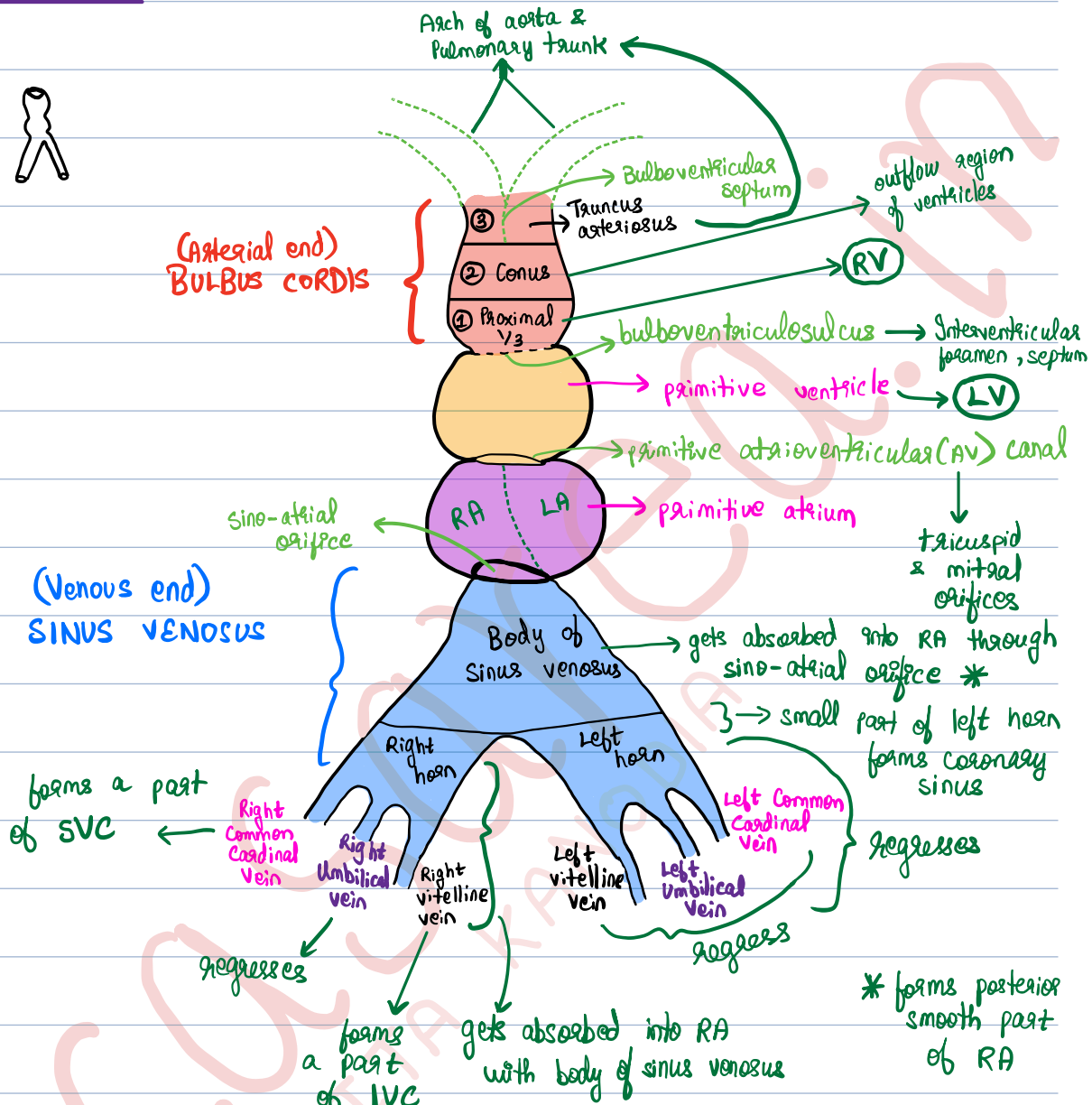
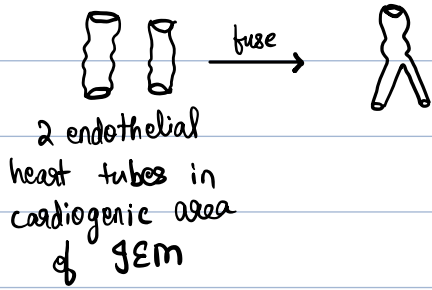
ventral duct $\rightarrow$ major duodenal papilla

Clinical Correlate

A defect in the rotation and fusion of the ventral and dorsal buds results in an **annular pancreas** which can **constrict or obstruct the duodenum** and result in **polyhydramnios**.

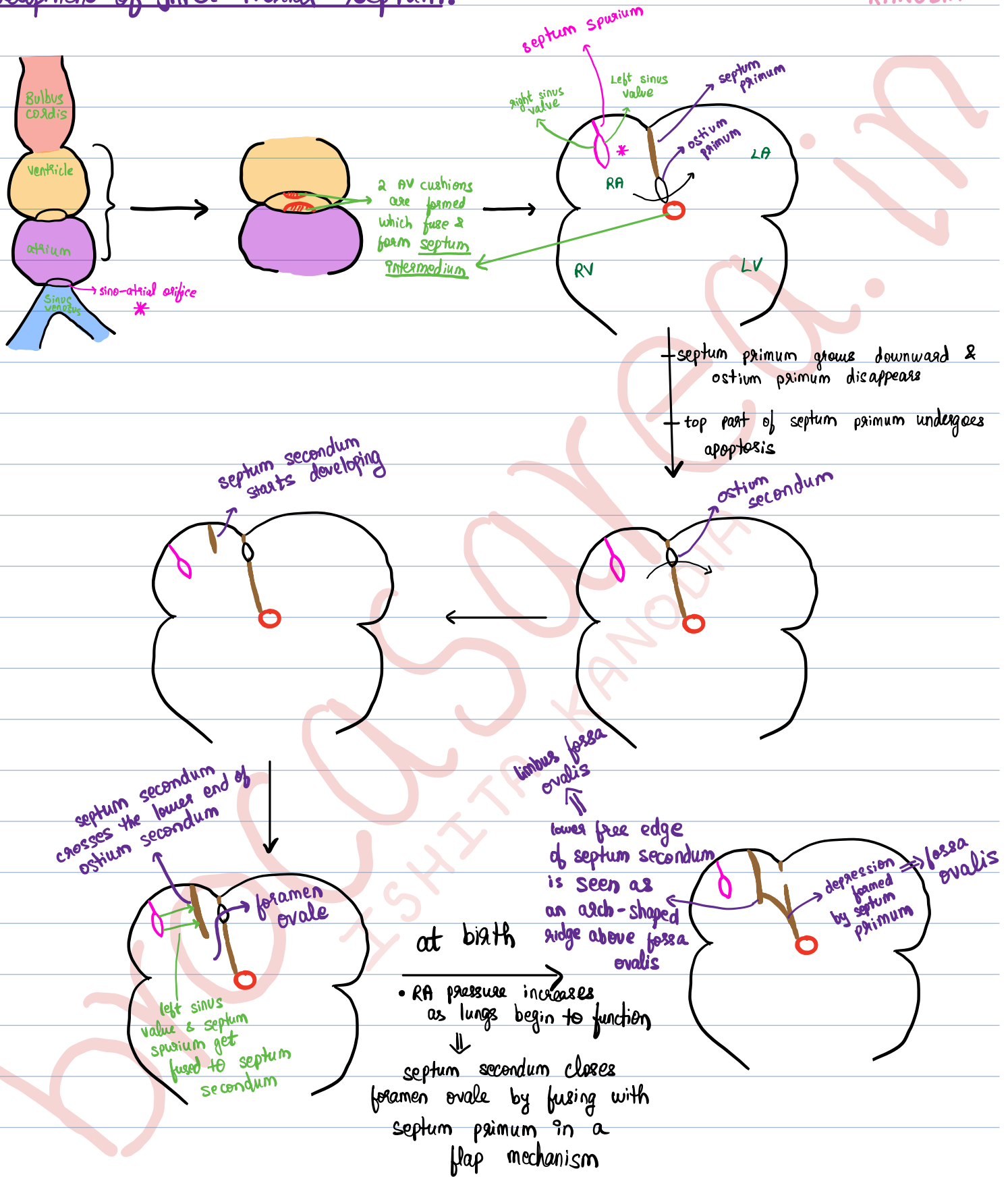


Development of Heart Tube:



Development of Inter-Atrial Septum:

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Ostium primum defect

Ostium secundum defect

Patent foramen ovale

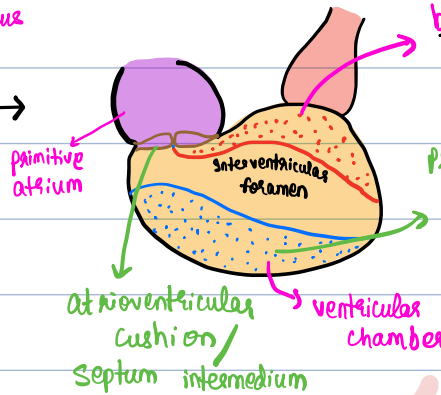
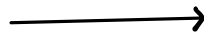
Prop-patency of foramen ovale → does not permit shunting of blood ∴ not dangerous.

Development of Interventricular Septum:

ISHITA KANODIA

↳ 3 sources

membranous part → from AV cushion & part of bulbar septum
muscular part → from ventricular muscle floor.



develops in truncus arteriosus to form separation b/w pulmonary trunk & aorta

bulbar septum forms upper portion of interventricular septum

proliferation of floor of ventricular chamber forms muscular part of interventricular septum

Interventricular foramen is filled by cells from the AV cushion & gets completed

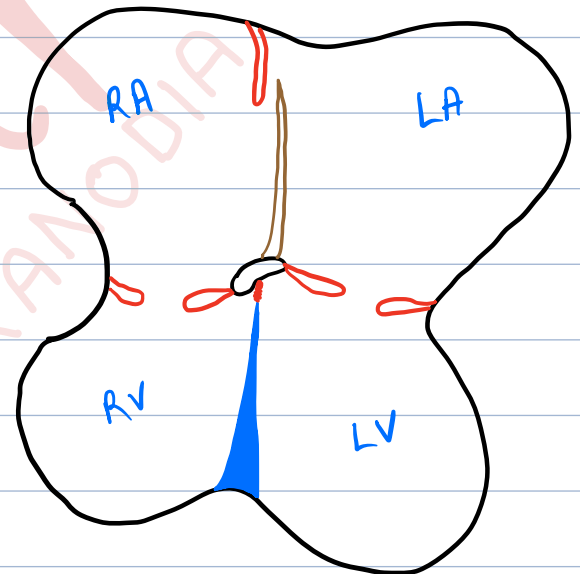
Ventricular Septal Defect (VSD): can affect muscular (mostly) or membranous (more dangerous) part

Postnatal Shunts

Right-to-left shunts are cyanotic conditions. Left-to-right shunts are non-cyanotic conditions.

Tetralogy of Fallot:

- pulmonary stenosis
- VSD
- Right ventricular hypertrophy
- Overriding of aorta



Clinical Correlate

Ventricular septal defect (VSD) is the most common of the congenital heart defects, and more common in males than in females. The most common form is a **membranous VSD**, associated with the failure of **neural crest** cells to migrate into the endocardial cushions. A membranous VSD is caused by the failure of the membranous interventricular (IV) septum to develop, and it results in **left-to-right shunting** of blood through the IV foramen.

Patients with left-to-right shunting complain of **excessive fatigue upon exertion**. Left-to-right shunting of blood is **noncyanotic** but causes increased blood flow and pressure to the lungs (pulmonary hypertension). Pulmonary hypertension causes marked proliferation of the tunica intima and media of pulmonary muscular arteries and arterioles. Ultimately, the pulmonary resistance becomes higher than systemic resistance and causes **right-to-left shunting** of blood and late **cyanosis**. At this stage, the condition is called **Eisenmenger complex**.

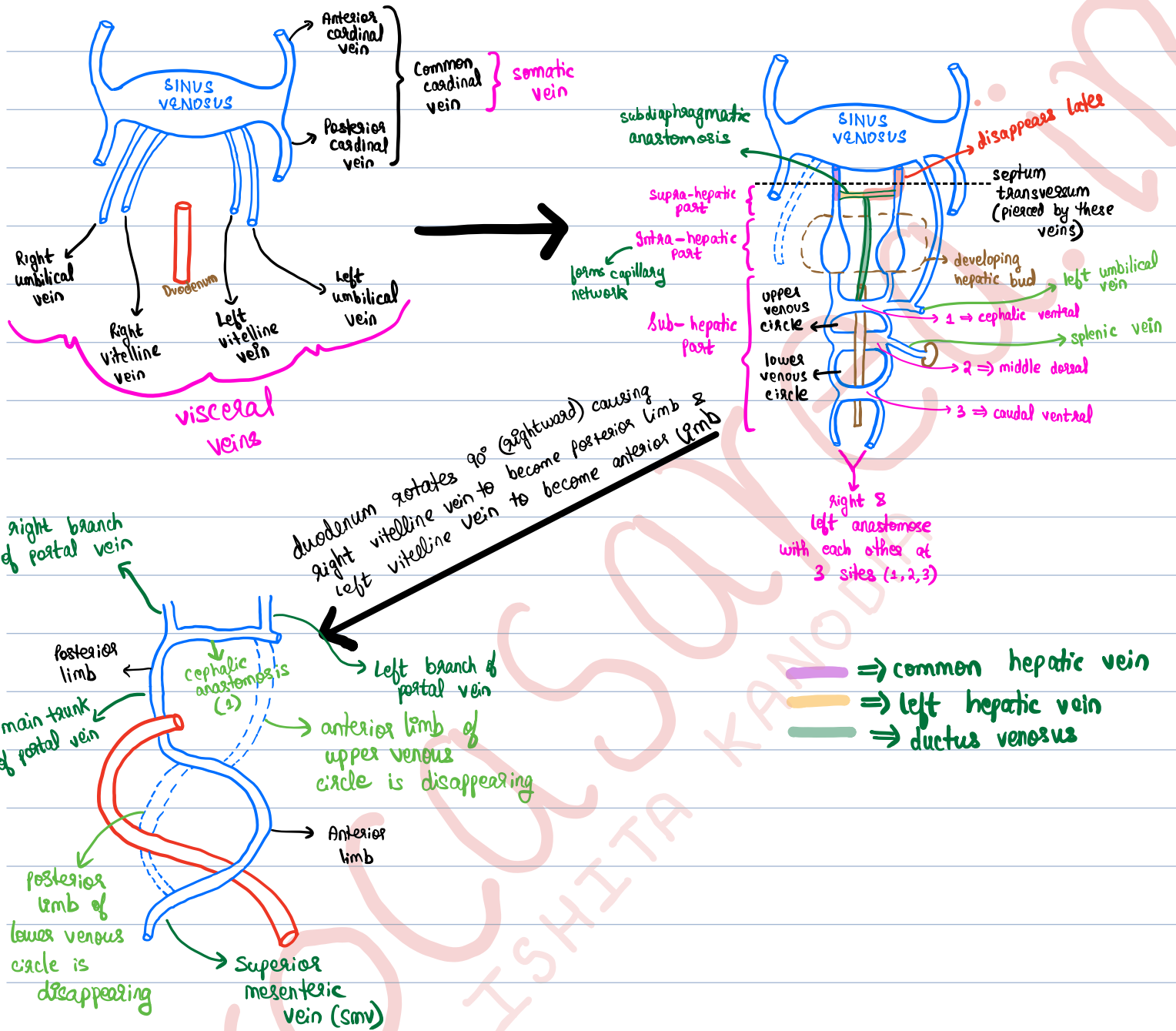
Non-cyanotic congenital (left-to-right) heart defects at birth:

- Atrial septal defect
- Ventricular septal defects
- Patent ductus arteriosus

Cyanotic congenital (right-to-left) heart defects at birth:

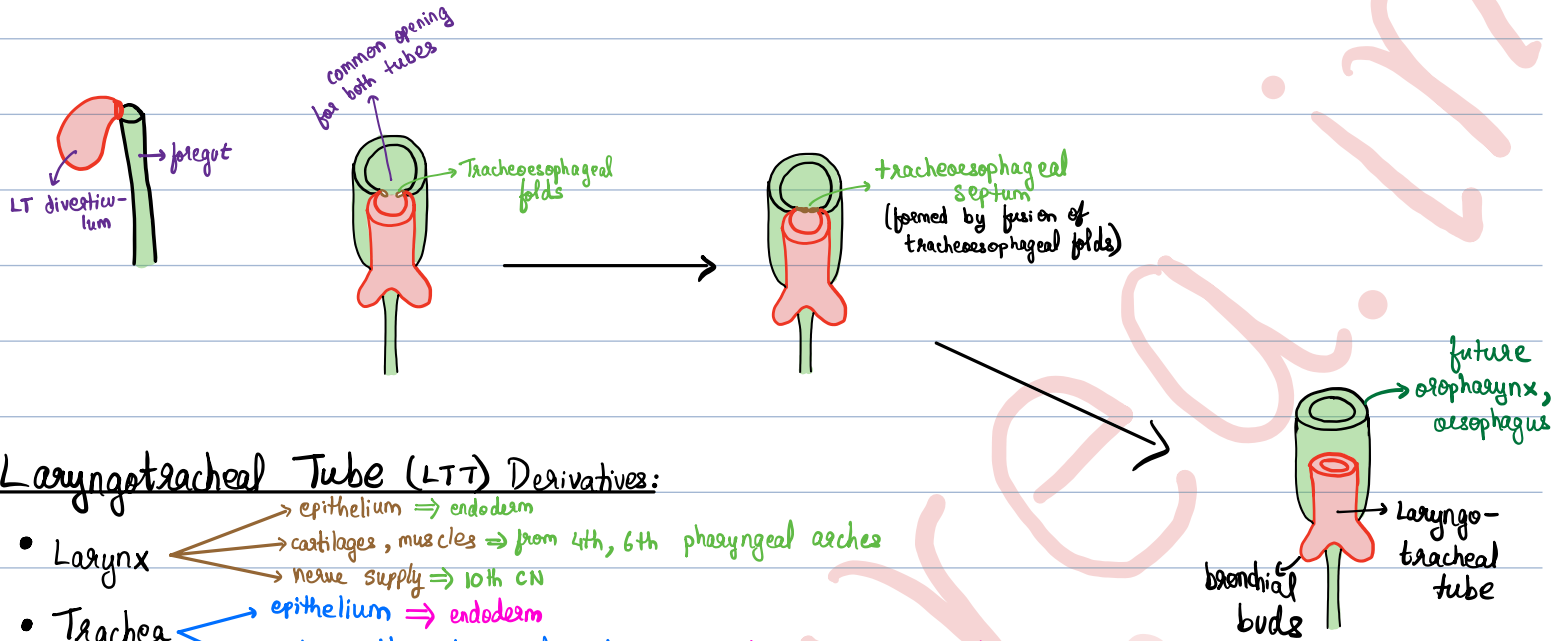
- Transposition of great vessels
- Tetralogy of Fallot
- Persistent truncus arteriosus

Development of Portal Vein:



Development of Respiratory System:

→ Laryngotracheal diverticulum develops from ventral part of foregut by end of 4th week.



Laryngotracheal Tube (LTT) Derivatives:

- Larynx**
 - epithelium $\Rightarrow$ endoderm
 - cartilages, muscles $\Rightarrow$ from 4th, 6th pharyngeal arches
 - nerve supply $\Rightarrow$ 10th CN
- Trachea**
 - epithelium $\Rightarrow$ endoderm
 - muscles, cartilages, fibroareolar stroma $\Rightarrow$ splanchnopleuric mesoderm
- Bronchi** $\rightarrow$ 2 bronchial buds from LT tube $\rightarrow$ by end of 5th week, there are 3 buds developing from right bud & 2 buds developing from left bud $\downarrow$ called **lobar bronchi**
- Lung Tissue**
 - 4th pharyngeal arch** (superior laryngeal N.)
 - thyroid
 - cuneiform
 - epiglottis
 - 6th pharyngeal arch** (recurrent laryngeal N.)
 - cricoid
 - arytenoid
 - corniculate

4 stages of maturation of lungs:

- Pseudoglandulae (6-16 weeks)** $\Rightarrow$ tubuloacinar gland
- Canaliculae (16-26 weeks)** $\Rightarrow$ lumen is enlarged until terminal bronchioles; cells which don't get differentiated form **Type II pneumocytes**.
- Terminal sac (26 - Birth)** $\Rightarrow$ establishment of blood-air barrier for proper gaseous exchange
- Alveolar stage (32 - Birth)**
 - $\Rightarrow$ alveolar sacs secrete surfactant
 - $\Rightarrow$ lung transforms from secretory phase to gas exchange phase
 - $\Rightarrow$ establishment of pulmonary & systemic circulation

$\rightarrow$ In 7th month of IUL, lung fibroblast stimulates release of FPF (fibroblast-pneumocyte factor) which in turn stimulates type II pneumocytes to secrete surfactant. (cortisol stimulates FPF)

Defects in Respiratory System:

VA C T E R L group of anomalies:

Vertebral anomalies

Anal atresia

Cardiac defects

Tracheoesophageal fistula

Esophageal fistula

Renal anomalies

Limb defects

Hyaline Membrane Disease / Respiratory Distress Syndrome:

→ absence of surfactant

→ presence of waxy lining inside alveolar sac ⇒ hyaline membrane

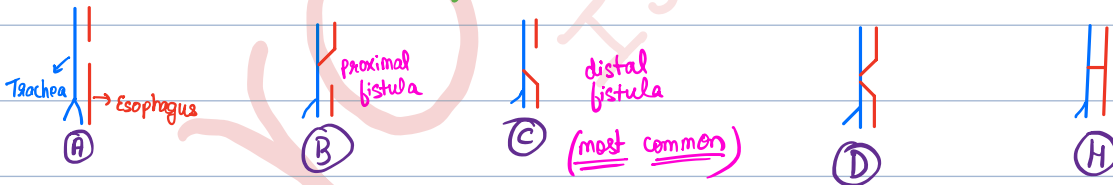
Supernumerary Lobes: extra lobes

Azygos Lobe: any increased no. of lobes in lung

Lobe of Azygos Vein: apical lobe arches over azygos vein.

Tracheoesophageal (TE) Fistula: 5 types (A, B, C, D, H)

→ defective formation of TE septum



→ Laryngeal inlet is occluded by 8th week of IUL due to proliferation of laryngeal epithelium & recanalized by 10th week resulting in formation of vestibular & vocal folds.

Clinical Correlate

Respiratory distress syndrome

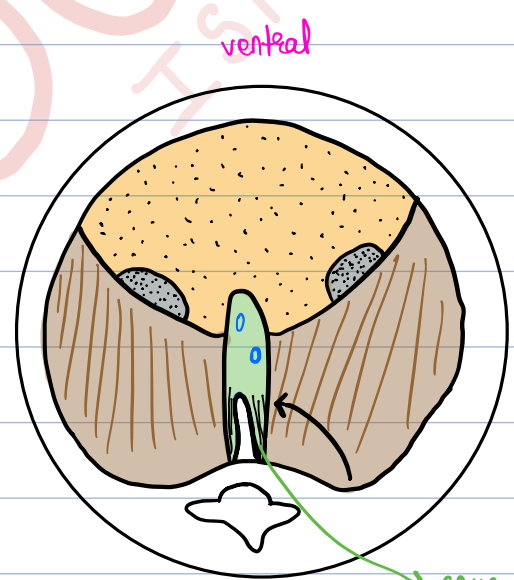
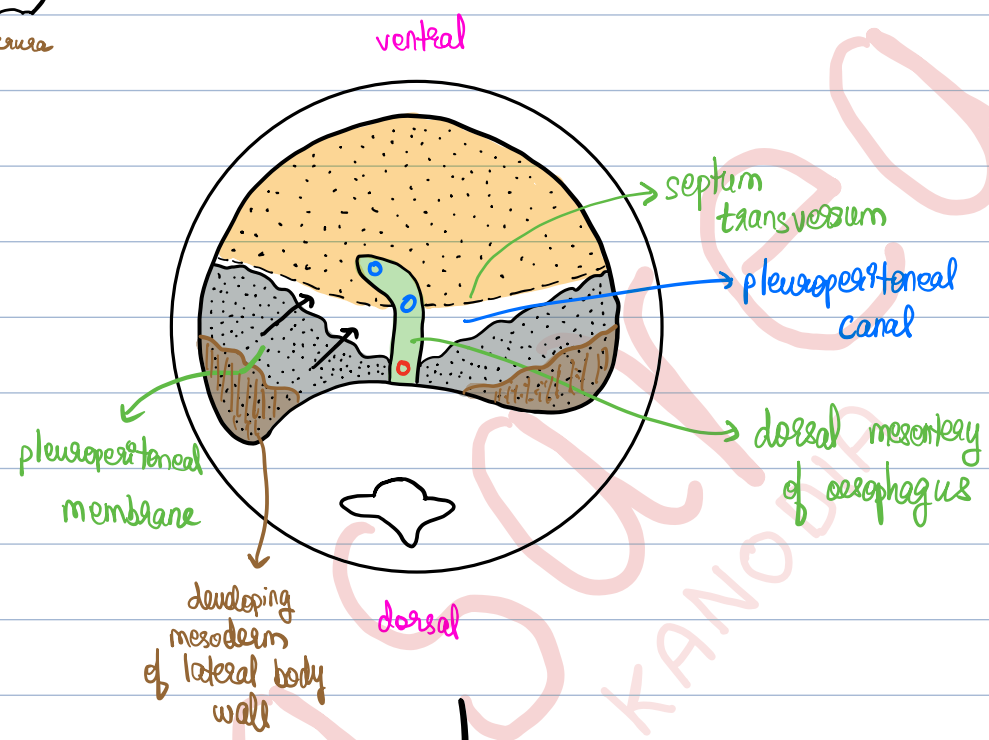
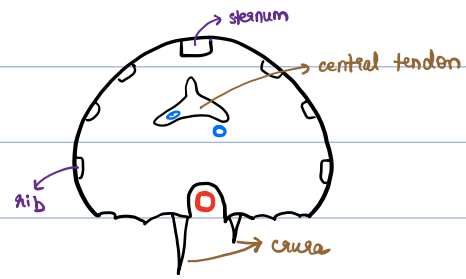
is caused by a deficiency of surfactant (type II pneumocytes).

This condition is associated with premature infants, infants of diabetic mothers, and prolonged intrauterine asphyxia. Thyroxine and cortisol treatment increase the production of surfactant.

Surfactant deficiency may lead to **hyaline membrane disease**, whereby repeated gasping inhalations damage the alveolar lining. Hyaline membrane disease is characterized histologically by collapsed alveoli (atelectasis) and eosinophilic (pink) fluid covering the alveoli.

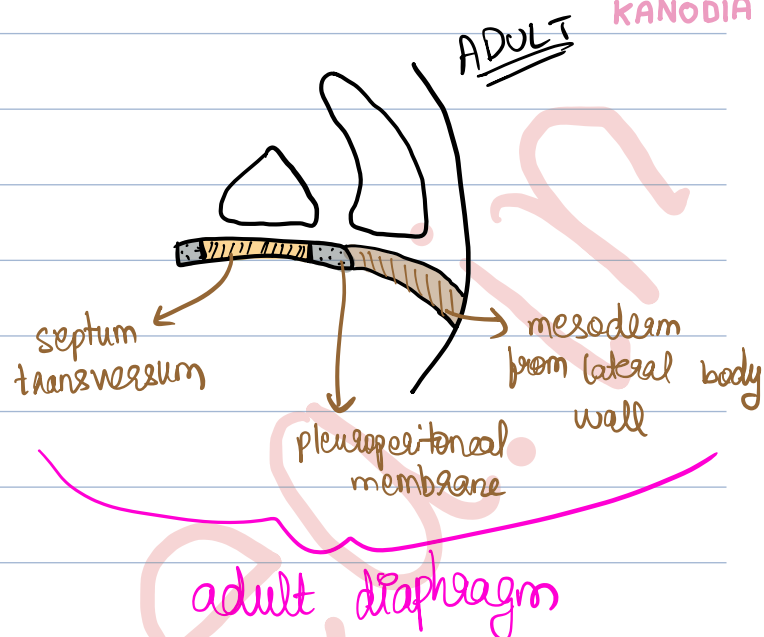
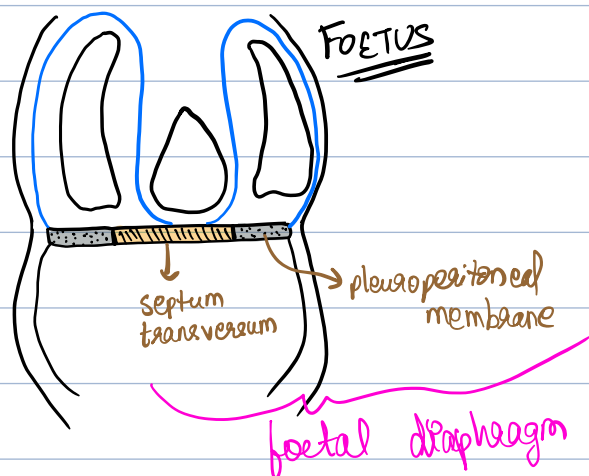
Development of Diaphragm: 4 sources $\Rightarrow$ septum transversum

- $\Rightarrow$ dorsal mesentery of oesophagus
- $\Rightarrow$ pleuroperitoneal membrane
- $\Rightarrow$ mesoderm of lateral body wall.



Genes on long arm of chromosome 15 are said to have a vital role in development of diaphragm.

$\Rightarrow$ caudal is formed majorly by invasion of myoblasts from lateral body wall into dorsal mesentery of oesophagus



Motor Supply: Phrenic N. (C3-C5)

Sensory Supply: Lower intercostal Ns.

Congenital Diaphragmatic Hernia (Bochdalek Hernia)

- contents of abdomen enter into thoracic cavity
- ∴ hypoplasia of lung
- most commonly seen on left side (∵ pleuroperitoneal membrane on right side closes first, followed by fusion on left side)

Retrosternal Hernia of Morgagni:

- defect in anterior portion of diaphragm
- herniation of abdomen contents into thoracic cavity.

Congenital Hiatal Hernia:

- 3 structures pierce the diaphragm — IVC, aorta, oesophagus
- opening of oesophagus is larger than normal
- ∴ oesophagus tries to drag stomach lying below

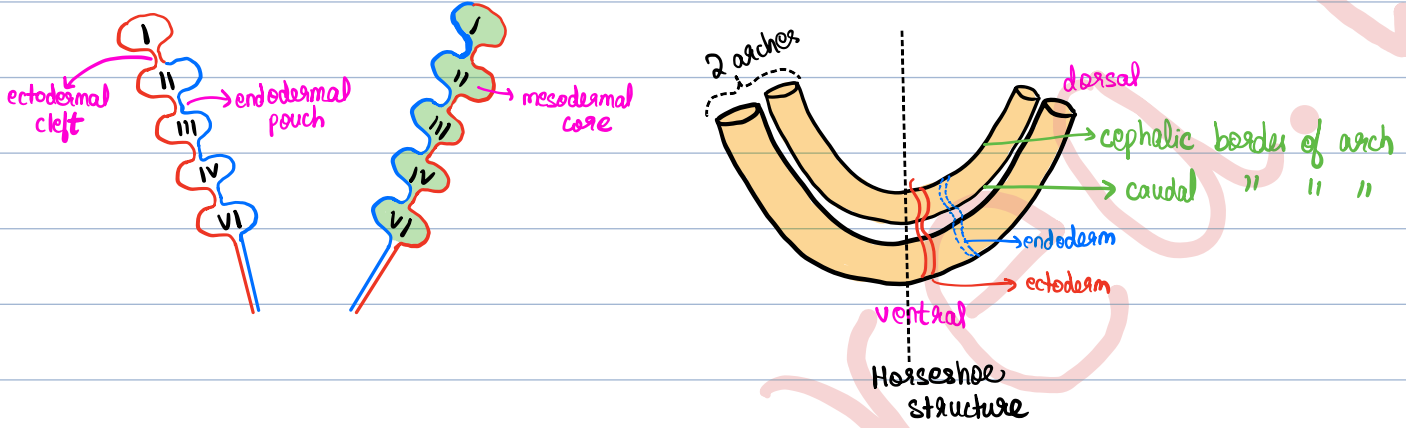
Eventration of Diaphragm: one half of diaphragm is thinned out (muscular portion absent, only membranous portion present); most commonly seen on left side; contents of abdomen push upwards.

Pharyngeal / Branchial Apparatus: → seen on ventral aspect

→ Pharyngeal apparatus has an ectodermal lining, endodermal lining & mesodermal core

→ Pharyngeal arch refers to the mesodermal core.

→ 6 arches are present in neck region (5th arch disappears later)

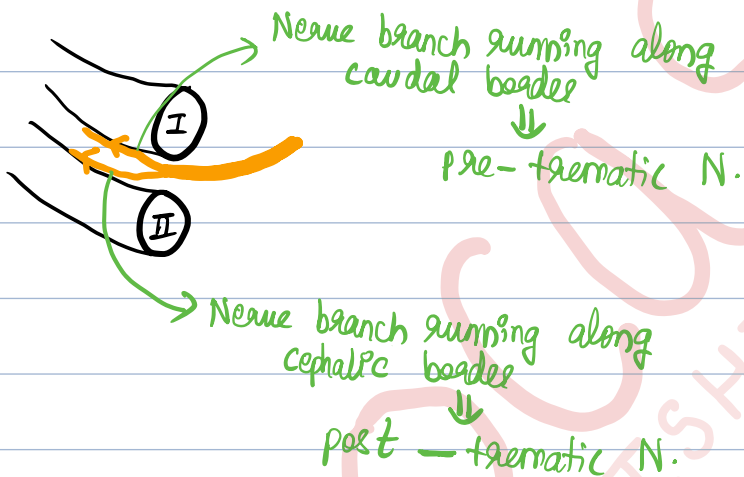


Arch	Skeletal Derivatives
I	Maxillary process - upper lip, upper jaw, palate Mandibular process - meckel's cartilage → malleus, incus, body of mandible ↳ ant. lig. of malleus, sphenomandibular ligament.
II	Reichert's cartilage → stapes, styloid process, lesser cornu & upper part of body of hyoid bone ↳ styloid hyoid lig.
III	Greater cornu & lower part of body of hyoid bone
IV	Thyroid, cuneiform cartilages
VI	Cricoid, Arytenoid, Corniculate cartilages.

Arch	Muscles
I	- Muscles of mastication - Tensor tympani - Myelohyoid - Tensor veli palatini - Ant. belly of digastric MT ATM
II	- Facial muscles - Post. belly of digastric - Stylohyoid - Stapedius - Auricular muscles - Occipitalis Facial PSOAS
III	Stylopharyngeus
IV	- Cricothyroid - Muscles of palate (except tensor veli palatini) - Muscles of pharynx (except stylopharyngeus & cricopharyngeus) MMC
VI	- Cricopharyngeus - All muscles of larynx (except cricothyroid) CA

Arch	Vessels
I	Maxillary artery
II	Stapedial artery
III	Common carotid artery
IV	Right subclavian artery
VI	- Pulmonary artery (right & left) - Hyoid artery - Internal Carotid Artery - Arch of Aorta - Ductus arteriosus

Arch	Nerve Supply
I	Post-trigeminal N. $\Rightarrow$ Mandibular N. (V_3) Pre-trigeminal N. $\Rightarrow$ Chorda tympani
II	Facial N. (VII)
III	Glossopharyngeal N. (IX)
IV	External laryngeal N. $\rightarrow$ branch of superior laryngeal N. branch of CN X $\leftarrow$
VI	Recurrent laryngeal N. $\rightarrow$ branch of CN X.



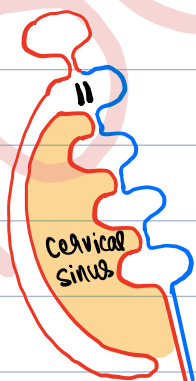
$\rightarrow$ In humans, all pre-trigeminal Ns. degenerate except that of I arch

Fate of Endodermal Pouches:

- I** $\Rightarrow$ • Inner layer of tympanic membrane • Middle ear cavity
• Mastoid antrum • Auditory tube
- II** $\Rightarrow$ • Palatine tonsil (lining epithelium)
- III** $\Rightarrow$ • Thymus • Inferior parathyroid gland
- IV** $\Rightarrow$ • Superior parathyroid gland
- Ultimobranchial body** $\Rightarrow$ remnant of pouch b/w (rudimentary) 5th arch & 6th arch
 $\hookrightarrow$ Neural crest cells (NCC) migrate into ultimobranchial body & give rise to parafollicular C cells of thyroid gland.

Fate of Ectodermal Clefts:

- I** $\Rightarrow$ • Lining of external auditory canal
• Outer layer of tympanic membrane
- II, III, IV** $\Rightarrow$ get obliterated due to overshoot of Arch II



Clinical Correlate

Normally, the second, third, and fourth pharyngeal grooves are obliterated by overgrowth of the second pharyngeal arch. Failure of a cleft to be completely obliterated results in a branchial cyst or lateral cervical cyst.

$\rightarrow$ Cervical sinus eventually gets obliterated.

$\rightarrow$ If it persists

$\Rightarrow$ Branchial cysts

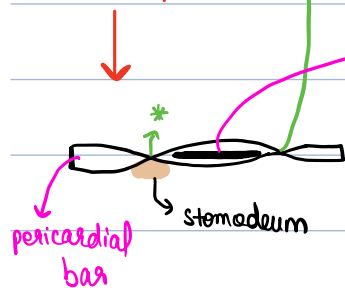
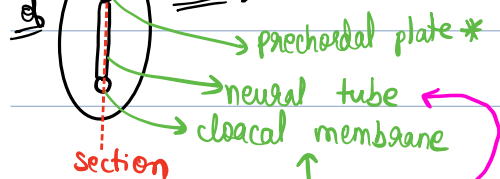
$\rightarrow$ seen as swelling along anterior border or beneath sternocleidomastoid

Development of Face: 3 sources: $\Rightarrow$ frontonasal process (FNP)

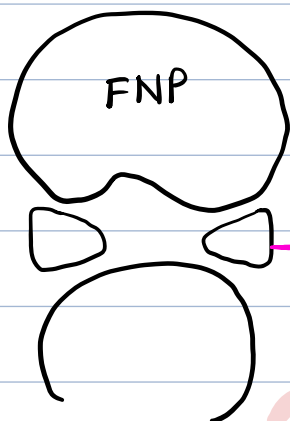
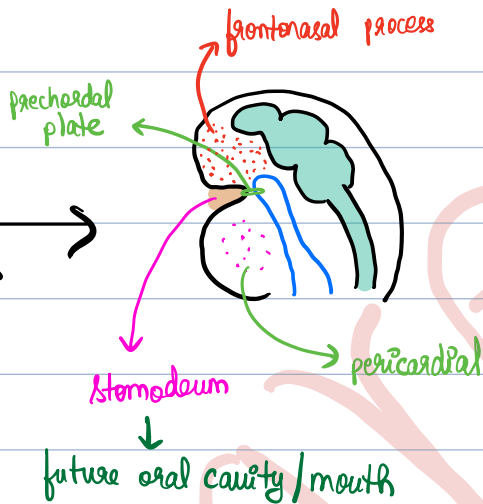
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At the end of
3rd week
of IUL,

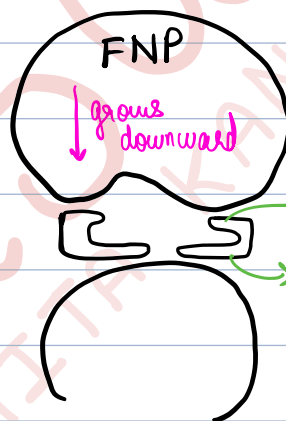
$\Rightarrow$ 1 arch $\begin{cases} \text{maxillary process} \\ \text{mandibular process} \end{cases}$



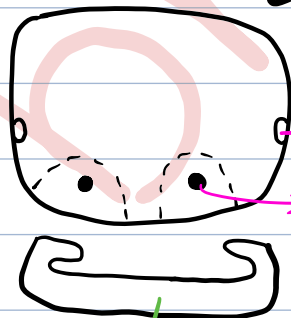
folding
begins



1st arch



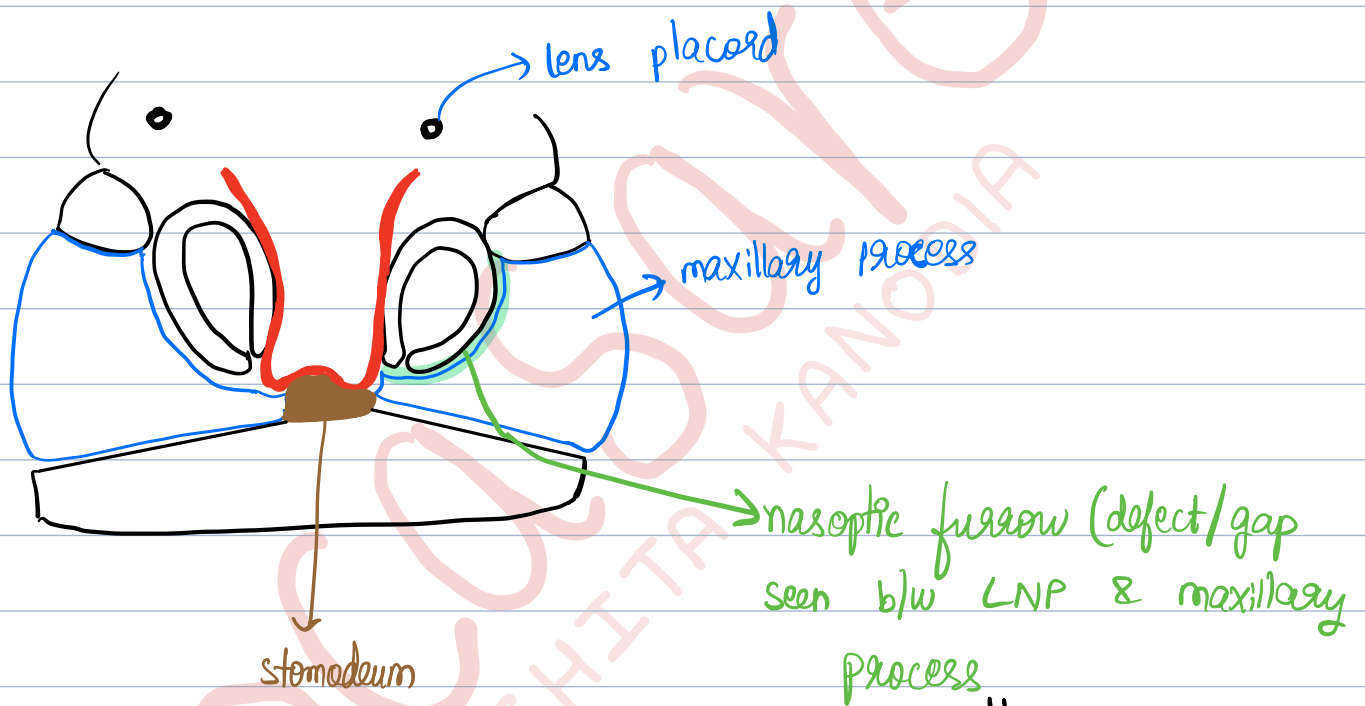
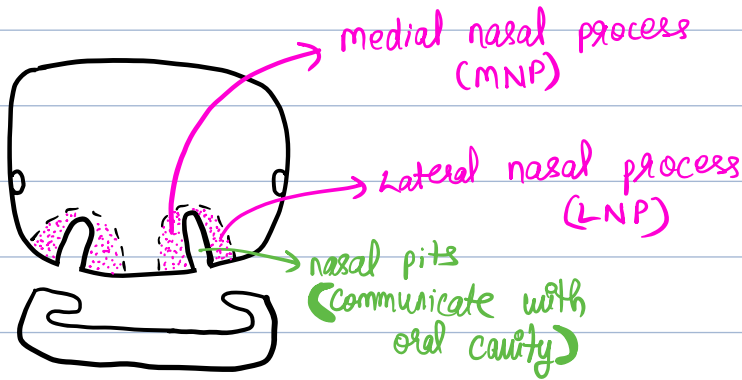
maxillary process
mandibular process



lens placoid

thickening formed in FNP = nasal placoid

mandibular processes approximate & fuse



Lower lip → formed by → Mandibular processes

Upper lip → MNP + maxillary processes

philtrum of lip

filled by ectodermal cells to form a cord which extends from eye to nose

cord is later canalised to form nasolacrimal duct

Forehead → upper part of FNP

Cheek → maxillary process

ala of nose $\rightarrow$ LNP

tip of nose $\rightarrow$ MNP

Facial Cleft: non-fusion of maxillary process & LNP
(oblique cleft)

$\rightarrow$ nasolacrimal duct is exposed

Macrostomia: large oral cavity/mouth

Microstomia: smaller oral cavity/mouth

Midline cleft Lower Lip: non-fusion of both mandibular processes

Midline cleft Upper Lip: non-fusion of both MNPs

Clinical Correlate

Robin sequence presents with a triad of poor mandibular growth, cleft palate, and a posteriorly placed tongue.

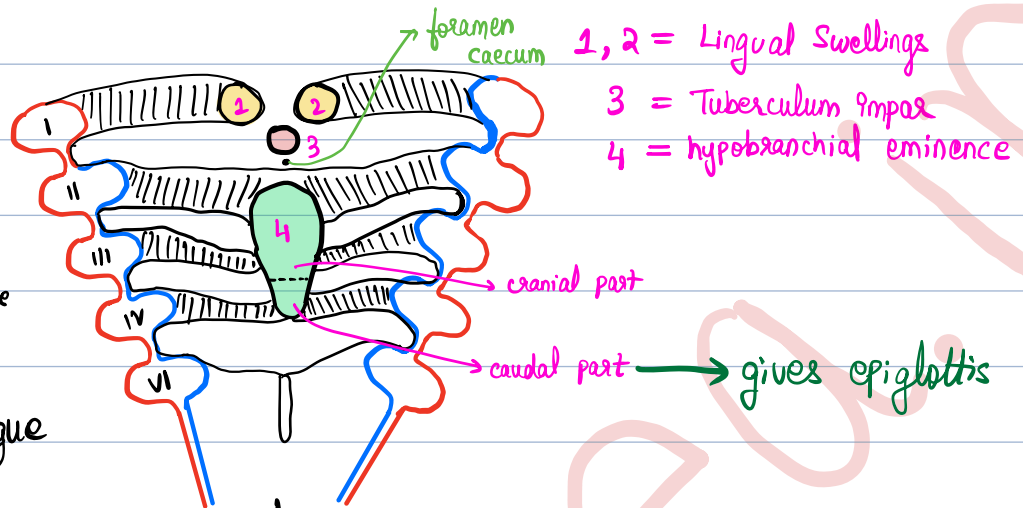
Treacher Collins syndrome also presents with mandibular hypoplasia, zygomatic hypoplasia, down-slanted palpebral fissures, colobomas, and malformed ears.

Development of Tongue:

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View of posterior aspect of pharyngeal arches

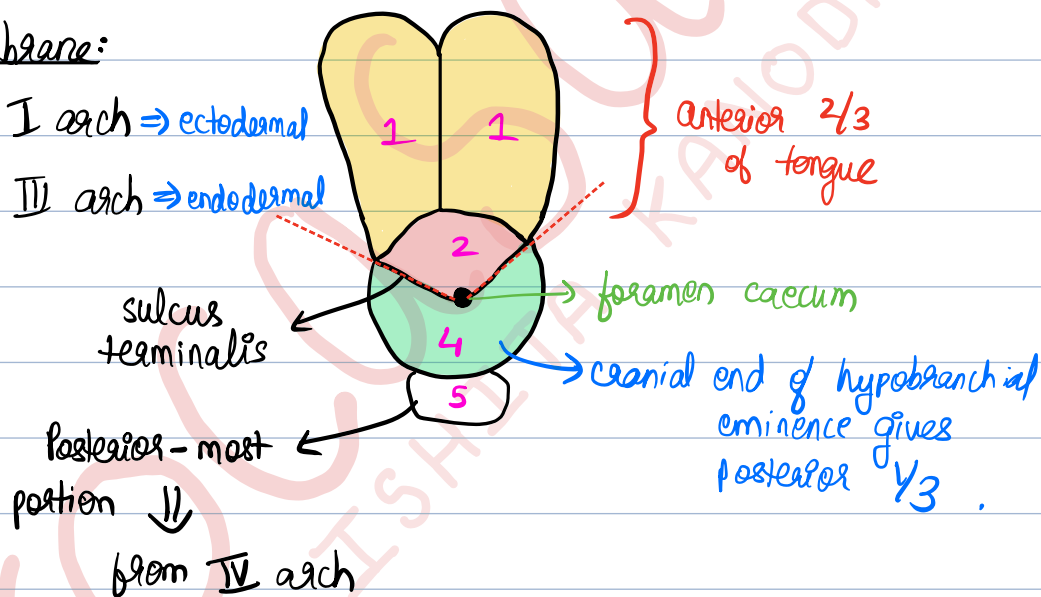
Tongue $\left\{ \begin{array}{l} \text{mucous membrane} \\ \text{fibrous stroma} \\ \text{muscles of tongue} \end{array} \right.$



mucous membrane:

Ant $\frac{2}{3} \Rightarrow$ I arch $\Rightarrow$ ectodermal

Post $\frac{1}{3} \Rightarrow$ III arch $\Rightarrow$ endodermal



Muscles of Tongue: derived from occipital somites

(except palatoglossus

$\Rightarrow$ from IV arch)

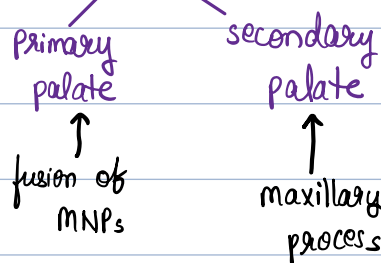
Hemiglossia: in anterior $\frac{2}{3}$, only one half of the tongue is developed

$\hookrightarrow$ due to absence of a lingual swelling

Aglossia: absence of tongue

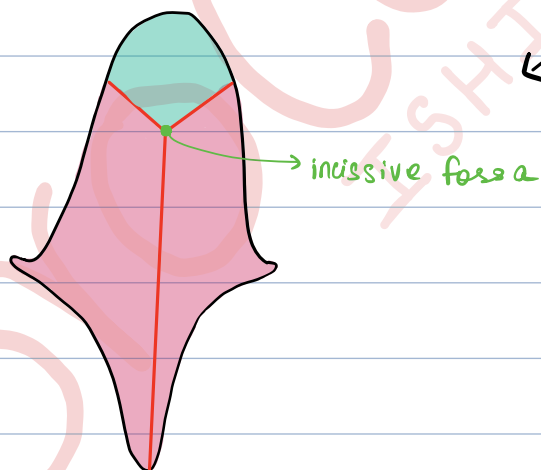
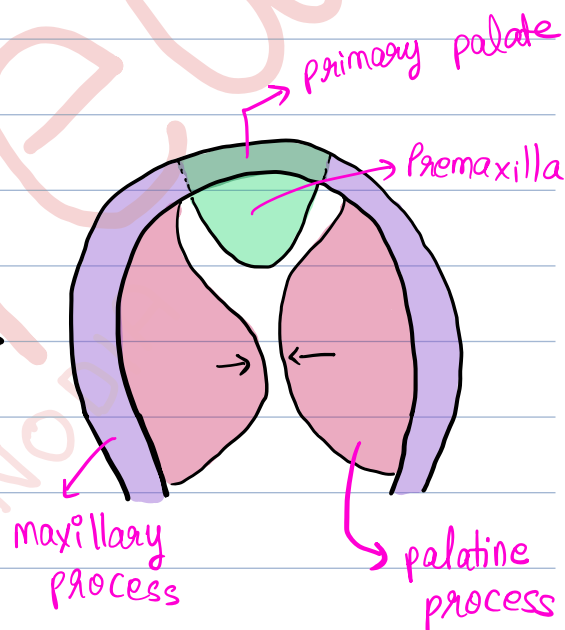
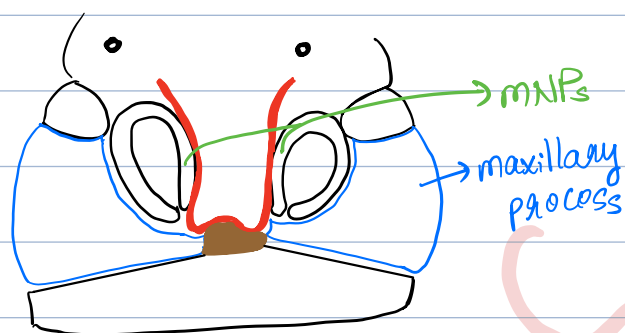
Development of Palate:

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Premaxilla: posterior elongation of primary palate

Palatine process: develops from maxillary processes



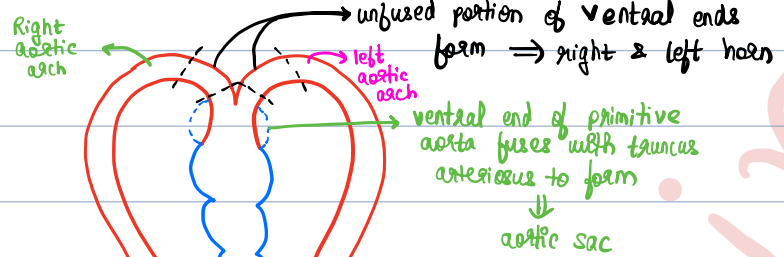
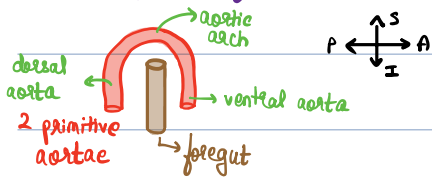
Complete Cleft Palate: non-fusion of both palatine process with each other & with premaxilla

Anterior cleft Palate: non-fusion of premaxilla with palatine processes

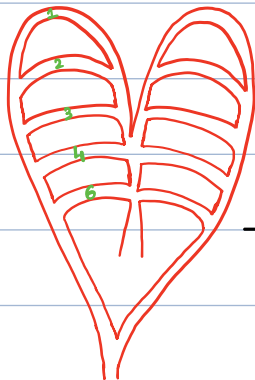
Posterior cleft Palate: non-fusion of both palatine process to each other.

Development of Arches of Aorta:

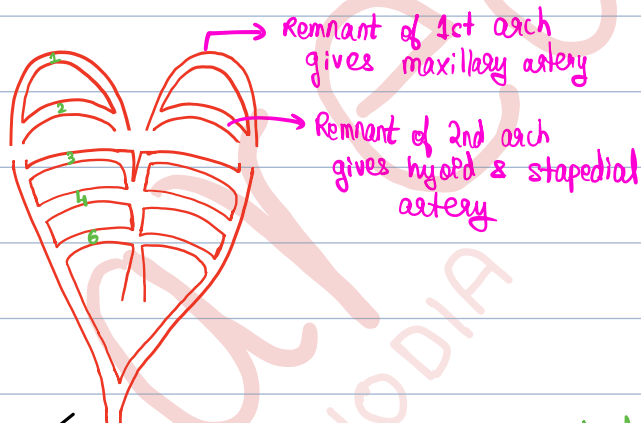
ISHITA KANODIA



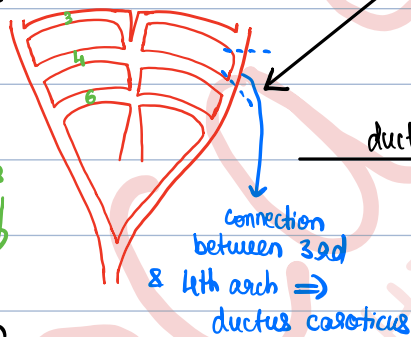
There is one aortic arch for each pharyngeal arch



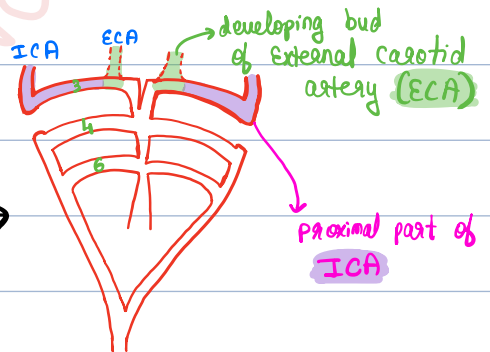
1st & 2nd arch disappear



- carries blood from ascending aorta
- 3rd & 4th aortic arches open into ventral part of aortic sac
- 6th aortic arch opens into dorsal part of aortic sac.
- carries blood from pulmonary trunk

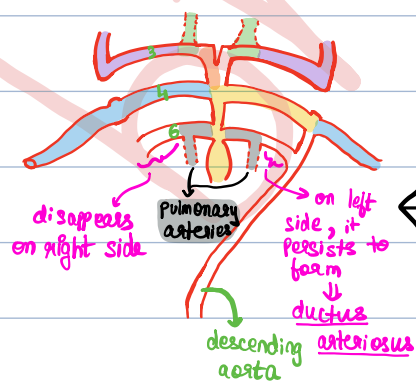


ductus caroticus disappears



formation of 7th intersegmental artery *

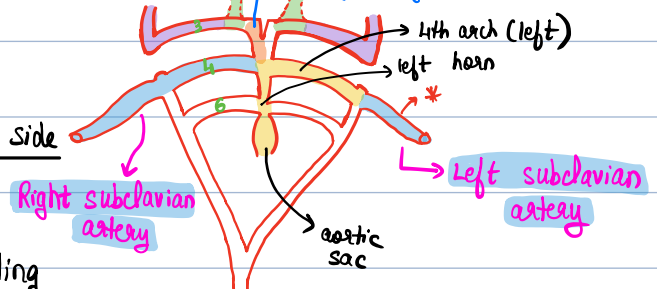
Brachiocephalic trunk (from right horn)



- dorsal aorta on right side disappears
- dorsal aorta on left side gives => descending aorta

Arch of aorta

{ Aortic Sac + Left horn + 4th arch (left)



Patent Ductus Arteriosus: persistent ductus arteriosus

Coarctation of Aorta: narrowing of aorta

↳ due to migration of cells from ductus arteriosus

Development of Kidney:

→ Nephrogenic cord develops from intermediate mesoderm (IM) which gives the Kidneys

- Pronephros ⇒ fish kidneys
- Mesonephros ⇒ kidney of higher vertebrates
- Metanephros ⇒ human kidneys

→ 3 parts are derived from nephrogenic cord

Pronephros ⇒ situated in cervical region ⇒ many ductules develop from pronephros to form a duct connected to cloaca

Mesonephros ⇒ thoraco-lumbar region

↓
following this, pronephros degenerates.

Metanephros ⇒ sacral region

Cloaca { primitive rectum
urogenital sinus ⇒ urinary bladder

[Mesonephric duct = Wolffian duct]

↓
mesonephros gives many ductules which join the pronephric duct, now called mesonephric duct

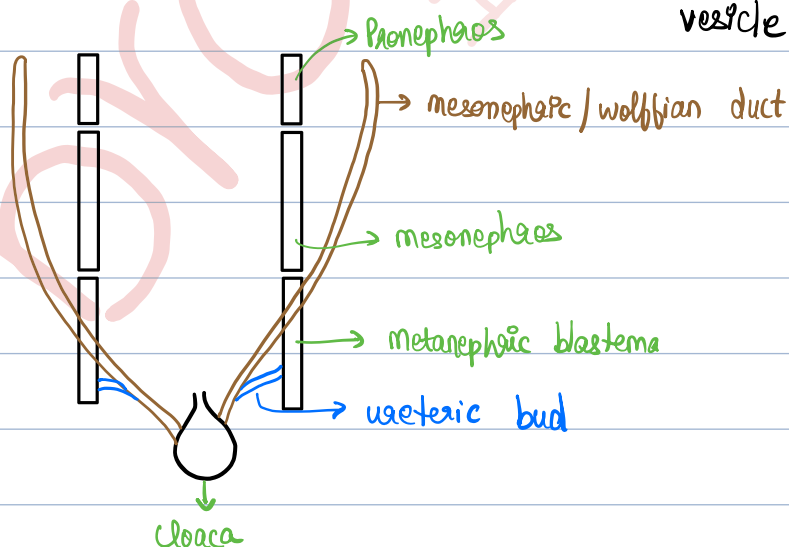
→ a bud arises from mesonephric duct called ureteric bud

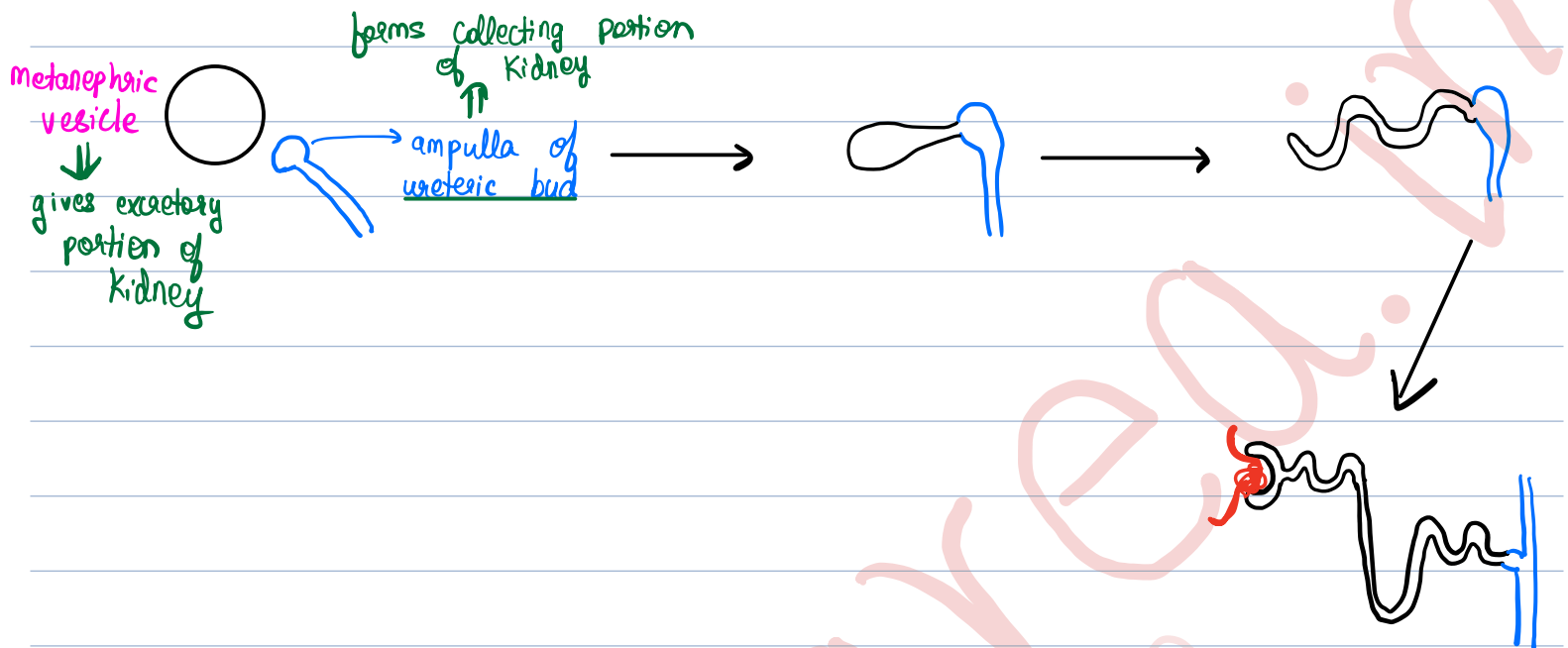
↓
mesonephric duct & ductules

→ metanephros $\xrightarrow{\text{gives}}$ metanephric blastema

regress in females, but persist in males (towards the end of development)

metanephric vesicle





Ascent of Kidneys (From Sacral to Lumbar Region):

- when in sacral region $\Rightarrow$ blood supply from lateral sacral vessels.
- when in lumbar " $\Rightarrow$ " " " renal vessels.

$\rightarrow$ originally, hilum of kidney faces anteriorly, which undergoes rotation to face medially

Renal Agenesis: non-development of kidney (unilaterally or bilaterally)

Horseshoe Kidney: sometimes lower poles of both kidneys fuse

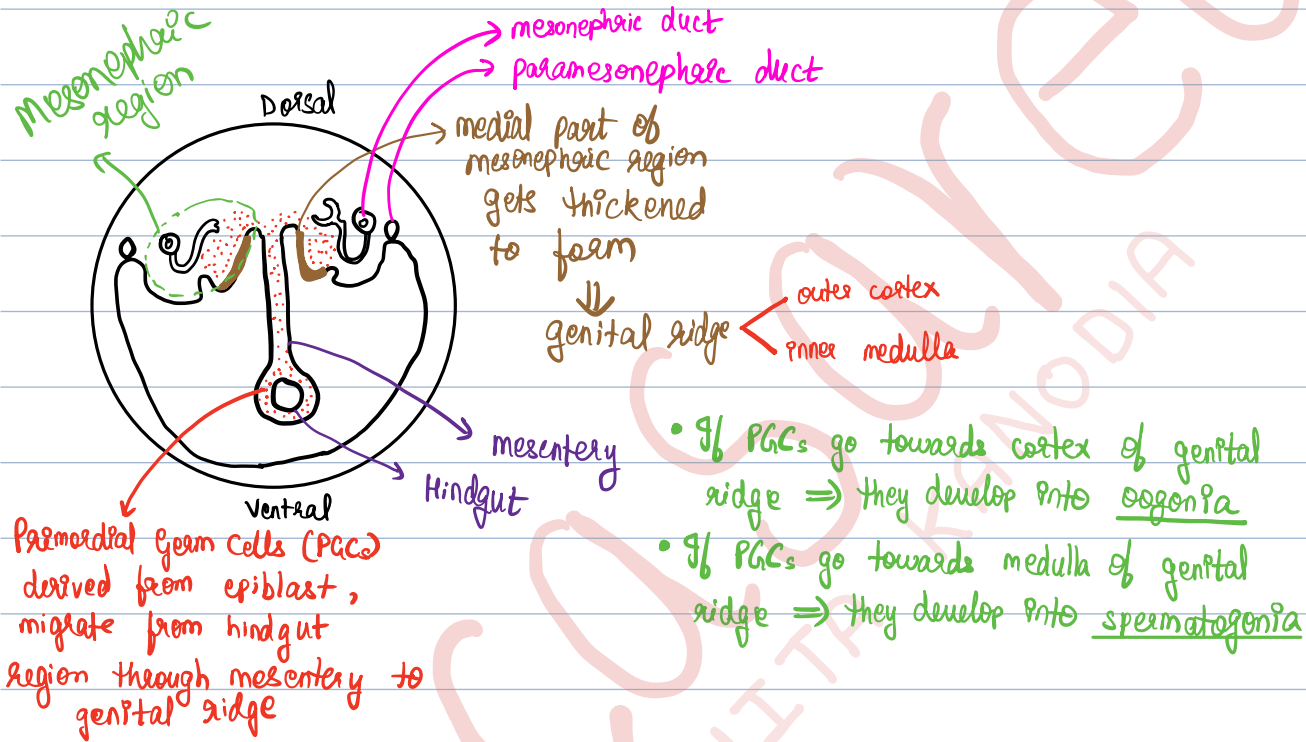
$\hookrightarrow$ impairs complete ascent of kidneys

Polycystic Kidney: non-fusion of metanephric vesicle with ampulla of ureteric bud

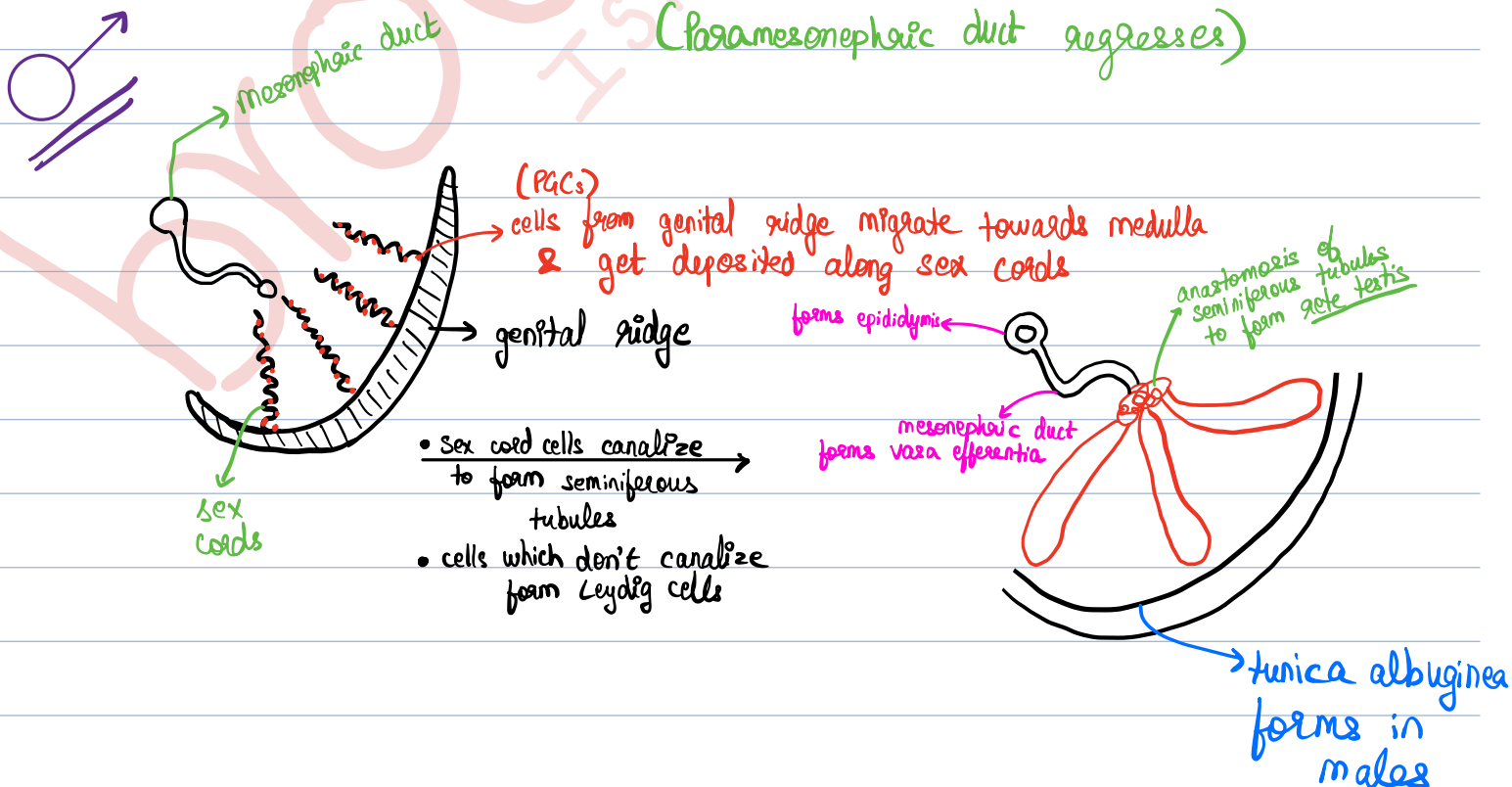
$\therefore$ excretory part & collecting part of kidney remain separate.

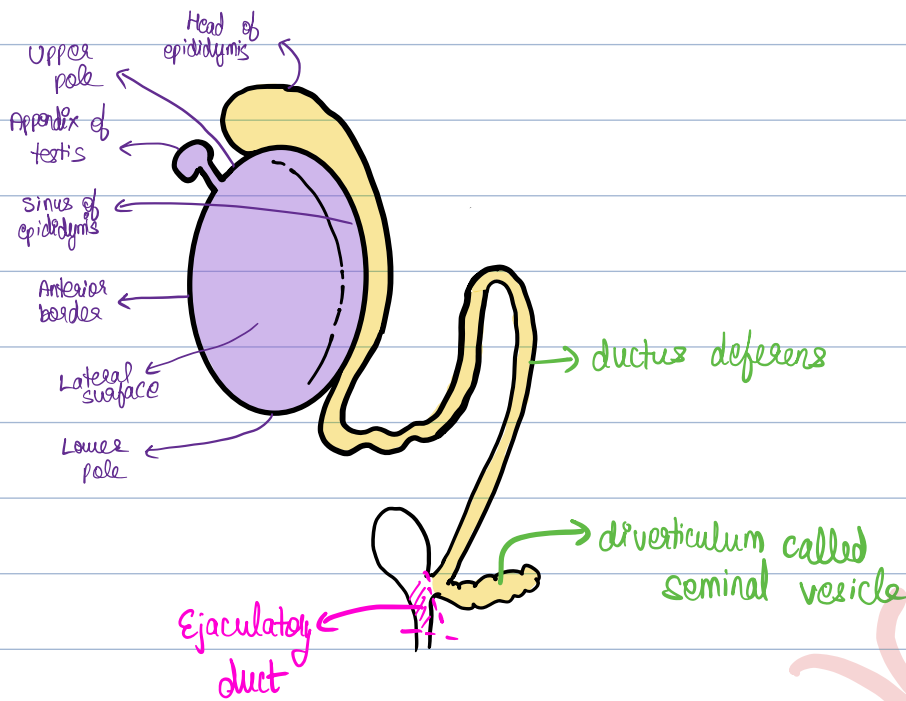
Development of Gonads:

- SRY gene: induces the formation of Leydig cells (from mesenchymal cells) & Sertoli cells (from sex cord cells) at 7th week of IUL.
- Leydig cells secrete testosterone which induces formation of male gonads
↳ they disappear at 18 weeks of IUL & reappear at puberty.
- Sertoli cells release Mullerian-inhibiting substance (MIS) which inhibits Mullerian duct system.



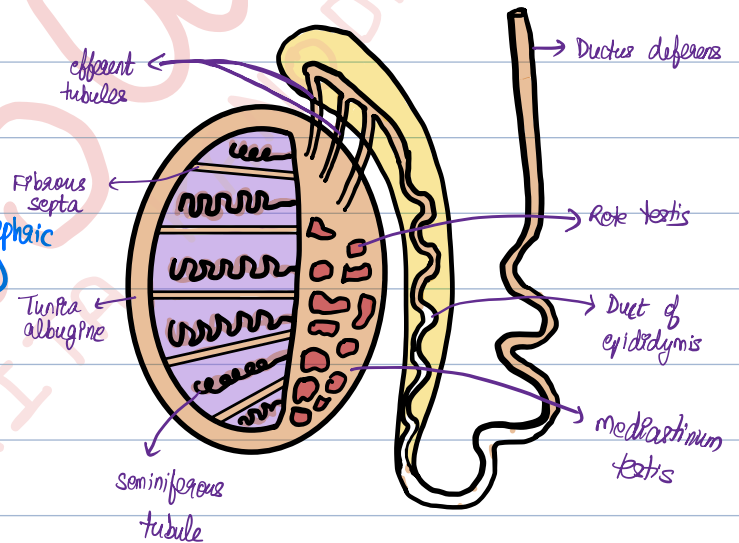
(Paramesonephric duct regresses)





Remnants of mesonephric duct:

- appendix of epididymis
- paradidymis
- appendix of testis ($\Rightarrow$ remnant of paramesonephric duct)
- superior aberrant ductules
- inferior aberrant ductules



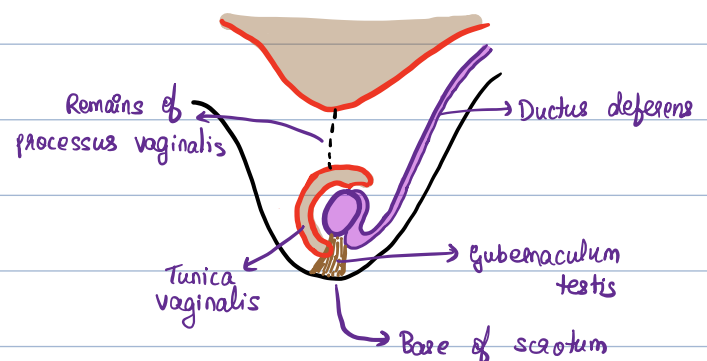
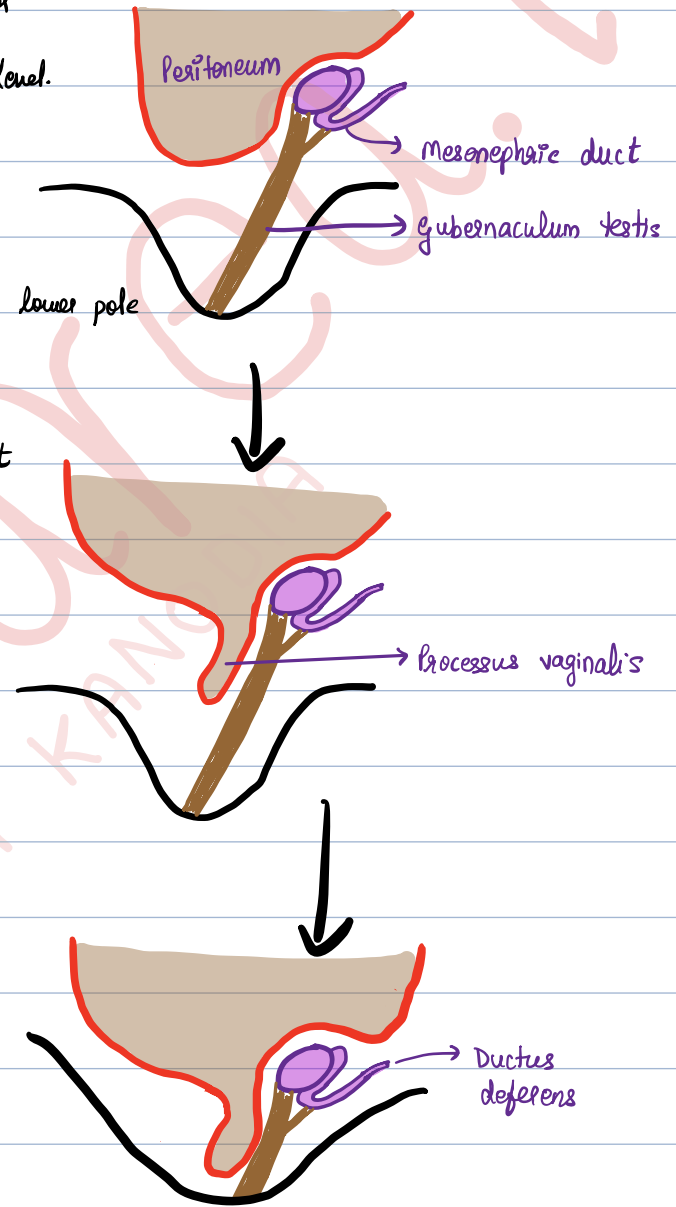
Descent of Testis: testis develop in abdominal cavity where temperature is high & not suitable for proper spermatogenesis.

- developing testis initially lies retroperitoneally on posterior abdominal wall on medial side of mesonephros at L2/3 level.
- a fold of peritoneum called processus vaginalis extends in front of testis & reaches scrotum.
- gubernaculum testis develops & connects mesonephric duct & lower pole of testis to base of scrotum
- differential body growth in embryo & fetus results in descent of testis into scrotum.
- Testis begin to descend in 2nd month of IUL.

- reaches iliac fossa at 3rd month
- rests at deep inguinal ring from 4th-6th month
- traverses inguinal canal during 7th month
- reaches superficial inguinal ring by 8th month
- enters scrotum at nine month.
- reaches base of scrotum at or just after birth.

Factors responsible for Descent of Testis:

- differential growth of body wall
- increased intra-abdominal pressure & temperature
- male sex hormones
- contraction of gubernaculum
- Calcitonin gene-related peptide (CGRP), a neurotransmitter secreted by genitofemoral N.



Applied Aspect:

Cryptorchidism (Incomplete descent of testis):

→ testis fail to reach base of scrotum

→ may be found at 5 sites:

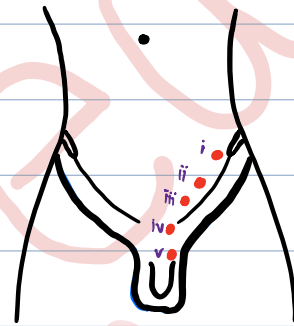
(i) within abdomen

(ii) at deep inguinal ring

(iii) within inguinal canal

(iv) at superficial inguinal ring

(v) high up in scrotum.



Cryptorchidism

→ Complications: — failure of spermatozoa production

— prone to undergo malignant changes

Ectopic Testis (Maldescent of Testis):

→ testis travel down along an abnormal path & fail to reach scrotum

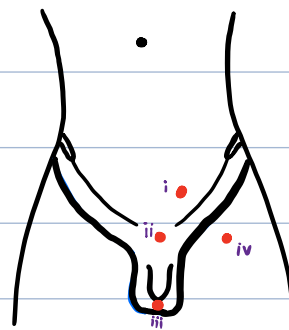
→ 4 sites:

(i) in superficial fascia of lower part of AAW

(ii) in front of pubis

(iii) in perineum

(iv) in the thigh



Ectopic Testis

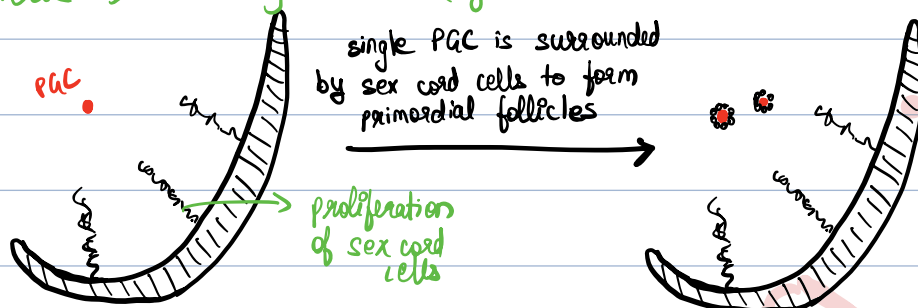
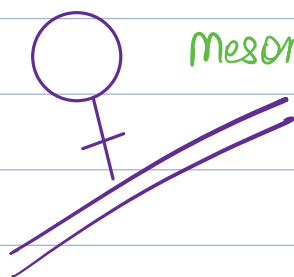
→ occurs because processus vaginalis reaches an abnormal site & the testis follows it.

→ necessary that undescended testis is brought down in scrotum (by a surgical procedure) before puberty

→ ectopic testis usually develops normally, but is susceptible to trauma

∴ it should also be placed in the scrotum surgically.

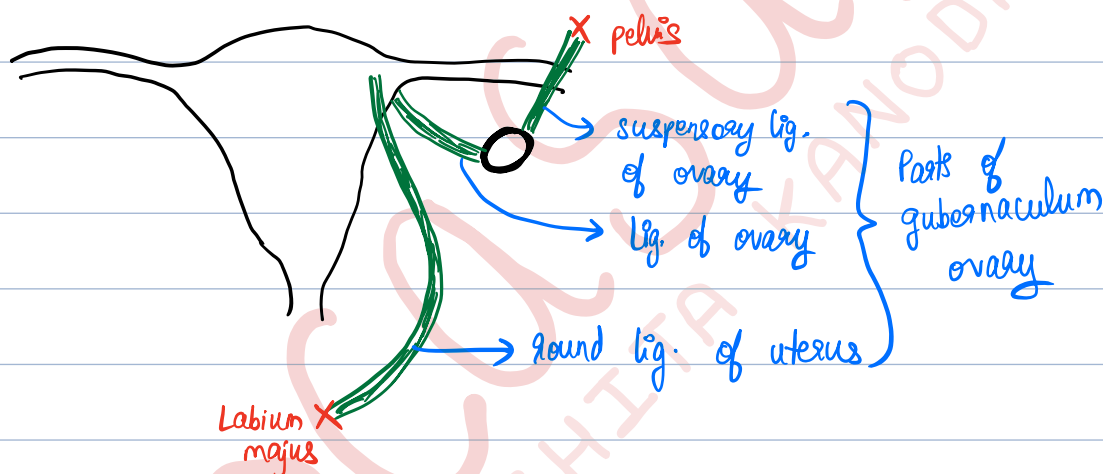
Mesonephalic Duct System Regresses.



(no formation of tunica albuginea in females)

Descent of Ovary:

→ By gubernaculum ovaries: attached to labium majus & pelvis
— = gubernaculum ovary



If mesonephalic duct persists:

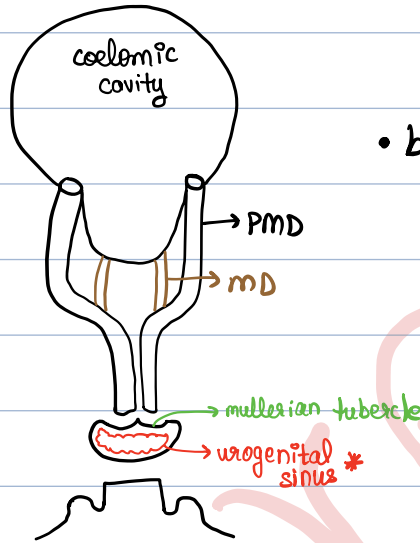
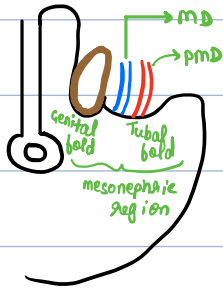
- Seen above ovary $\Rightarrow$ epophoron
- " below " $\Rightarrow$ paraphoron
- duct persists as a whole $\Rightarrow$ Gartner's duct.

Development of Uterus:

Mesonephric Duct = MD
Paramesonephric duct = PMD

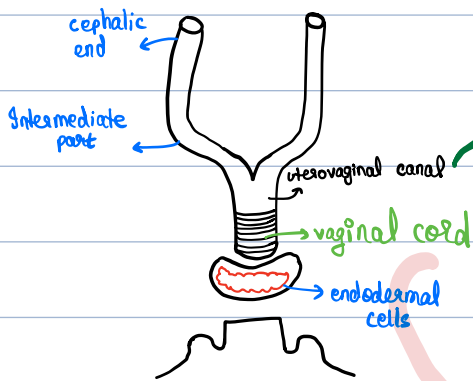
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- PMD is formed by 6th week of IUL
- by 8th week, PMD crosses from lateral to medial aspect in front of MD
- by 3rd month end, PMD of both sides come close to each other in midline

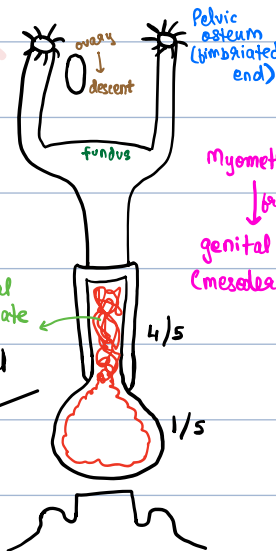


* in primitive gut, cloaca is divided by a urorectal septum into urogenital sinus anteriorly & primitive rectum posteriorly.

Caudal ends of both PMD fuse by end of 3rd month to form uterovaginal canal in caudo-cranial direction (↑).



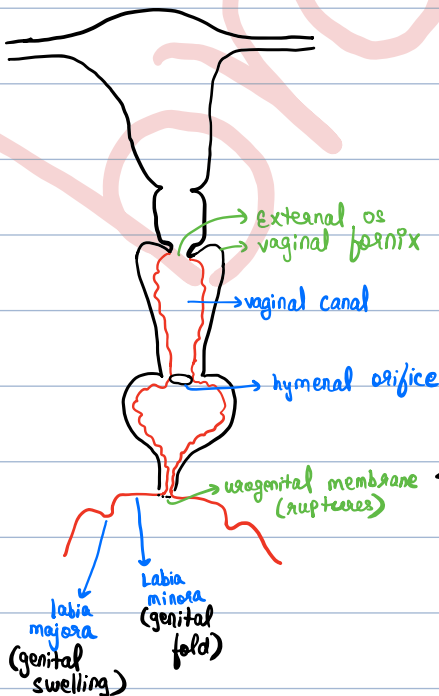
cranial end → uterine cavity & cervix



Myometrium
↓ from
genital cords
(mesodermal)

Endometrium + lining of cervix
↓ from
PMD
(mesodermal)

the endodermal cells of sinovaginal plate canalize to form vaginal cavity.



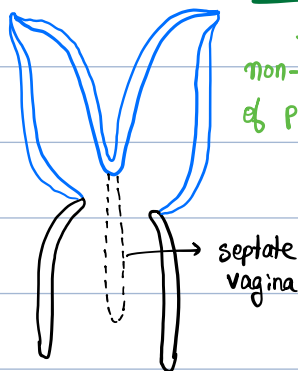
- Hymenal membrane ⇒ mesodermal
- Hymenal orifice ⇒ endodermal

Congenital Anomalies of Uterus & Uterine Tubes:

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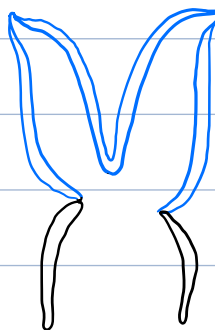
Didelphys

↓
non-fusion
of PMDs



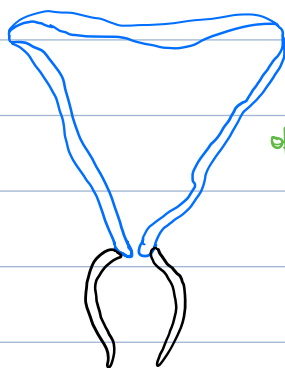
Bicornis Bicollis

non-fusion of
upper portion
of PMDs

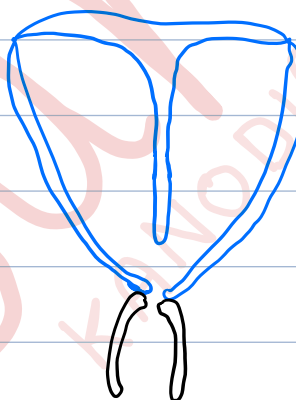


Aruate / Bicornuate uterus

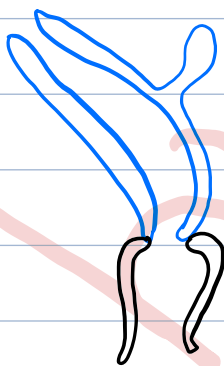
↓
incomplete fusion
of upper ends of PMDs



Septate / Subseptate uterus

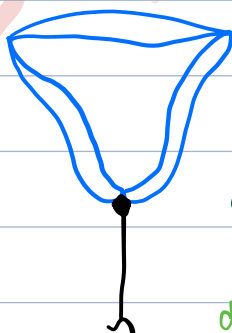


Unicornuate uterus



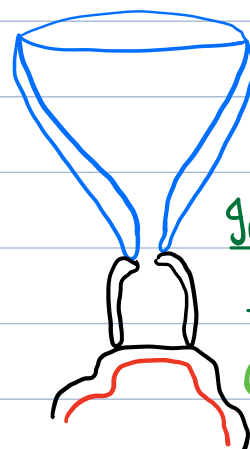
Congenital Atresia of vagina

↓
due to failure of
canalization of sinovaginal
bulb.



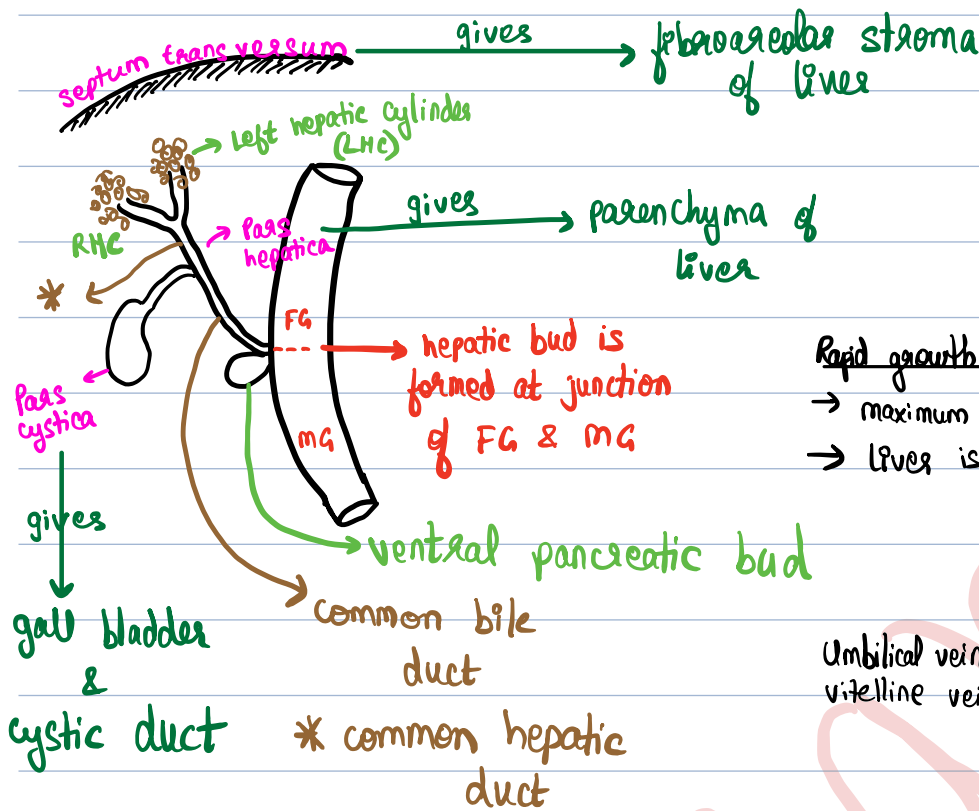
Imperforate hymen

(cryptomenstraea
on attaining
puberty)



Development of Liver & Extrahepatic Biliary Apparatus:

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Rapid growth of liver is due to:

- maximum oxygenated blood is first received by liver
- liver is acting as hematopoietic tissue

Umbilical veins, vitelline veins → form → sinusoids of liver

Derivatives of Ventral Mesogastrium:

- Falciform ligament
- Coronary ligament
- Triangular ligament
- Greater omentum

from ventral mesogastrium b/w liver & body wall

from ventral mesogastrium b/w liver & stomach

- Ligamentum Venosum ⇒ remnant of ductus venosus
- Ligamentum Teres hepatis ⇒ remnant of obliterated left umbilical vein

Atresia of Duct: any part of common hepatic/cystic/common bile duct is absent

Accessory Bile Duct: check during cholecystectomy

Riedel's Lobe: extra tongue-like lobe arising from right lobe of liver

Polycystic Liver Disease: ducts within the liver remain as cysts & fail to communicate with ducts of extrahepatic biliary apparatus

Caroli's Disease: congenital dilation of bile ducts

Agenesis of Gall Bladder (GB): absence of GB

Stalkless GB: absence of cystic duct

↳ GB directly arises from bile duct

Phrygian Cap: fundus of GB folds on itself to form a cap-like structure

Hartman's Pouch: formation of a pouch from neck of GB

Intrahepatic GB: GB grows inside liver due to close proximity of pars cystica with hepatic cylinders

Alpha-fetoprotein (AFP) levels may also be elevated in gastroschisis and omphalocele. AFP levels are low in pregnancy of Down syndrome fetus.