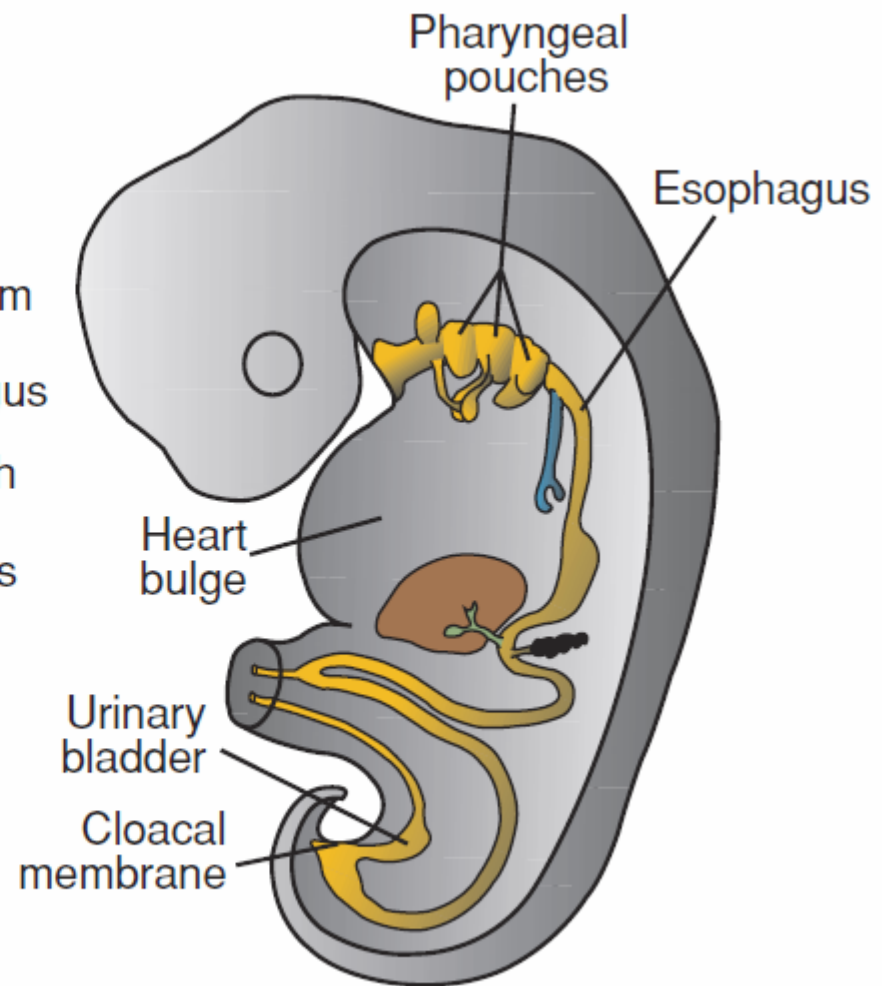
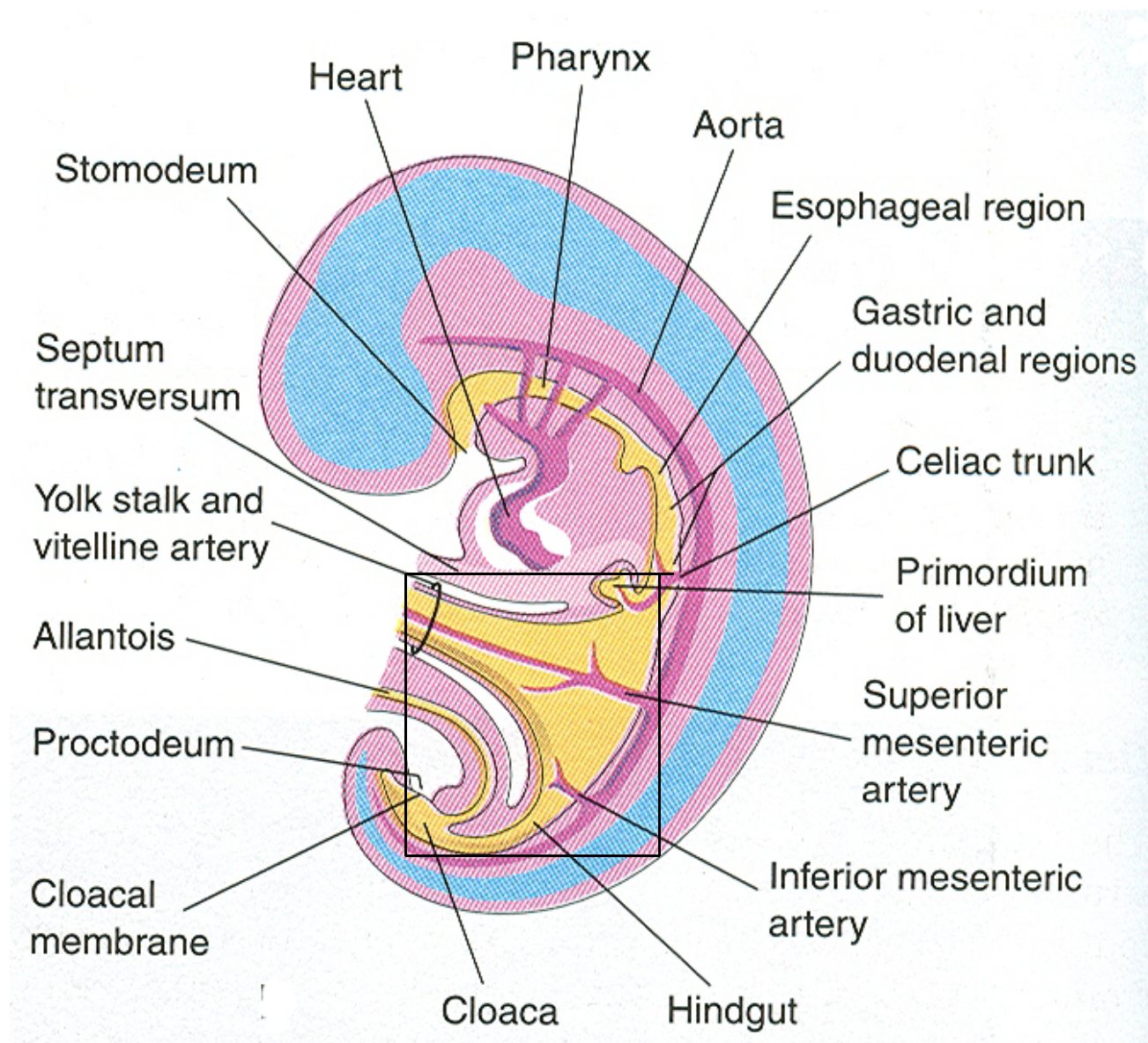
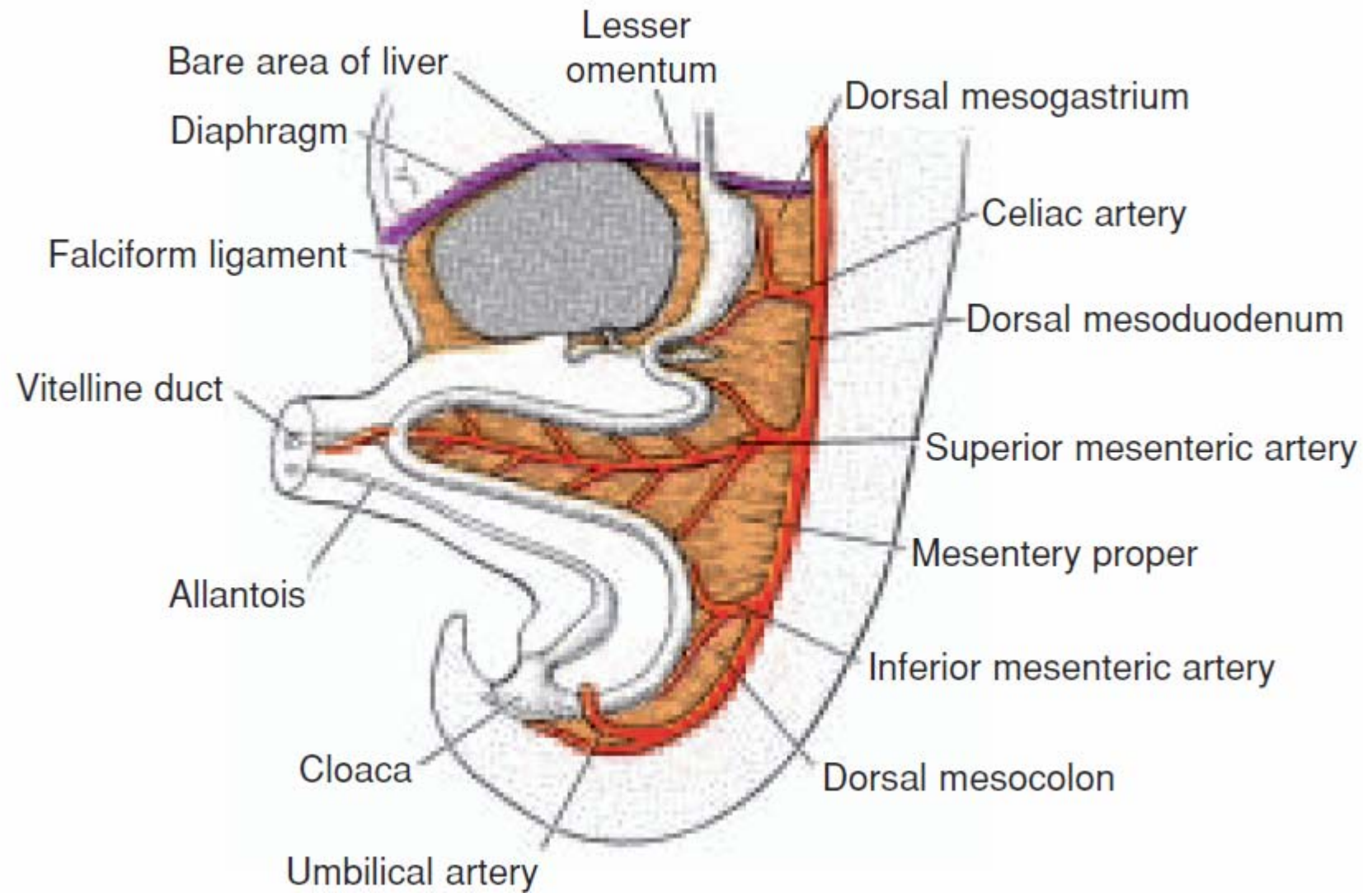


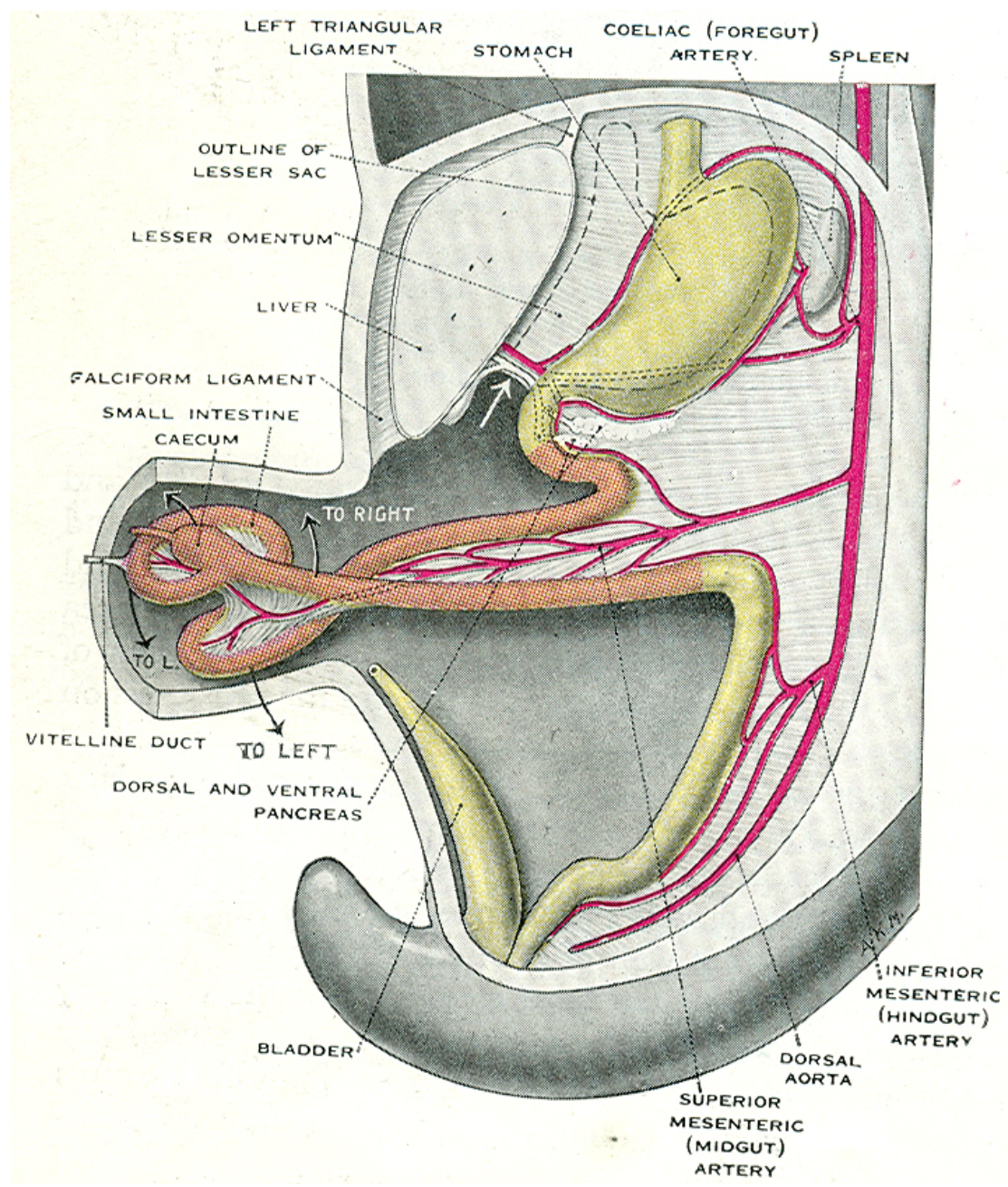
A

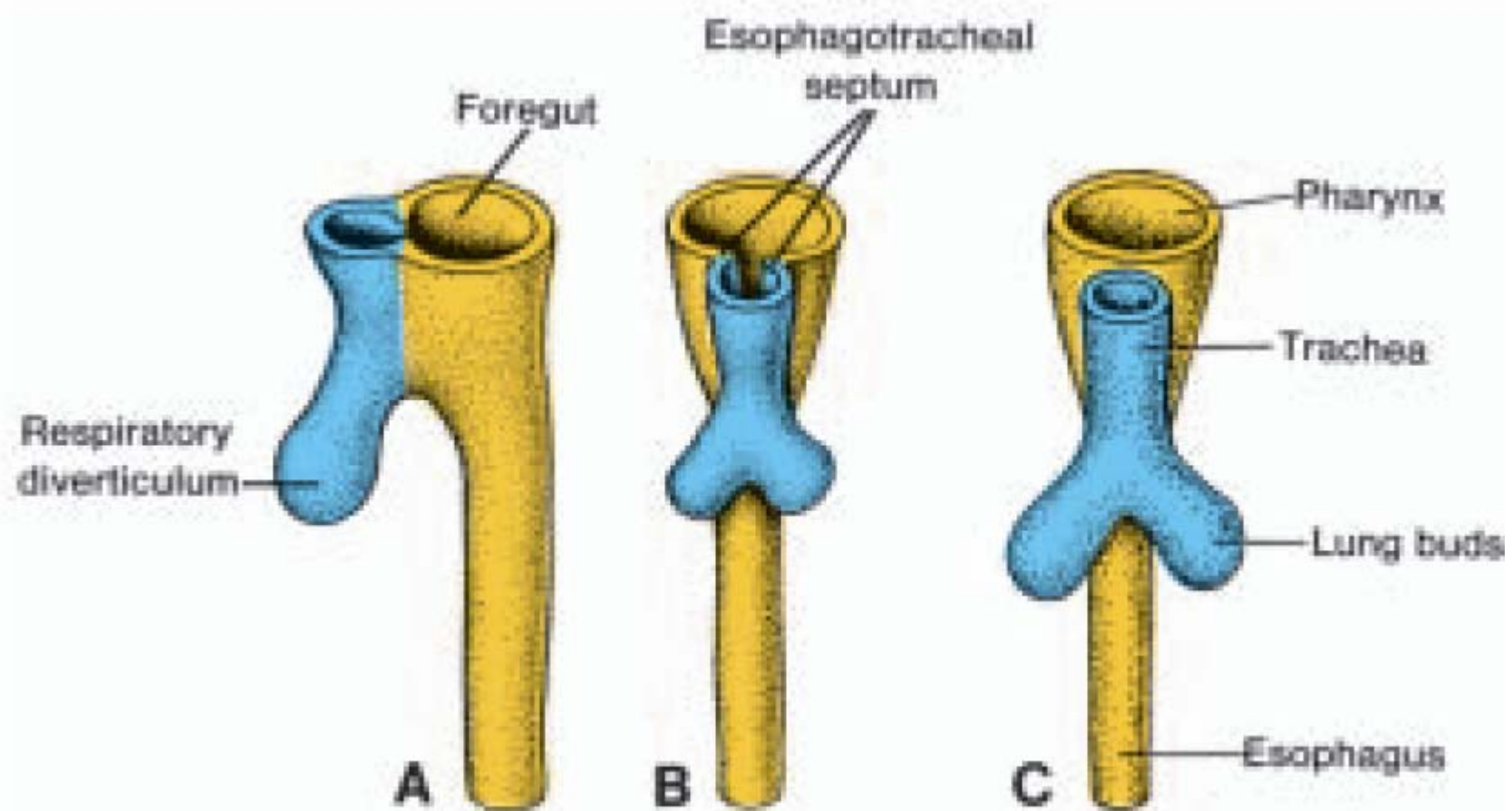


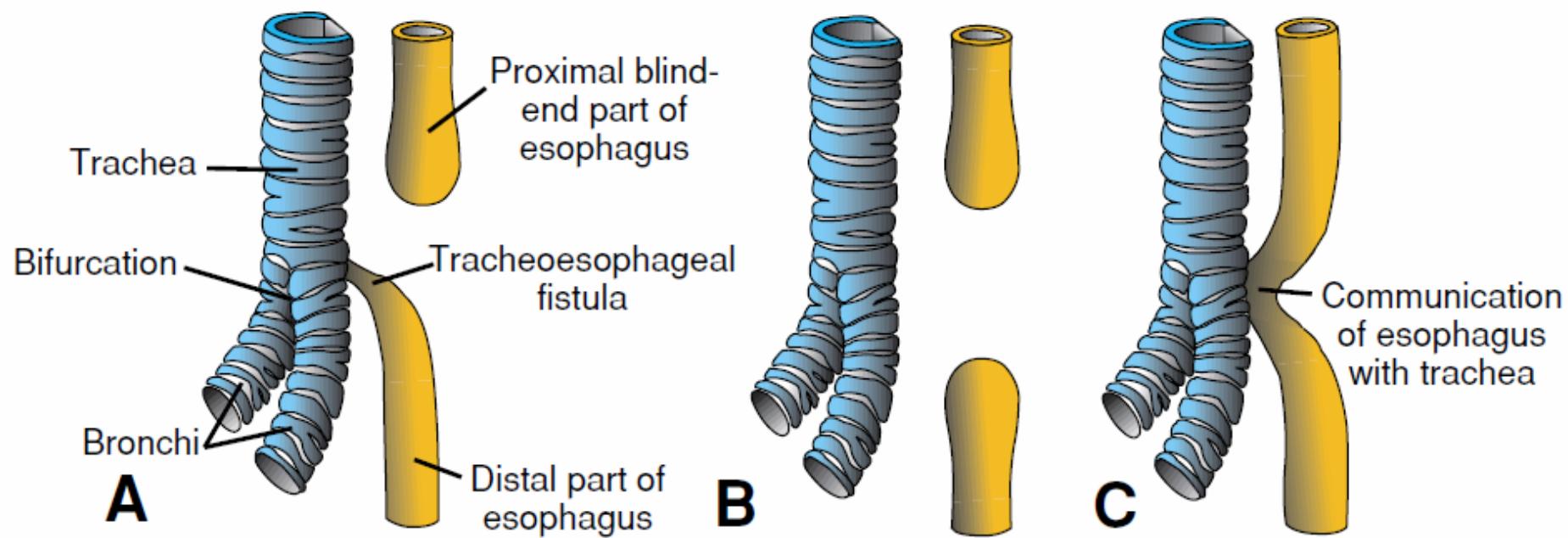
B







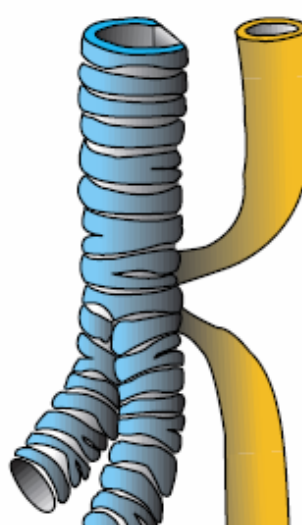
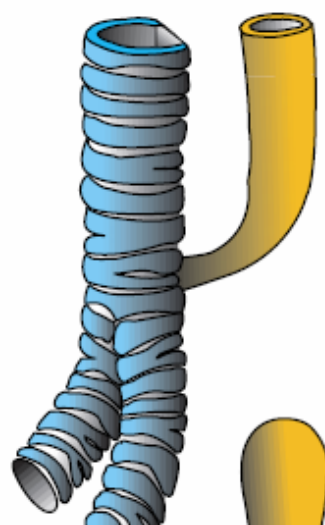


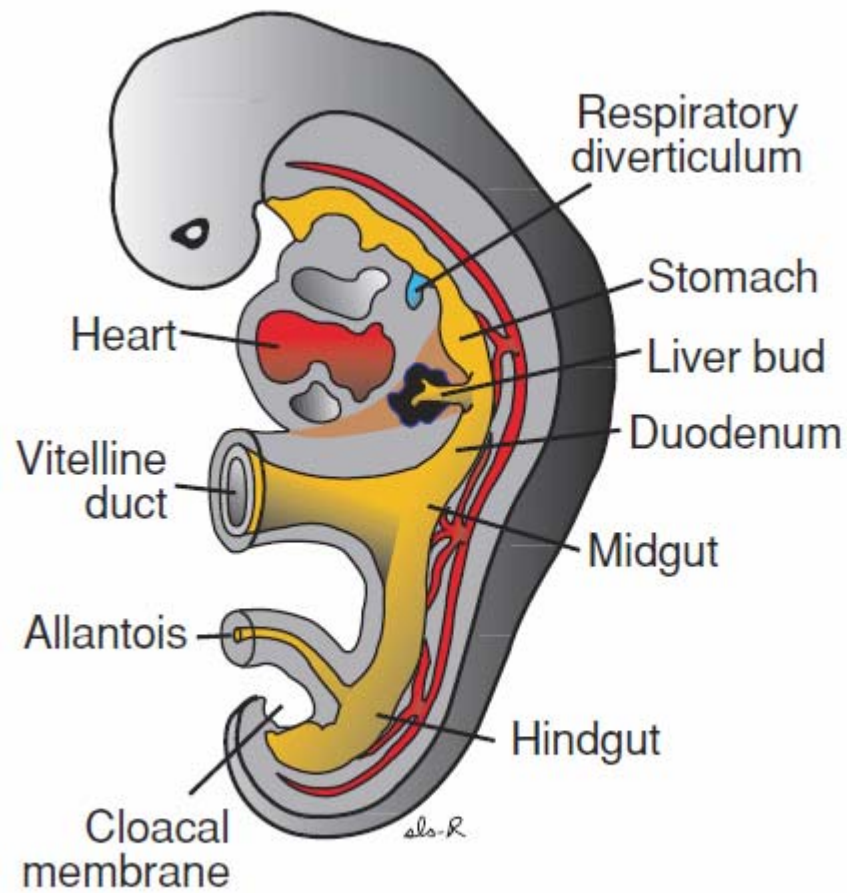


B

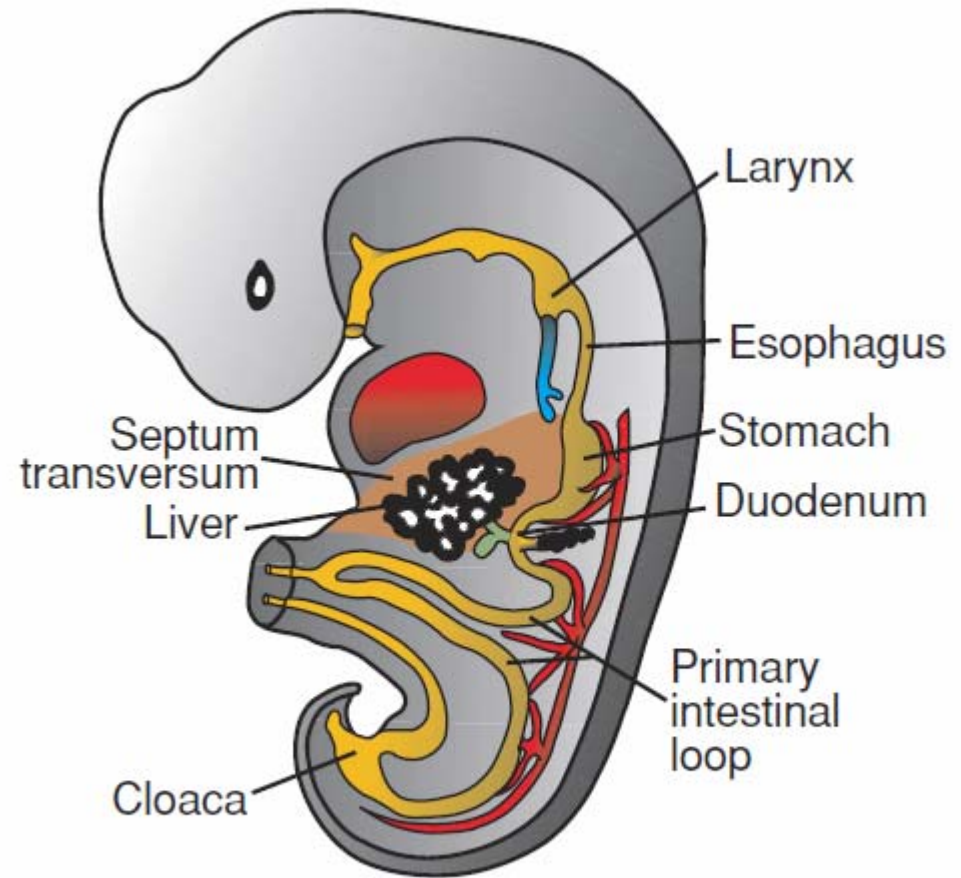
C

Communication of esophagus with trachea

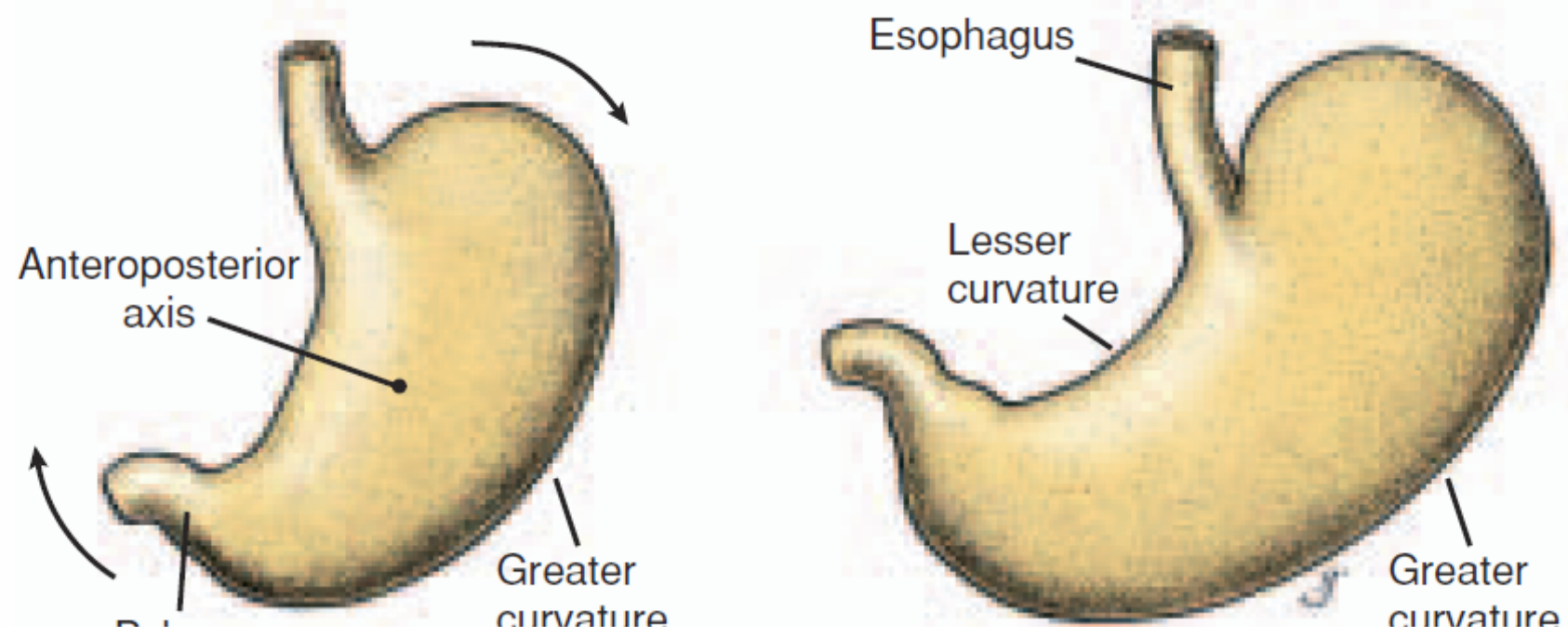
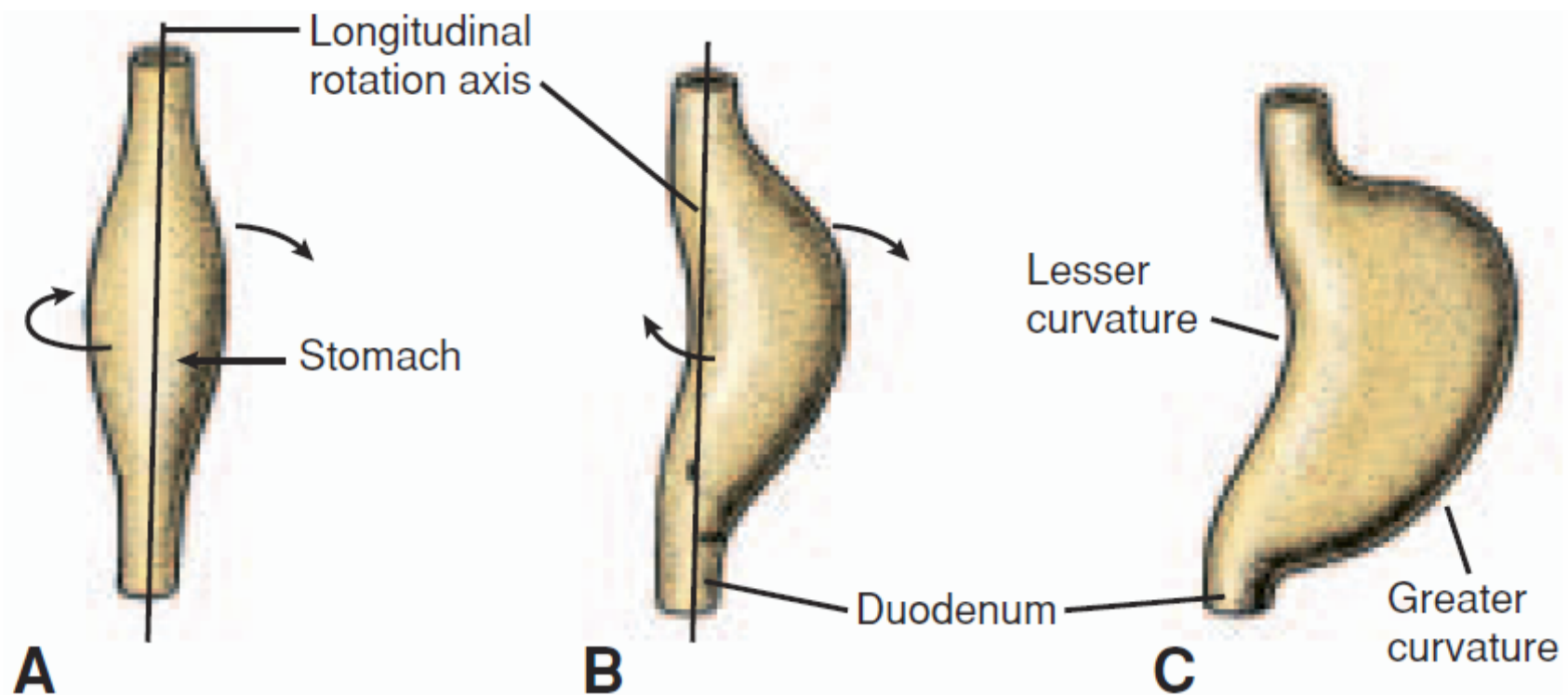


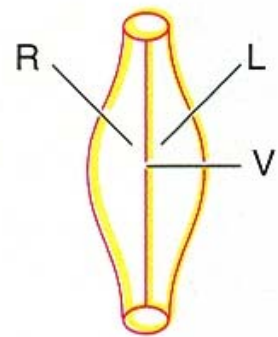


A

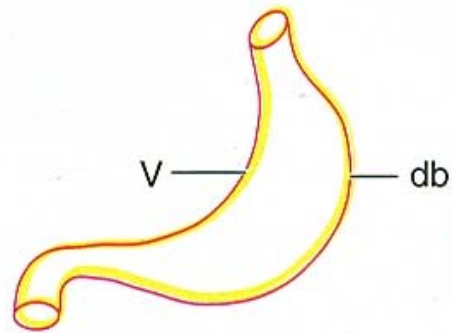


B

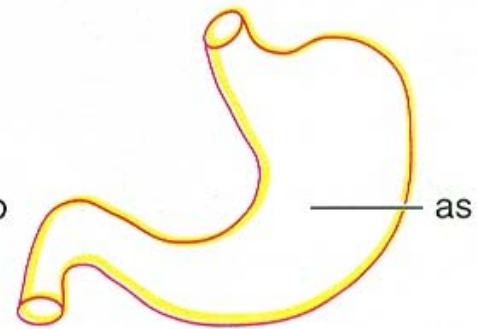




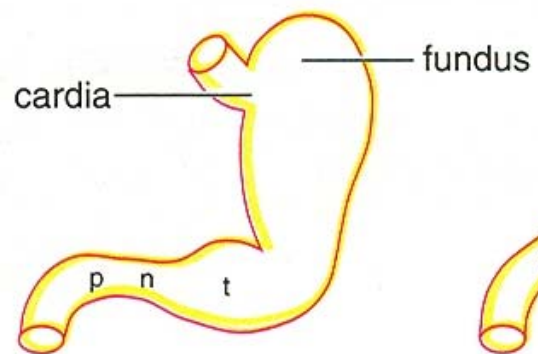
(a)



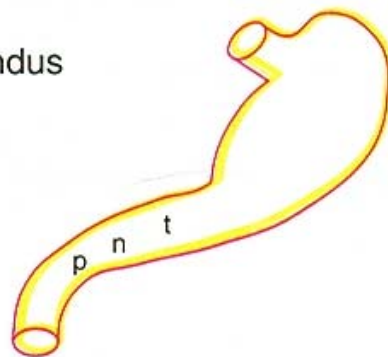
(b)



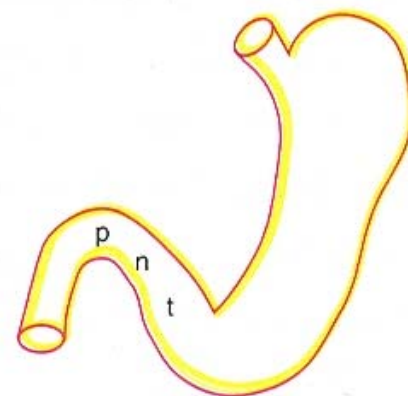
(c)



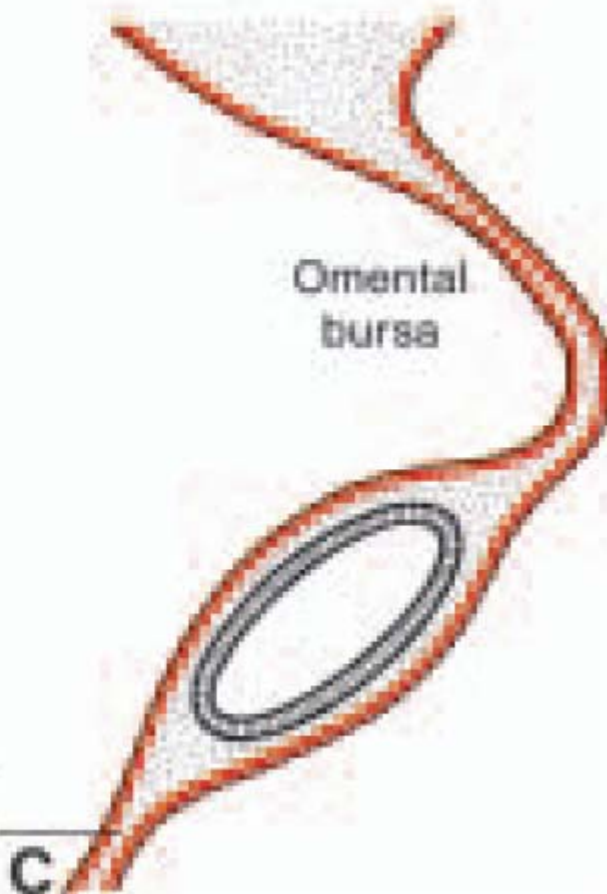
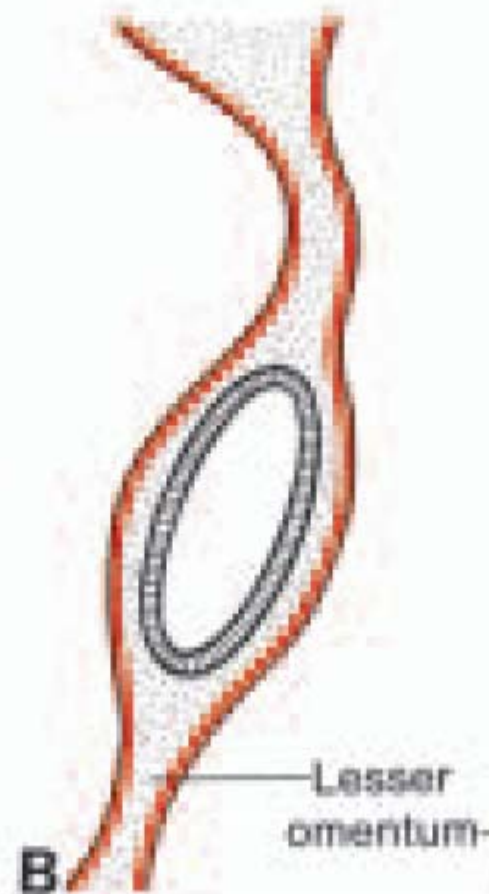
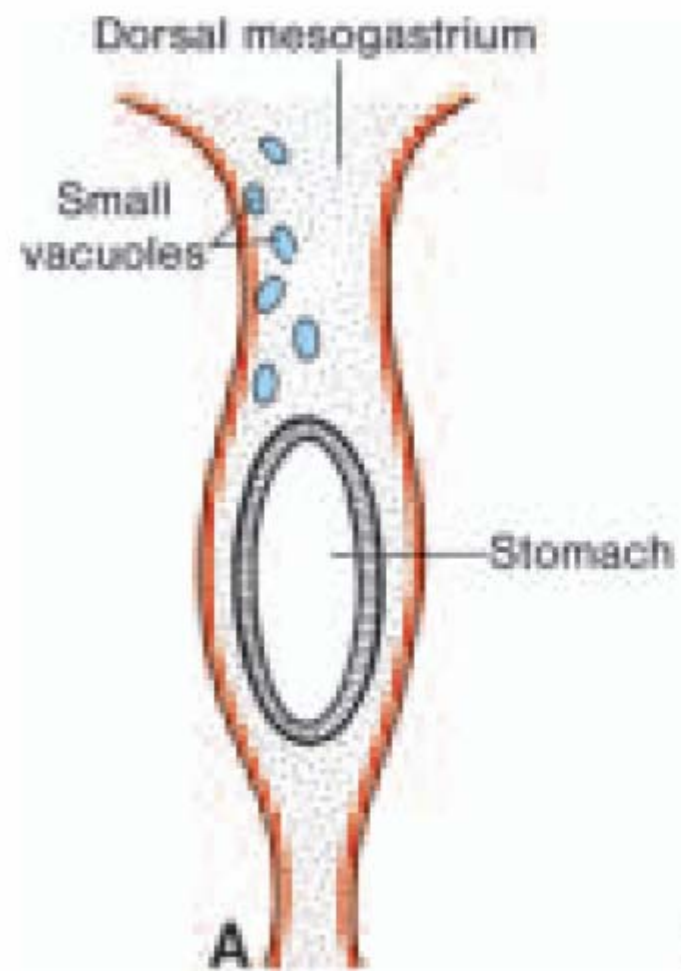
(d)

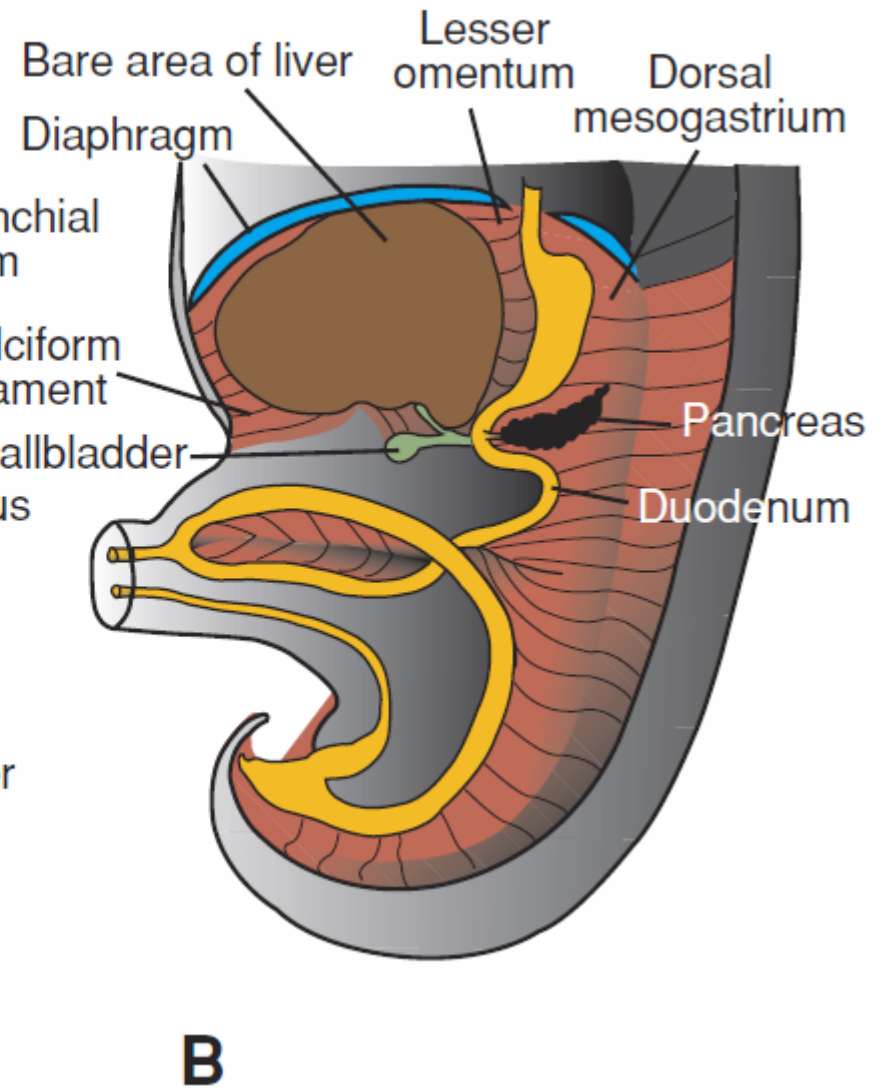
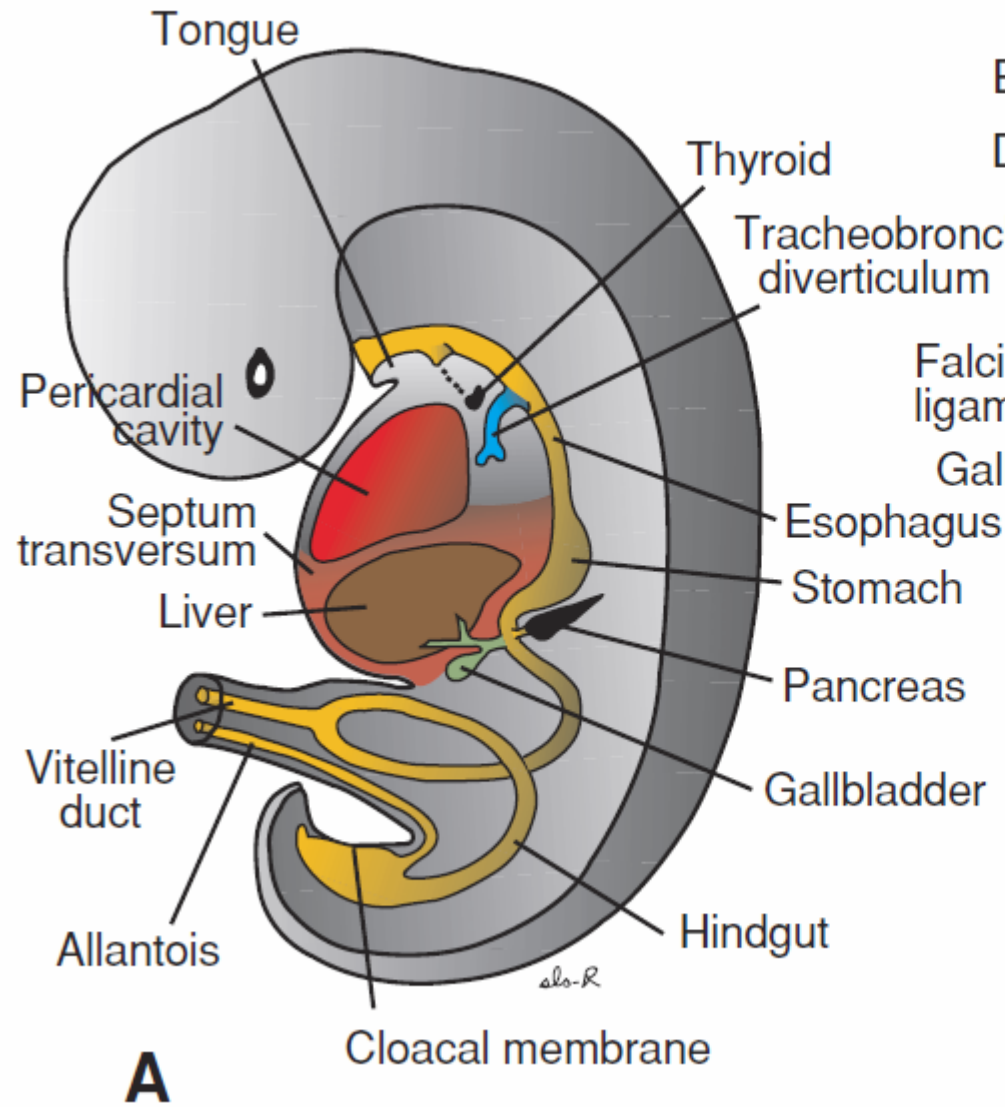


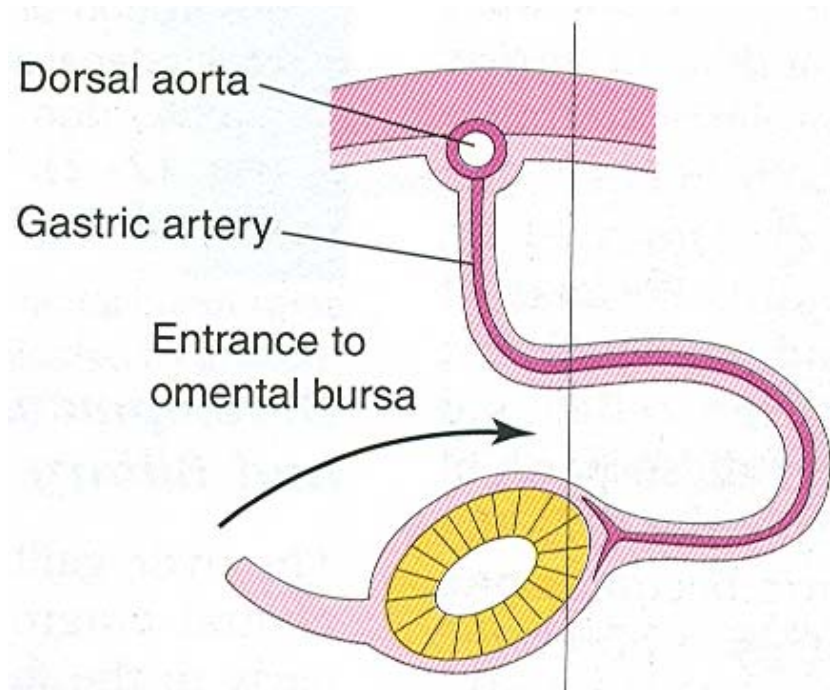
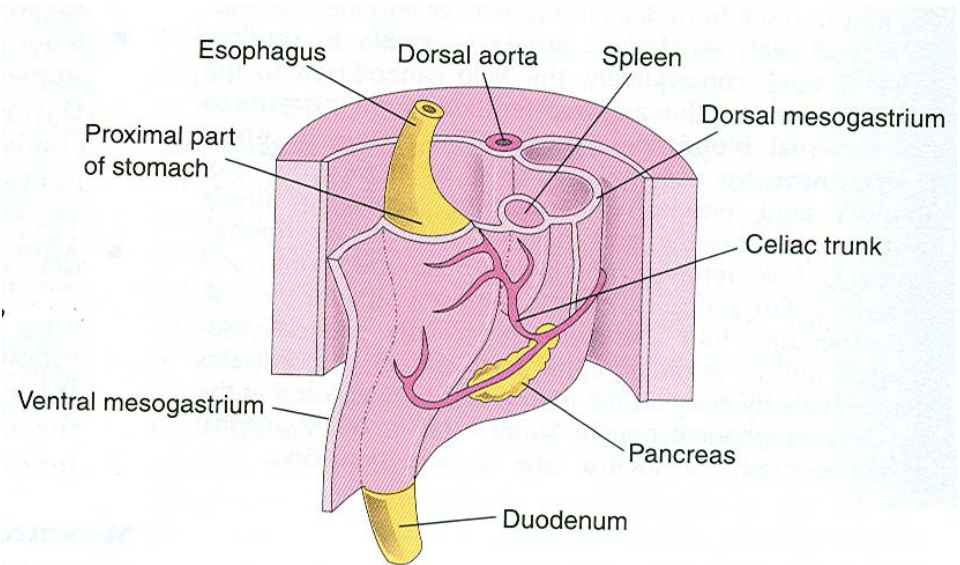
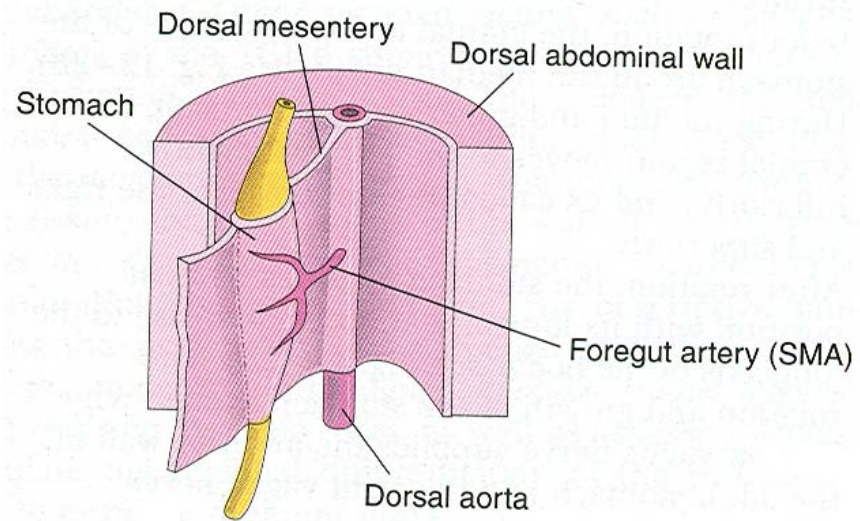
(e)

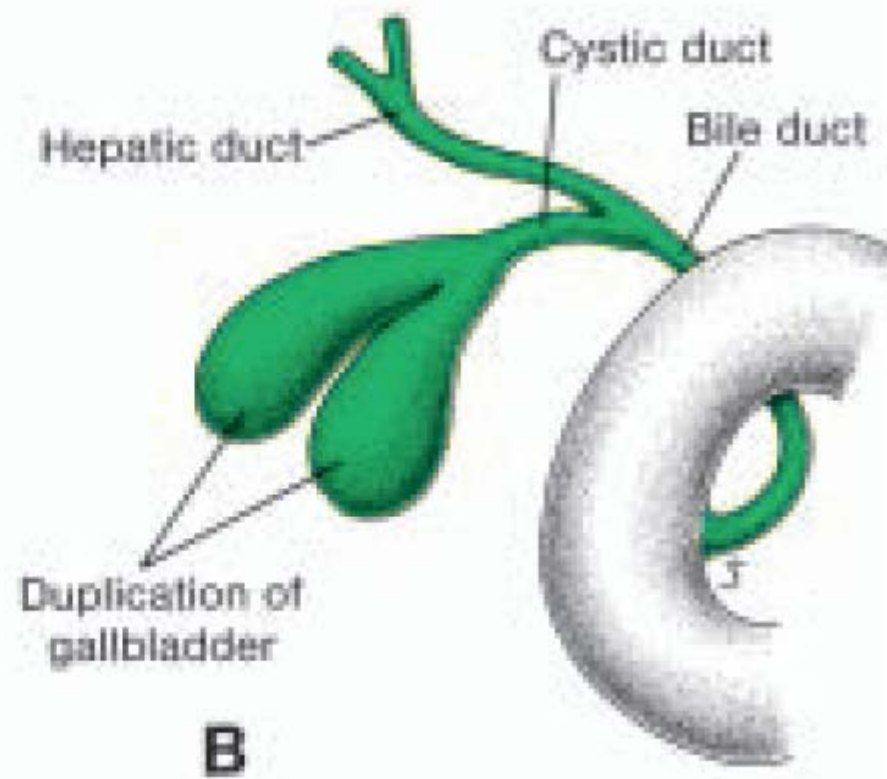
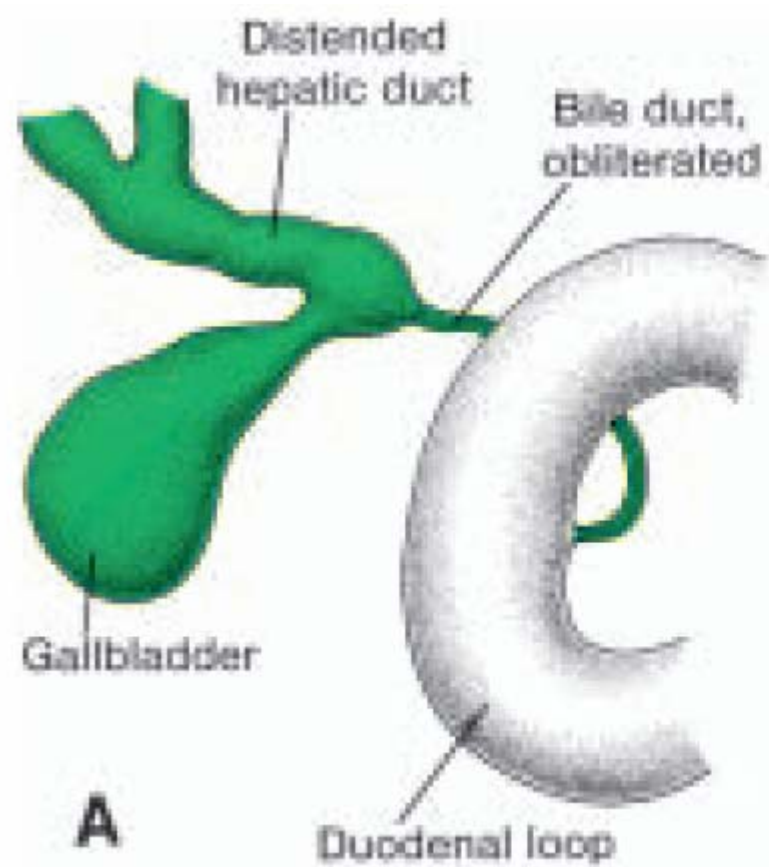


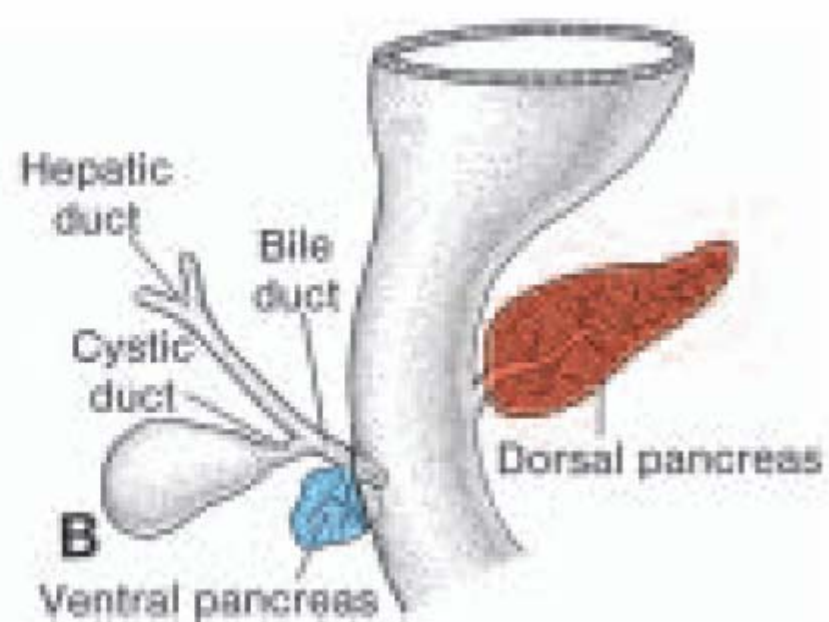
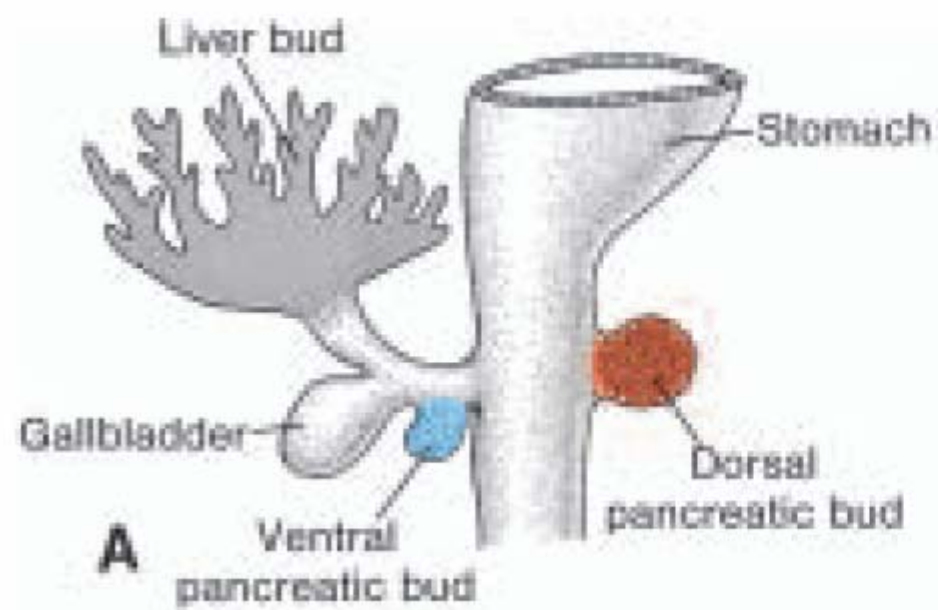
(f)

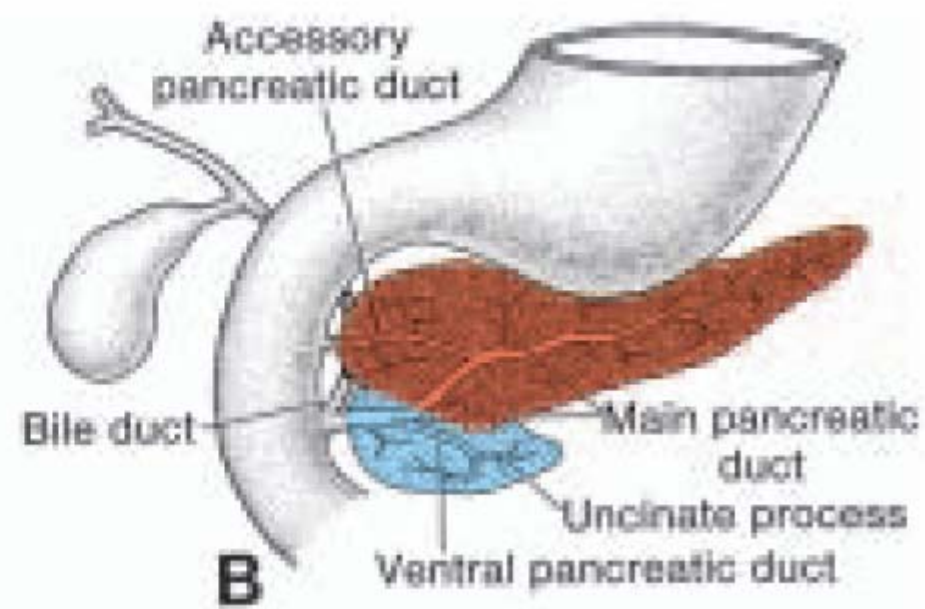
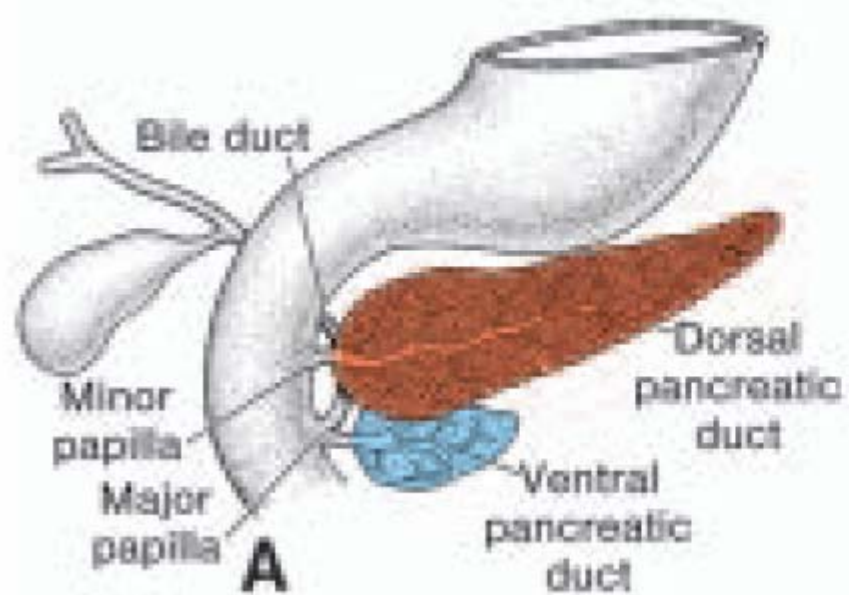


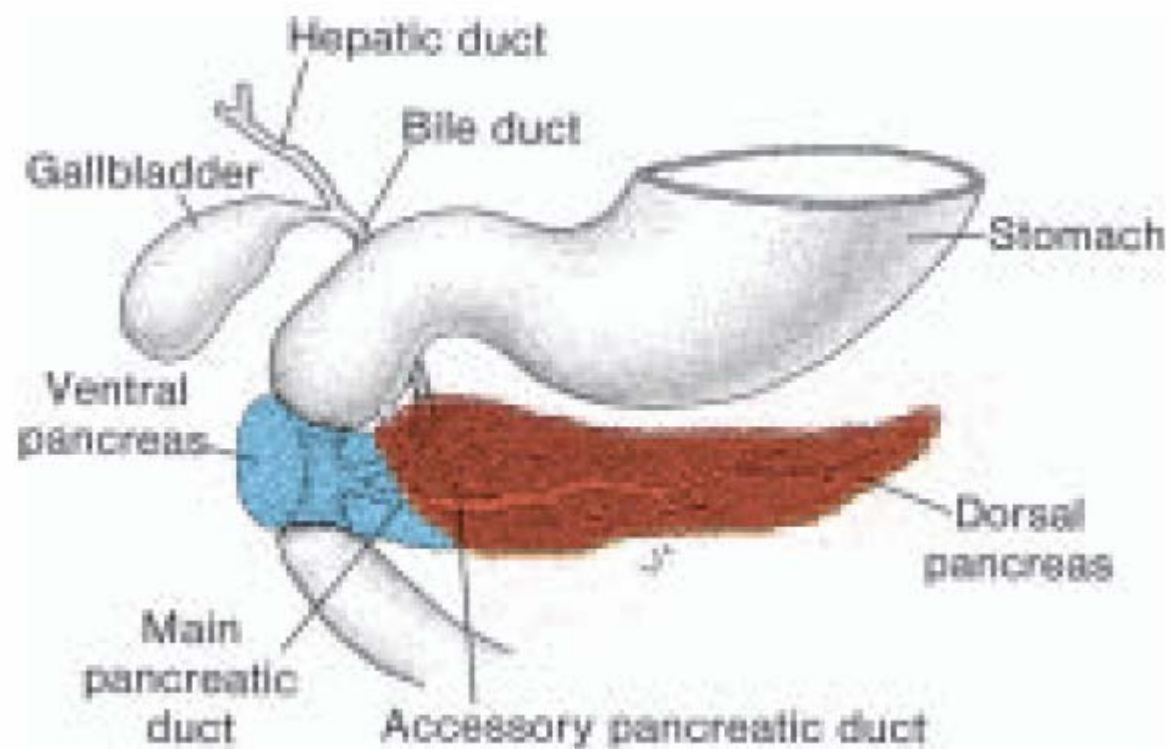


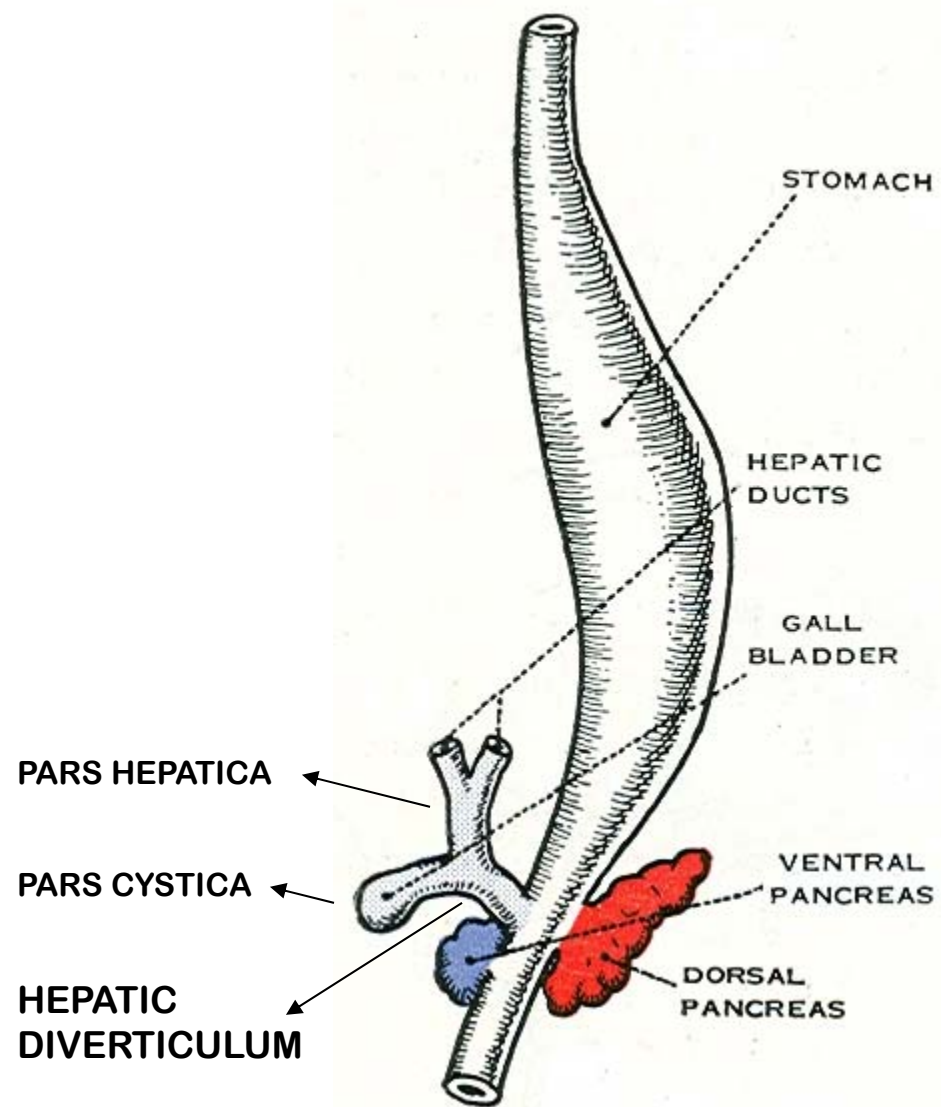


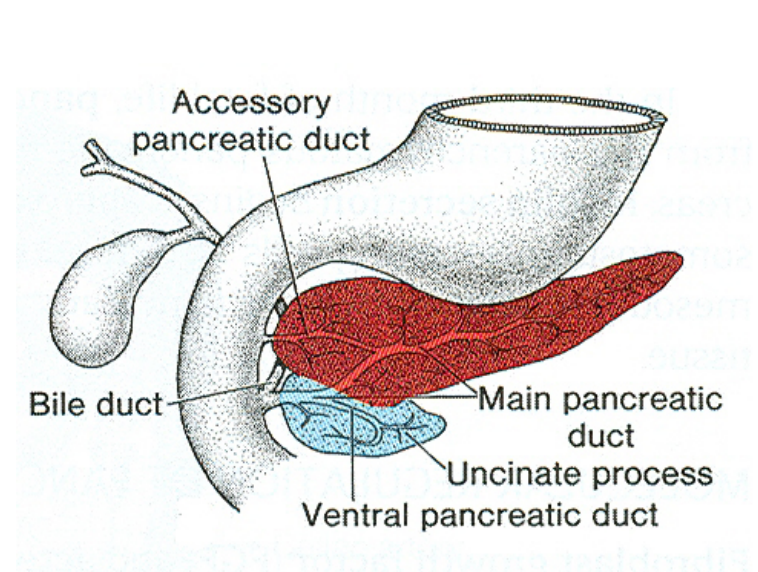
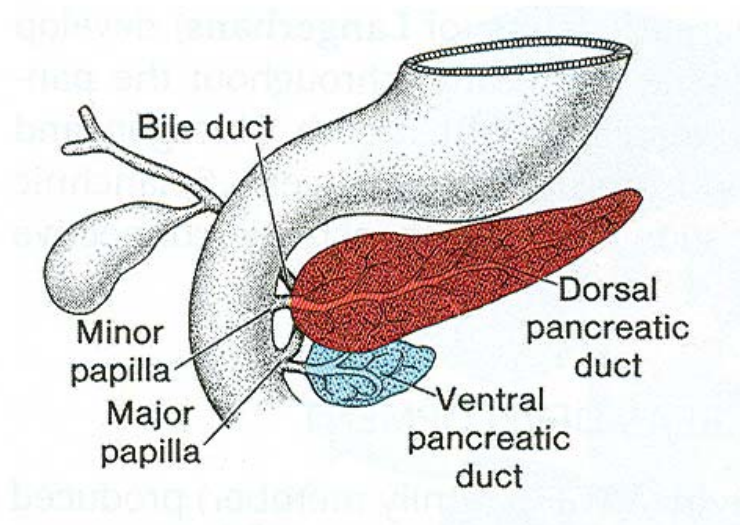
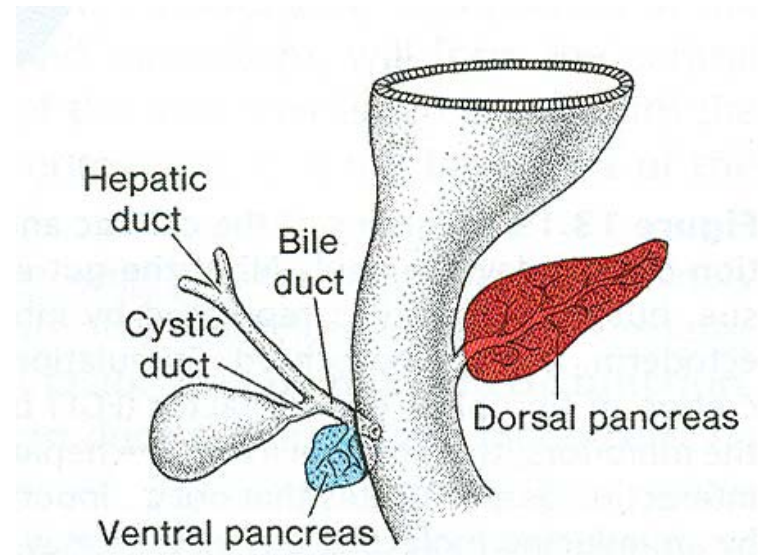
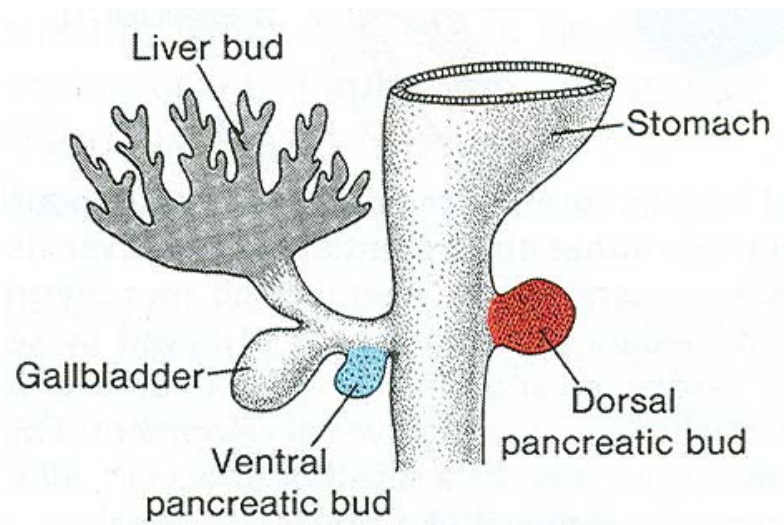


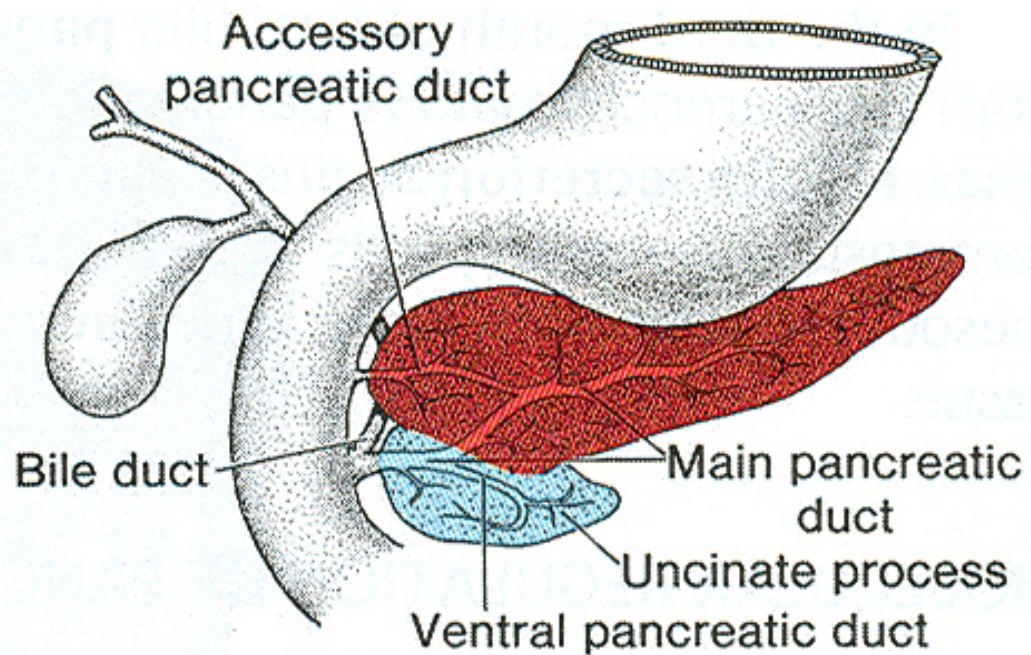
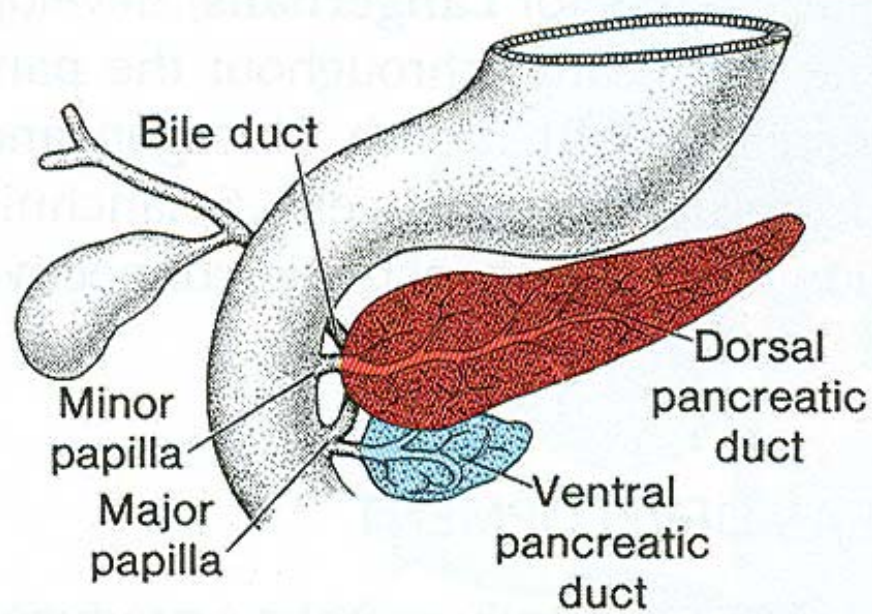


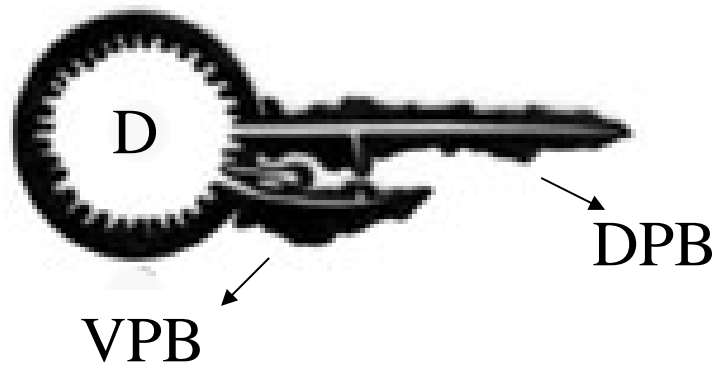
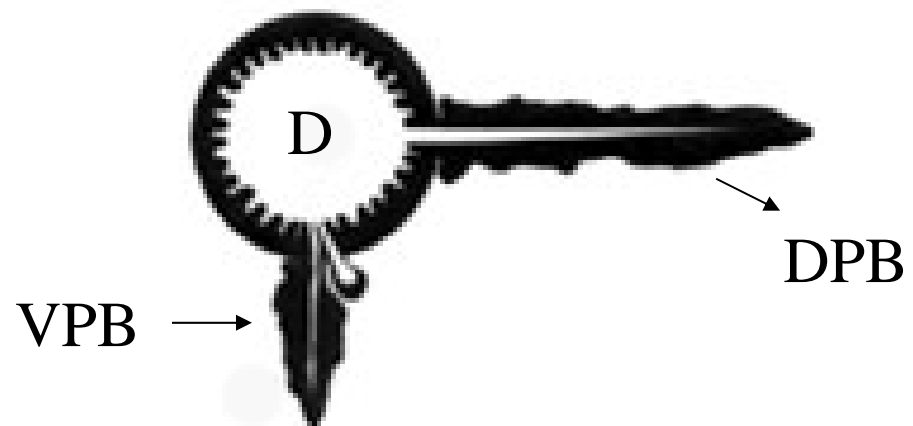
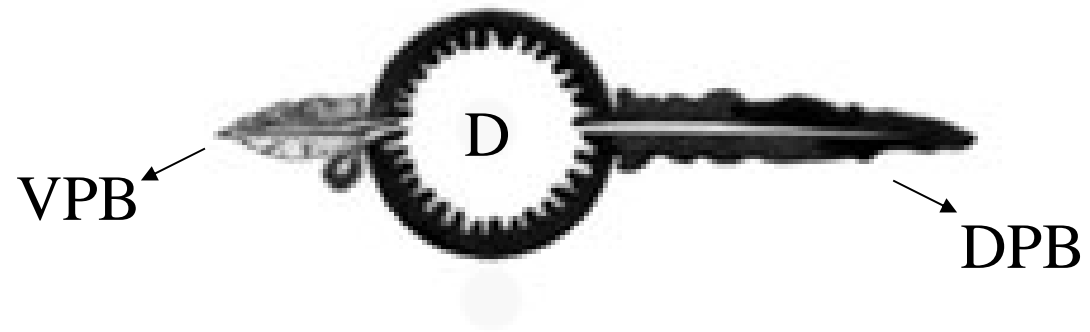


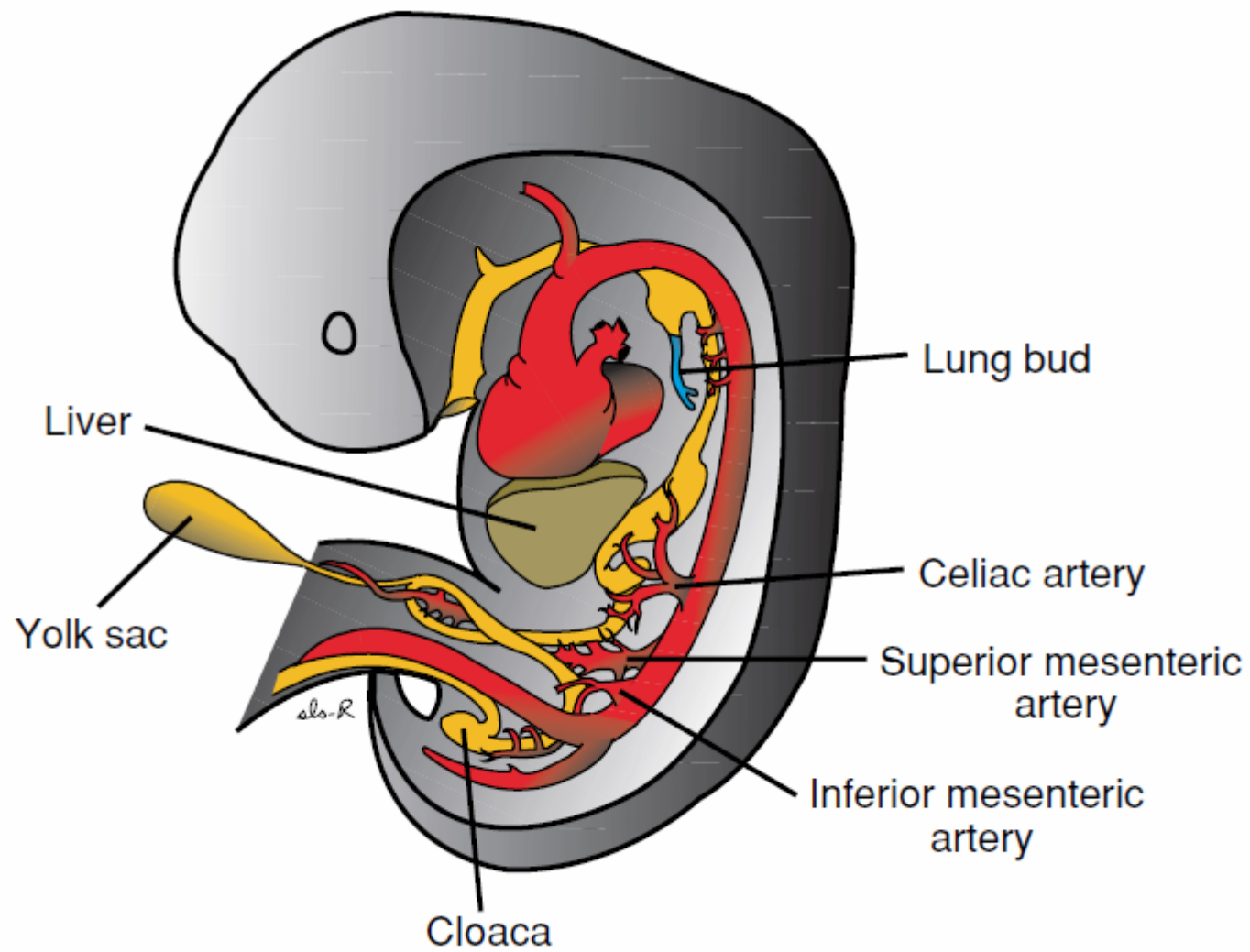






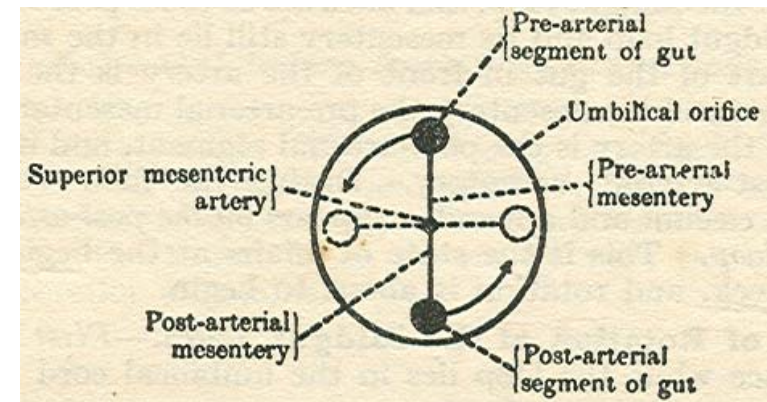
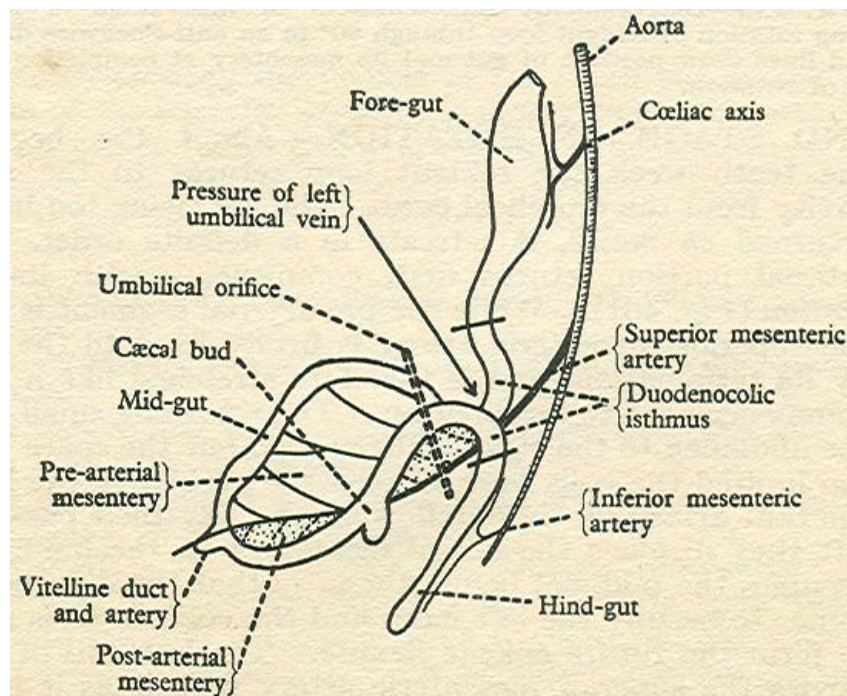
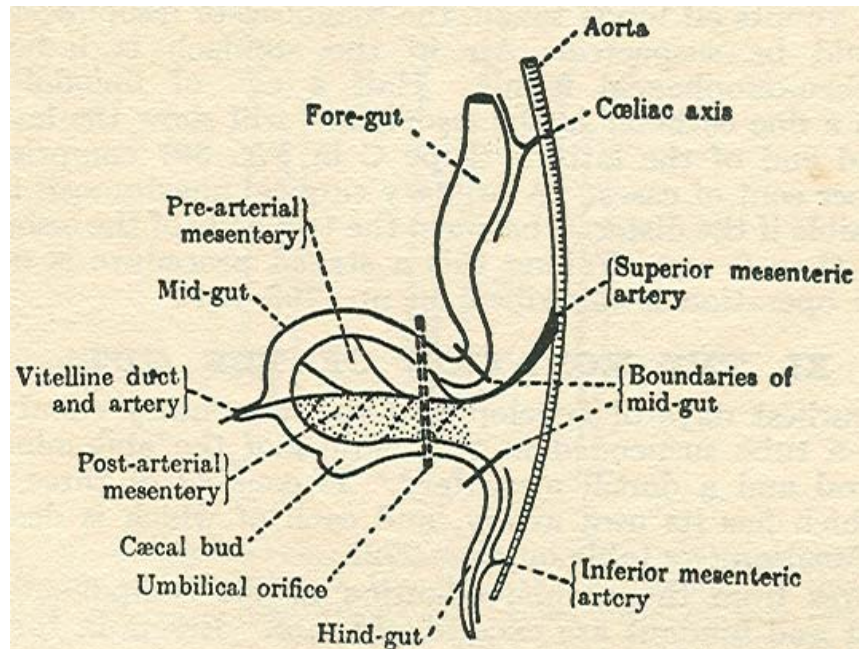




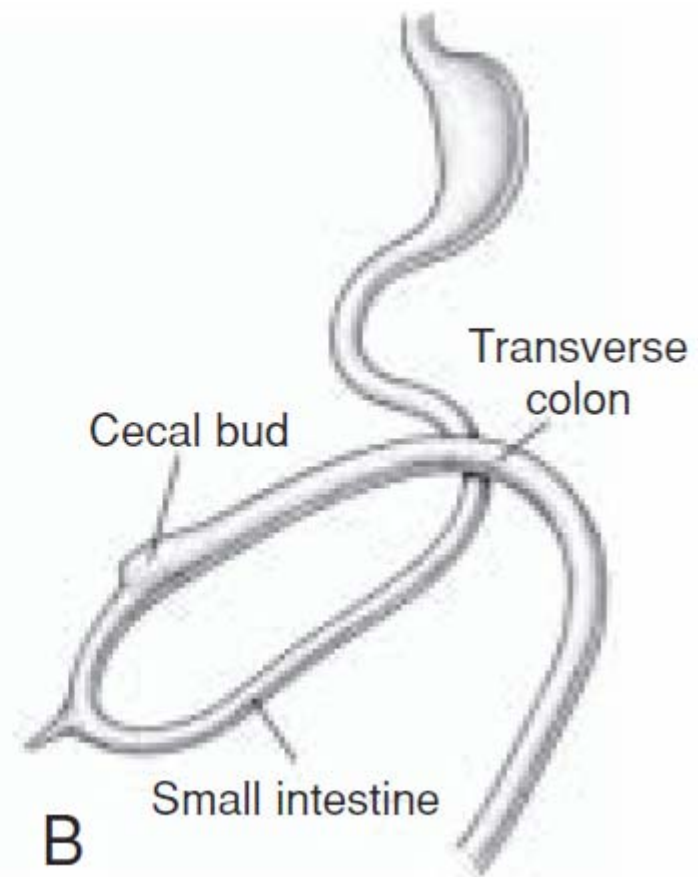
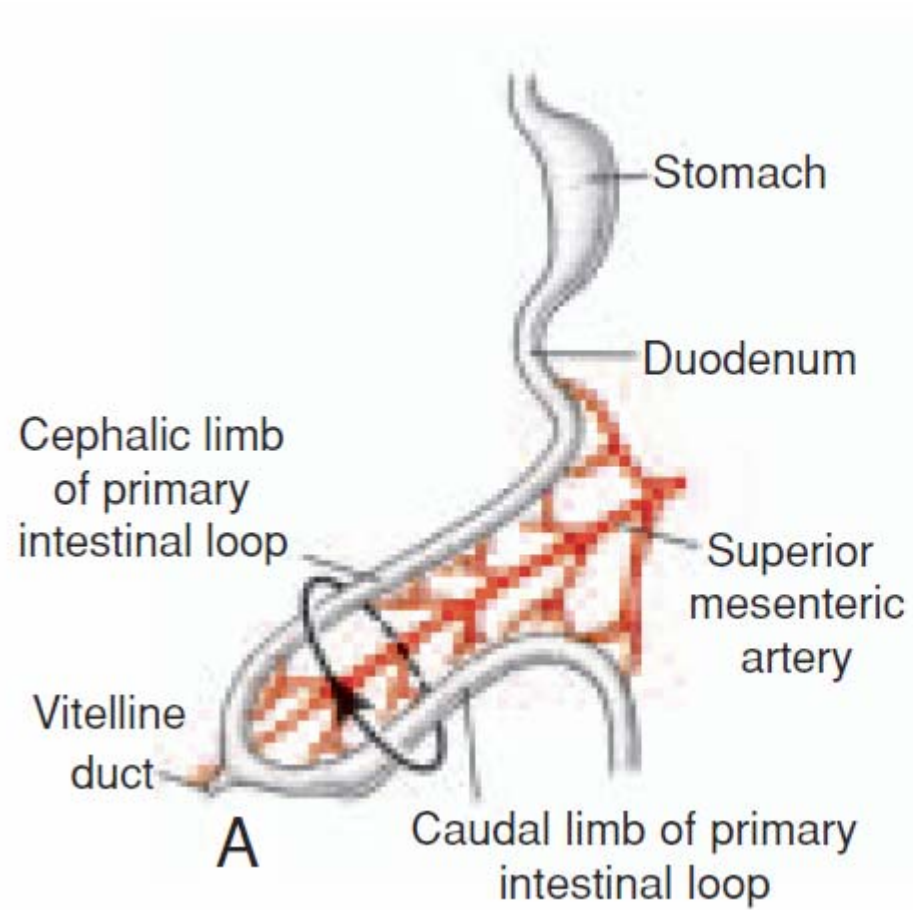


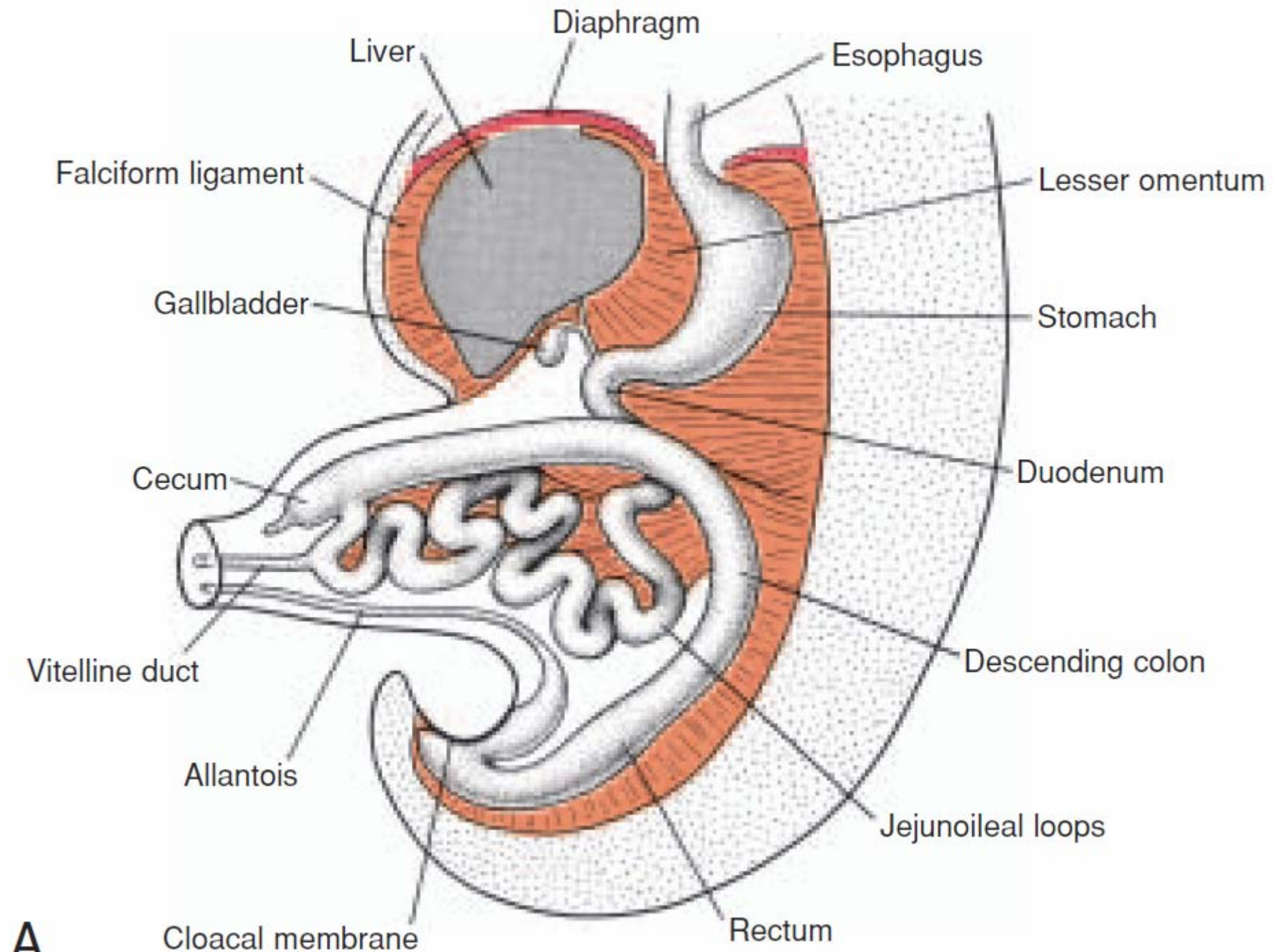
Factors causing physiological hernia:

1. Rapid growth & large size of liver which occupies a large area of abdominal cavity.
2. Small size of abdominal cavity because the lumbar segment is not yet formed.
3. Mid gut is growing at a fast pace.

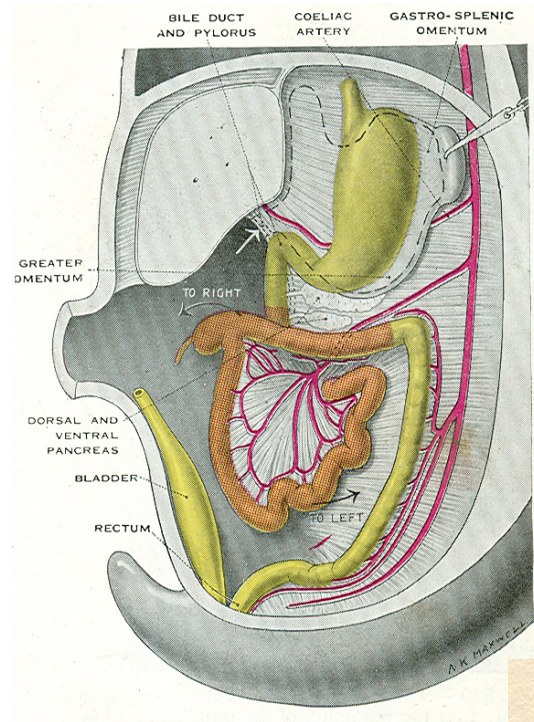


1st stage: Stage of physiological hernia; 90° anticlockwise rotation; 5-10th wks of IUL.

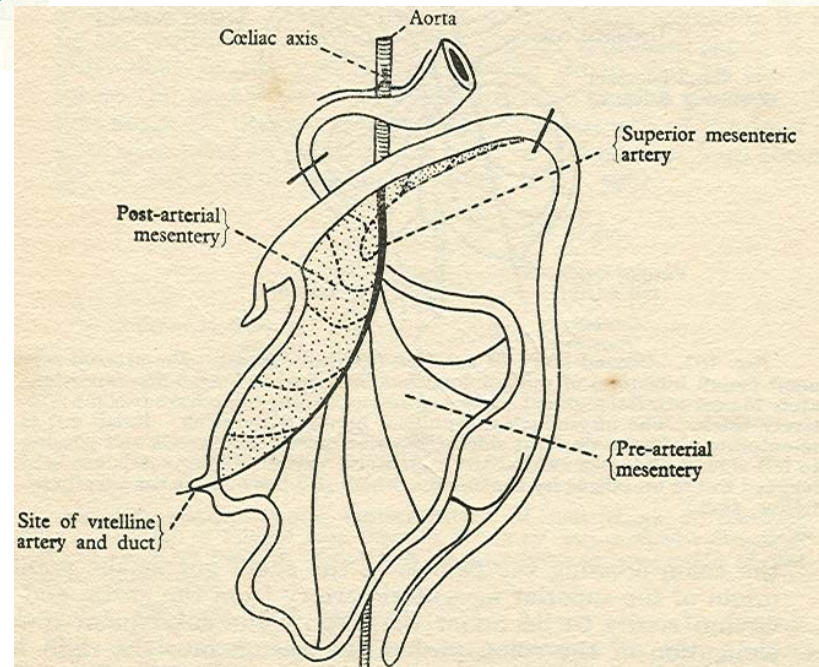
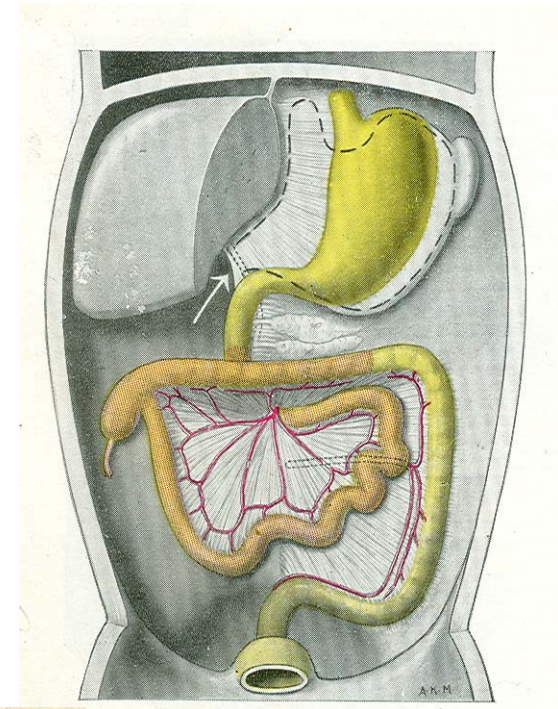




A



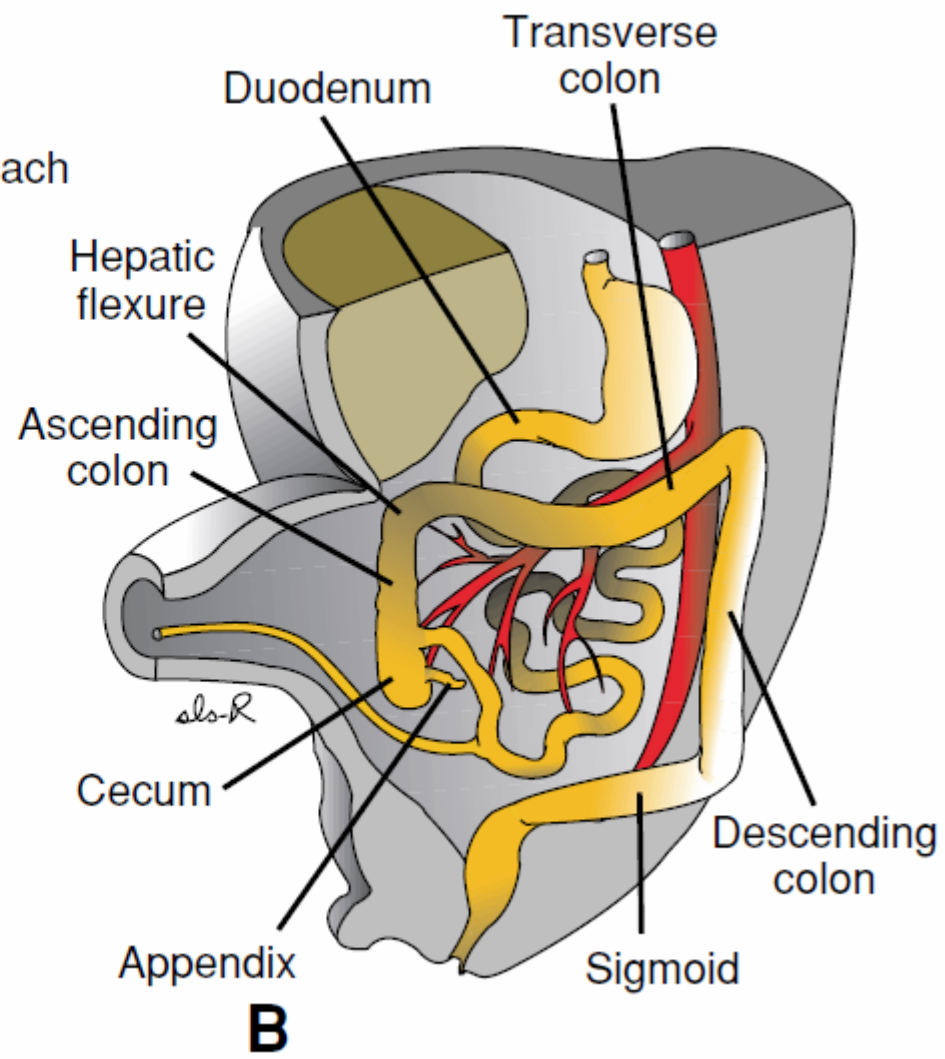
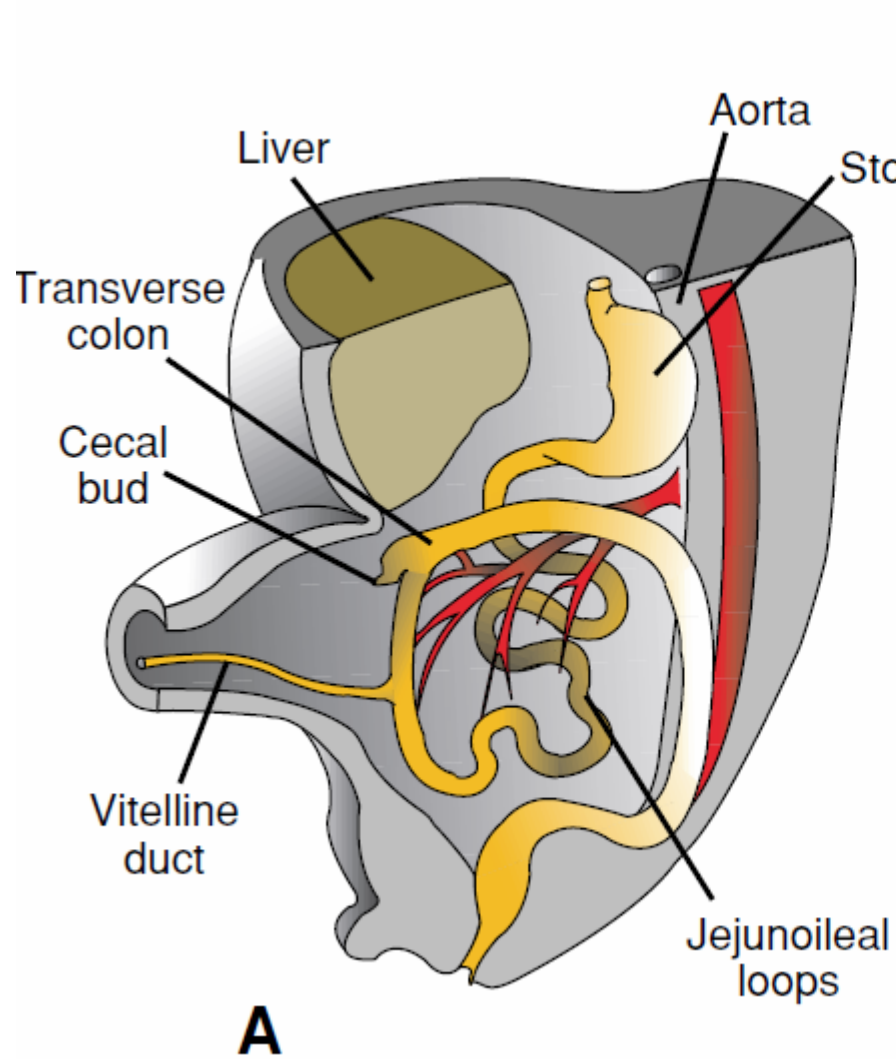
2nd stage: Reduction
of hernia;
180⁰ anticlockwise
rotation;
10-11th wks of IUL.



Factors causing reduction of physiological hernia:

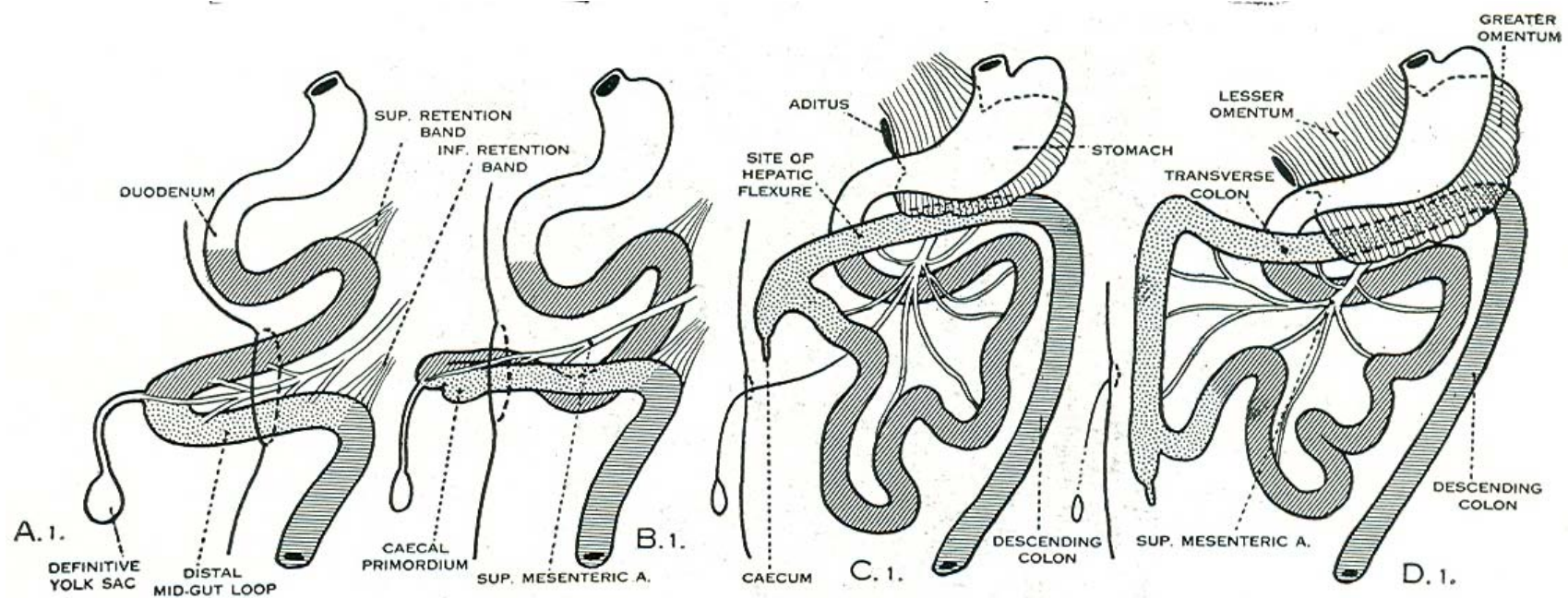
1. Liver occupies relatively less space (its haemopoietic function is over)
2. Abdominal cavity is larger now as the lumbar segment is formed.
3. Contraction of some fibromuscular bands in the mesentery helps in reduction of the herniated loop.
4. Contraction of longitudinal muscles of mid gut may also be of some help in reduction.

Because of the caecal bud, the post arterial segment can not reduce first. Pre arterial segment reduces first and occupies upper left part of abdominal cavity. Subsequent loops progressively settle towards the right lower segment of abdominal cavity.



Rotation of midgut

- Ist Stage: Herniation. Prearterial and postarterial segments. Caecal bud
- IInd stage: Reduction. Duodenum crosses behind artery; transverse colon in front; caecum in the right side; intestine from left upper to right lower segments of abdomen.
- IIIrd stage: Fixation. Caecum reaches right illiac fossa. Zygois occurs in some parts. Mesentry becomes adherent to post. Abdominal wall. Transverse mesocolon.



1st stage

Physiological
hernia; 90° of
acw rotation.
5-10th wks of
IUL.

2nd stage

Reduction of
hernia; 180°
of acw
rotation.
10-11th wks
of IUL.

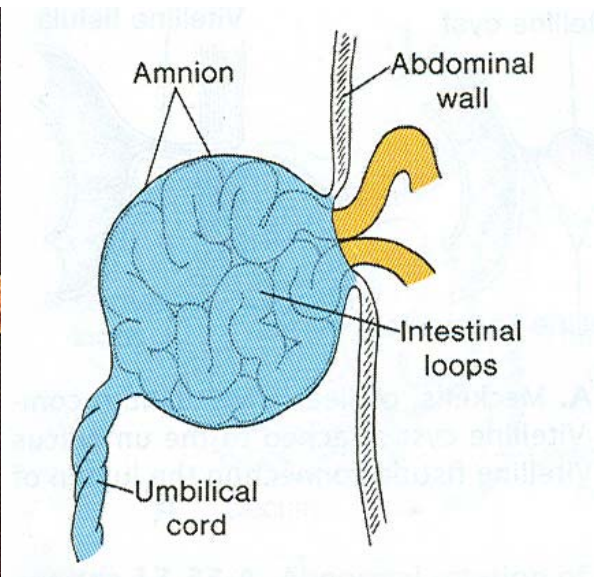
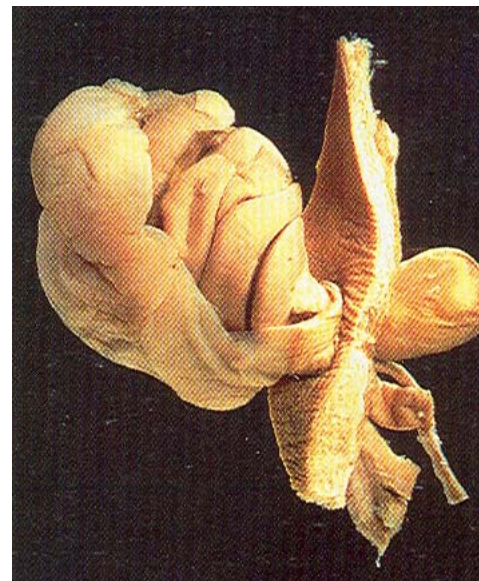
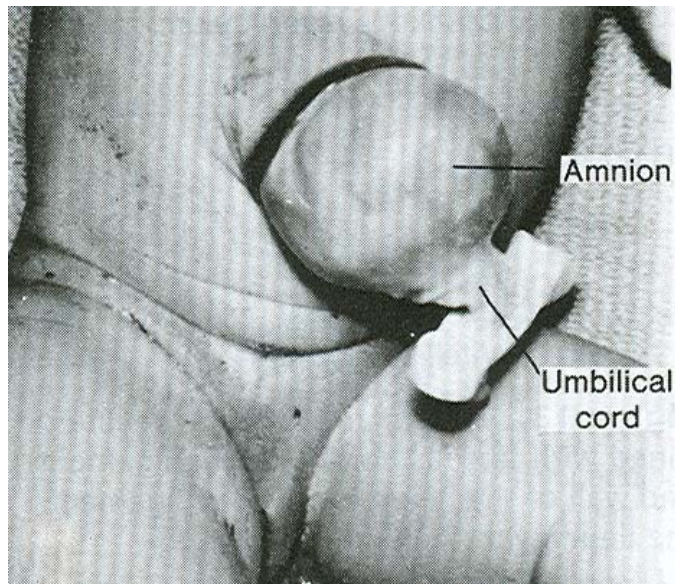
3rd stage

Fixation
11th wk to after
birth.

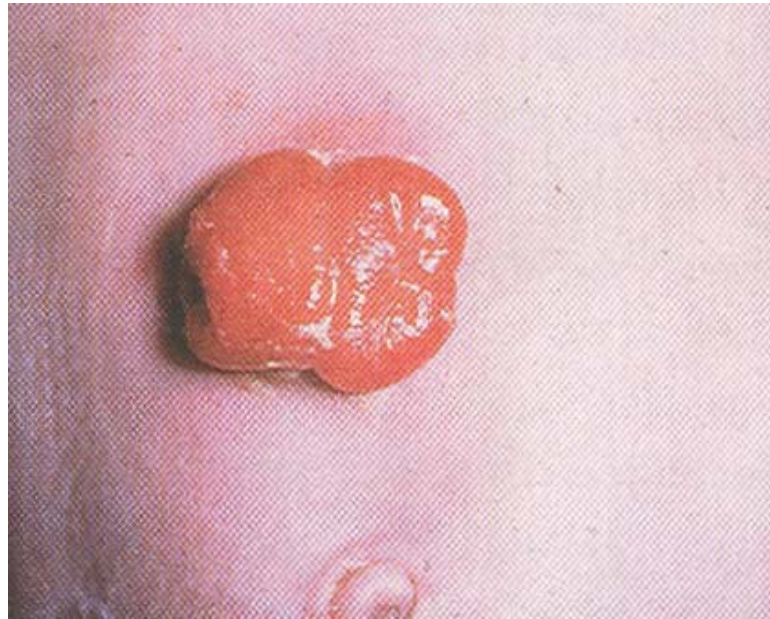
Errors of Rotation

A. 1st Stage:

Exomphalos (congenital omphalocele)



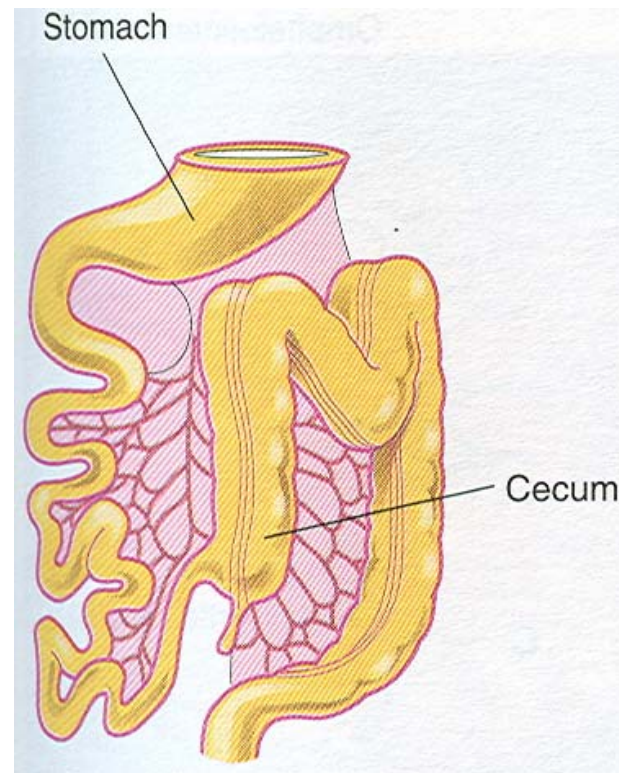
Gastroschisis: Defect in the ant. Abdominal wall; permits extrusion of viscera; present lateral to umbilicus.



Errors of Rotation

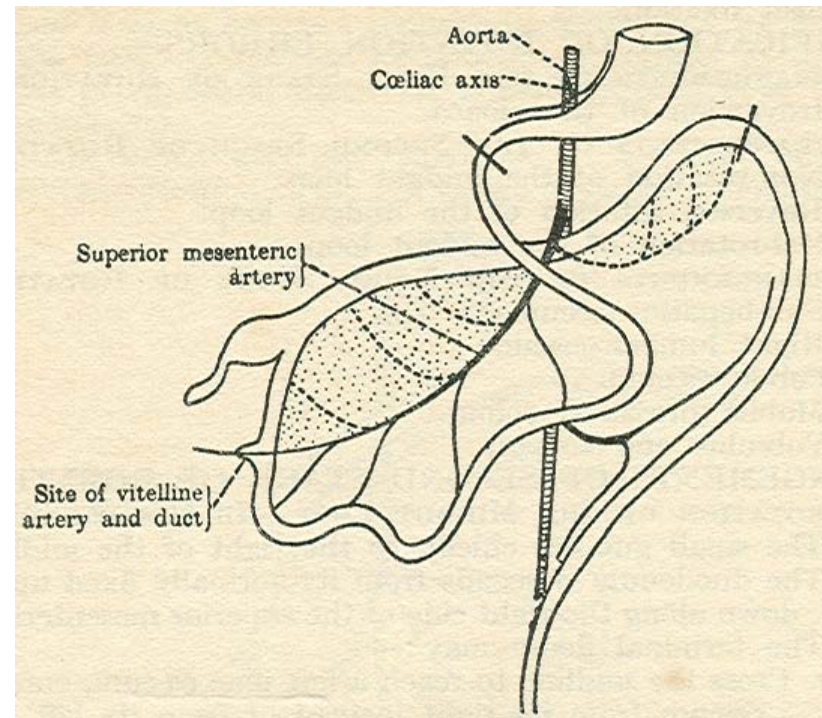
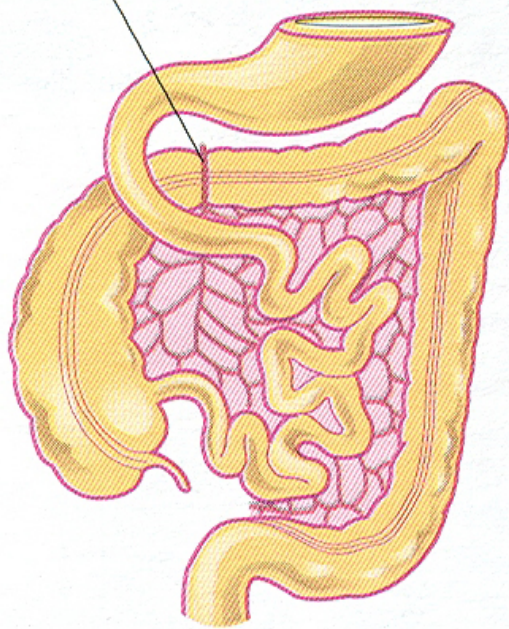
B. 2nd Stage:

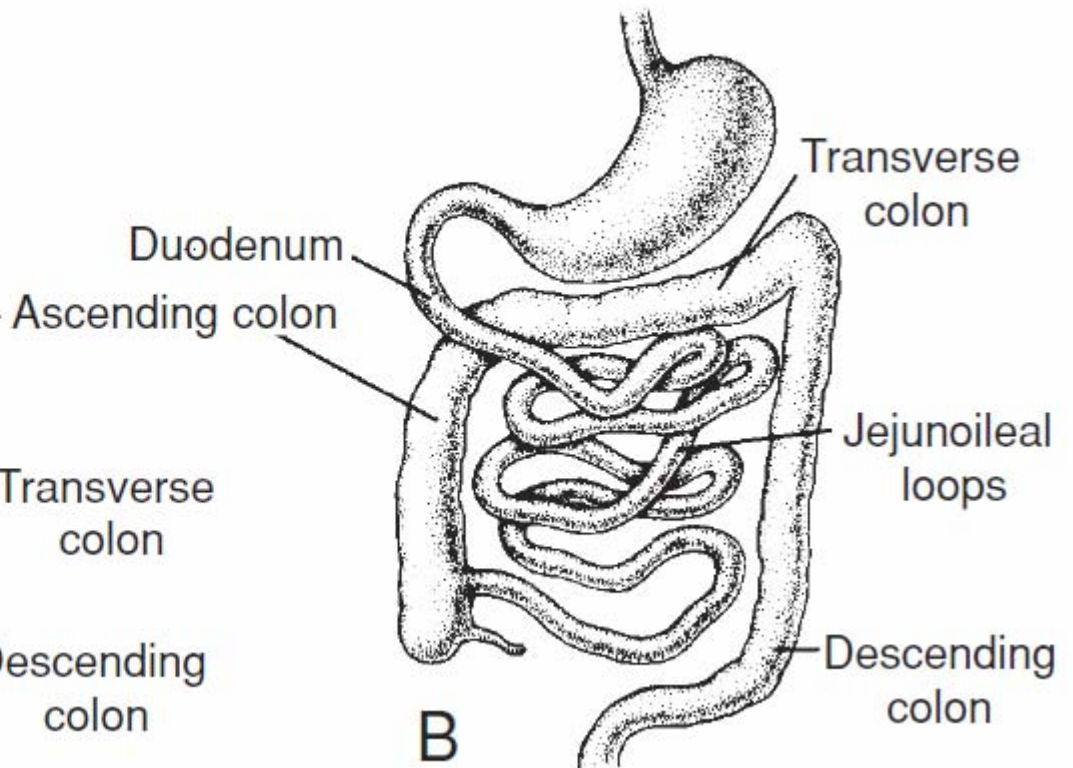
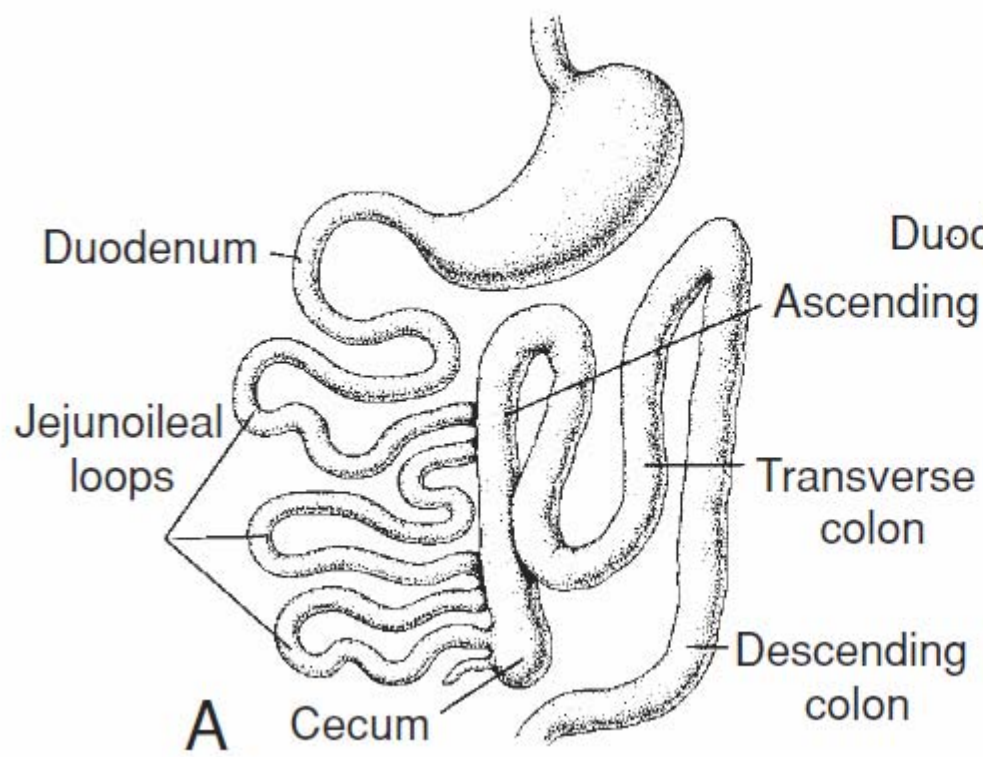
1. Nonrotation



2. Reversed rotation

Superior mesenteric artery
(compressing transverse colon)

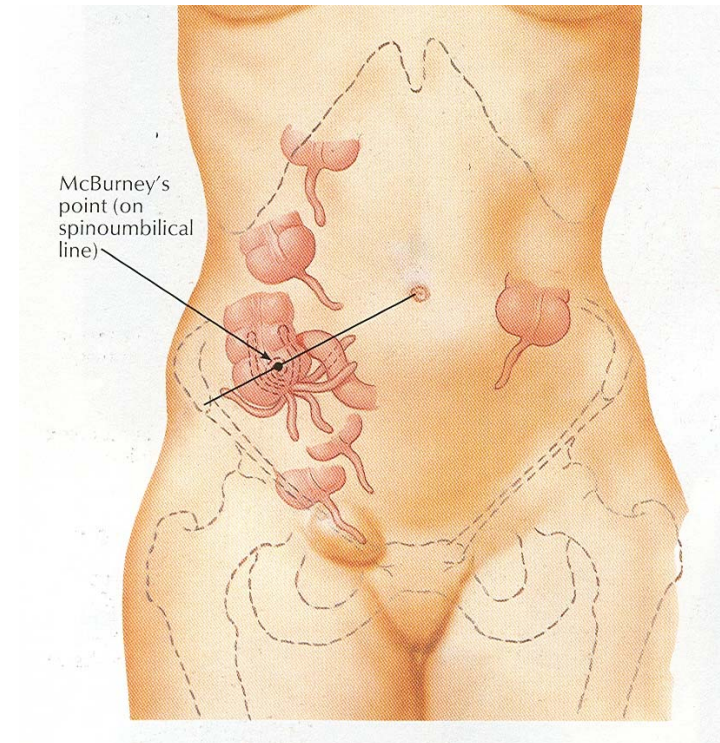
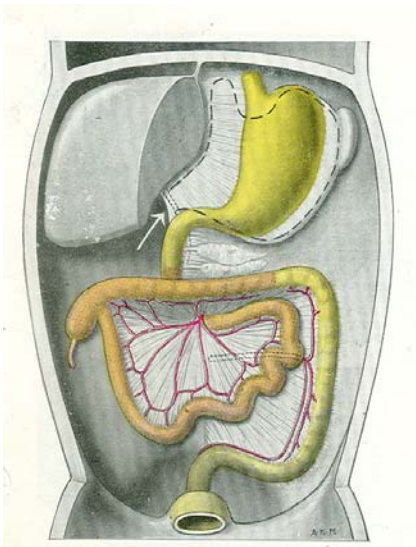


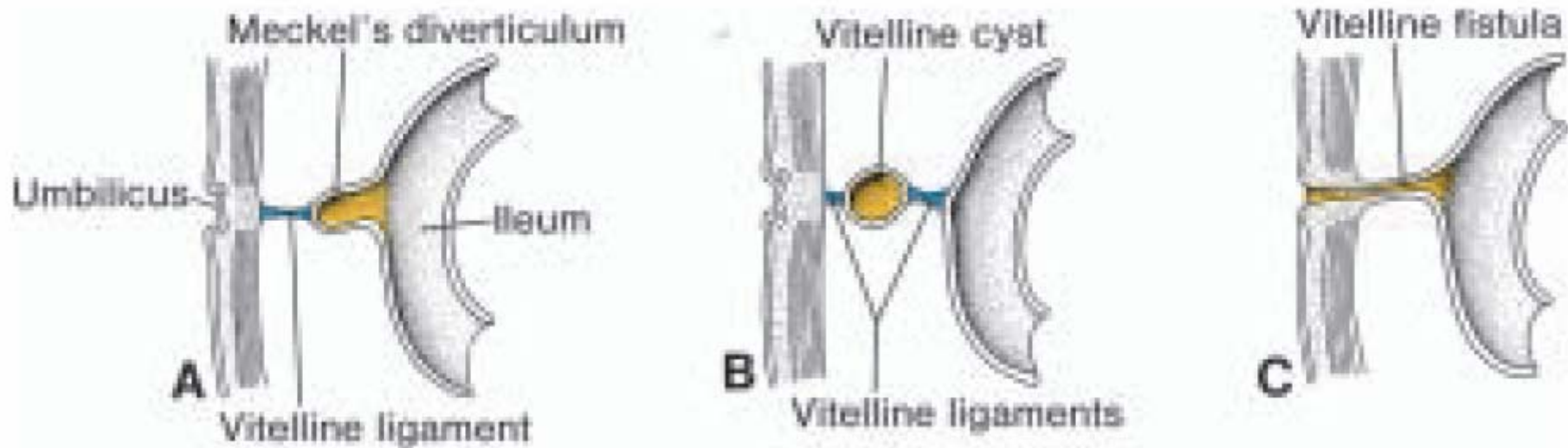


Errors of Rotation

C. 3rd Stage:

1. Abnormal positions of caecum
 - a. Subhepatic
 - b. Right lumbar
 - c. Pelvic
 - d. Mobile caecum



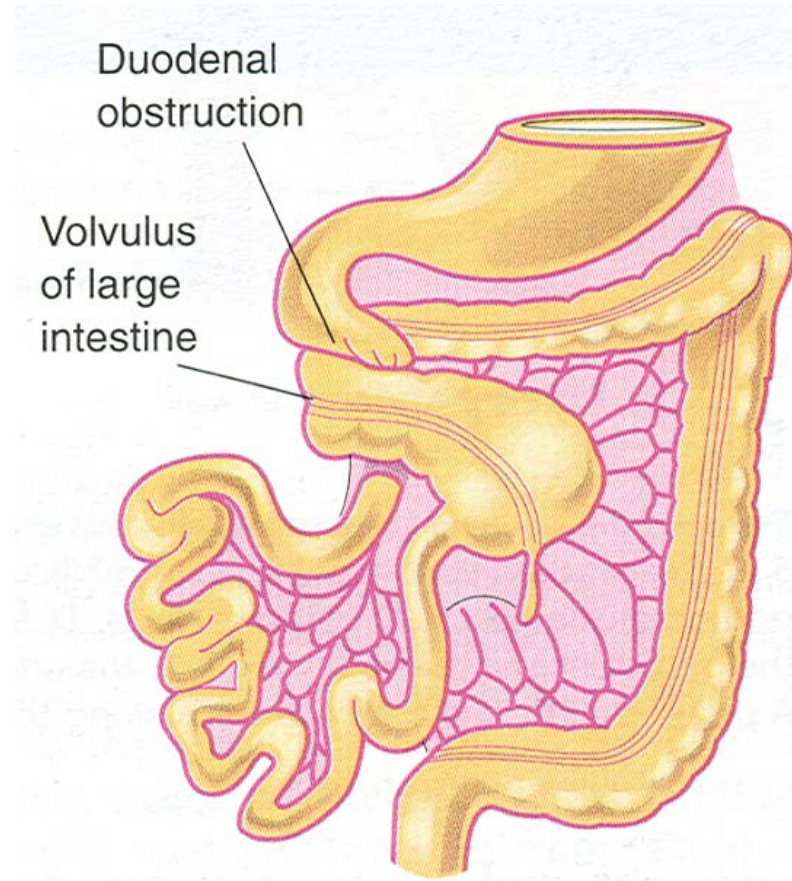


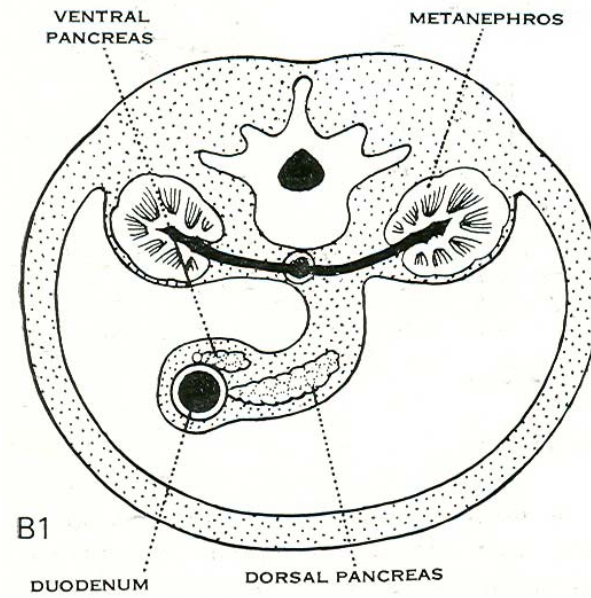
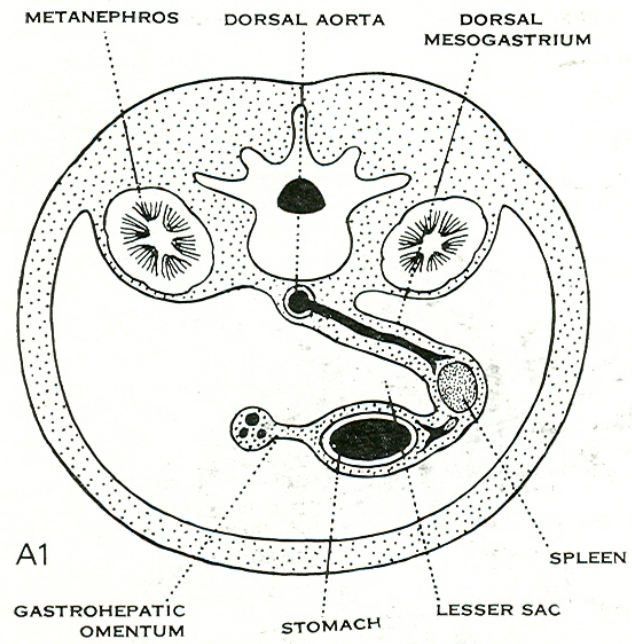
Meckel's diverticulum: remanant of vitello-intestinal duct, in 2% cases, 40-50 cm from ileocaecal junction, from antimesenteric border of ileum.

2. Persistent mesenteries of ascending/descending colon; duodenum



3. Volvulus





Development of Abdominal Digestive System

Oesophagus, stomach and duodenum up to the hepatopancreatic ampulla, liver, gall bladder and most of pancreas develop from the **fore gut**. These structures are supplied by branches of coeliac artery.

Lower part of duodenum, lower part head & uncinate process of pancreas, jejunum, ileum, appendix, caecum, ascending colon and right two thirds of transverse colon develop from **mid gut**. These structures are supplied by branches of superior mesenteric artery.

Left 1/3 of transverse colon, descending colon, sigmoid colon, rectum and upper anal canal develop from **hind gut**. These structures are supplied by branches of inferior mesenteric artery.

Stomach

develops as a fusiform dilatation of fore gut.

Spleen

develops by fusion of thickenings of mesodermal tissue (splenules) in the dorsal mesogastrium.

Duodenum:

- a) proximal part of duodenum (up to the hepatopancreatic ampulla) develops from the caudal part of fore gut and
- b) the remaining lower part of duodenum develops from the cranial part of mid gut.

Caecum

develops from a conical caecal bud which arises from the post arterial segment of mid gut.

The apical part of the caecal bud forms the [appendix](#).

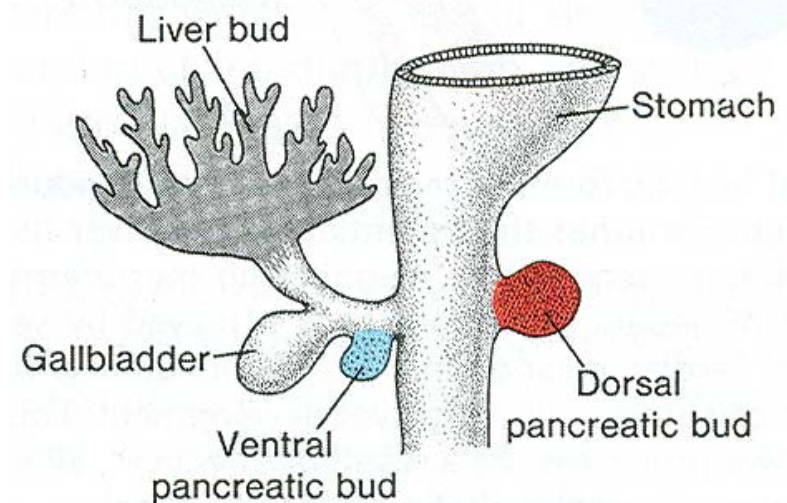
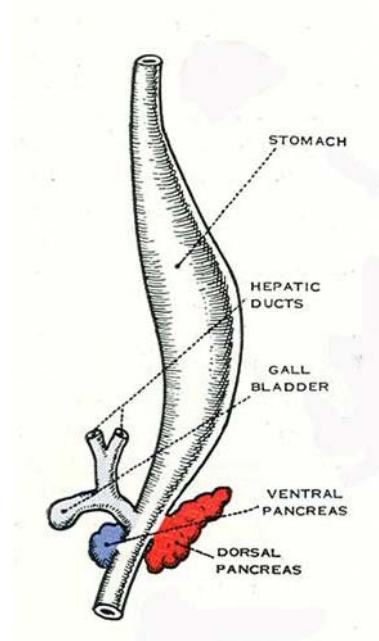
Liver

develops from the pars hepatica of endodermal hepatic diverticulum (which develops from the ventral wall of fore gut near its caudal end).

Glisson's capsule develops from the surrounding mesoderm of septum transversum.

Gall bladder and cystic duct

develop from the pars cystica of hepatic diverticulum.



Pancreas:

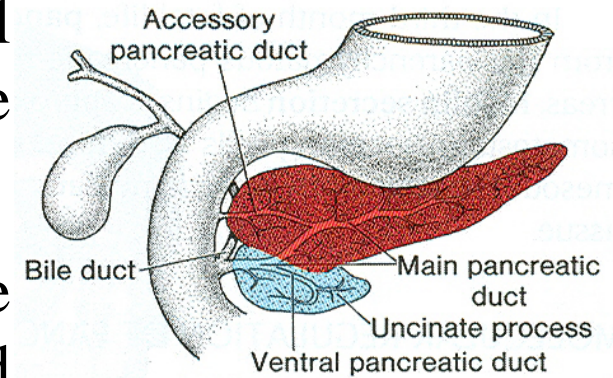
a) upper part of head, neck, body and tail of pancreas develop from dorsal pancreatic bud (which arises from end of fore gut) &

b) lower part of head and uncinate process develop from ventral pancreatic bud (which arises from beginning of mid gut).

c) duct of VPB and distal part of duct of DPB form the main pancreatic duct.

d) proximal part of duct of DPB forms the accessory pancreatic duct.

e) some terminal acini from the pancreatic ducts get separated to form the islets of Langerhans.



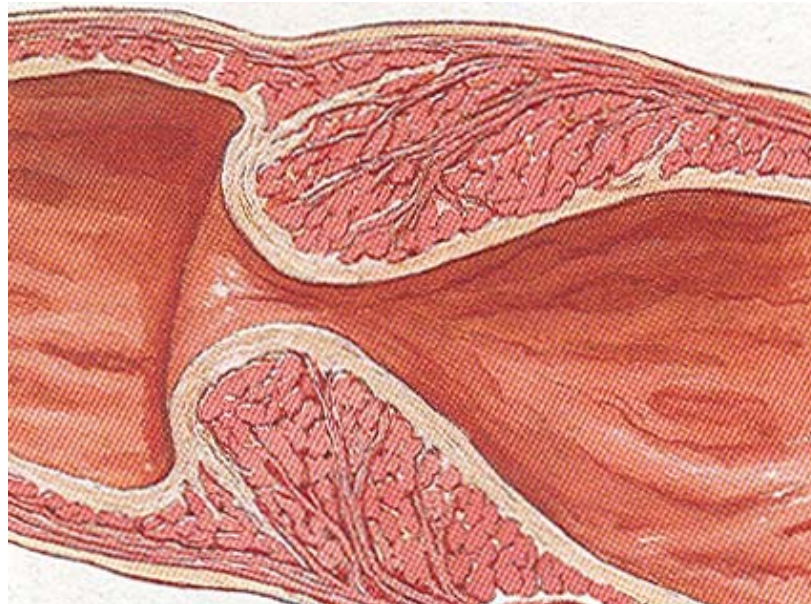
Congenital anomalies

1. Foregut:

a) Tracheo-oesophageal fistula

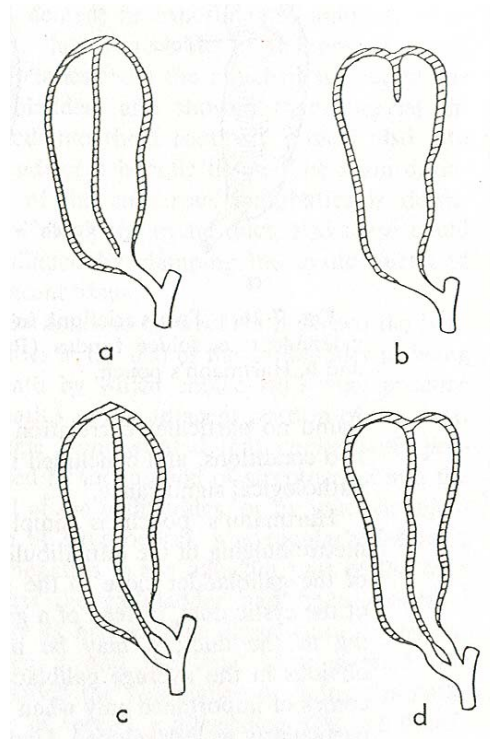
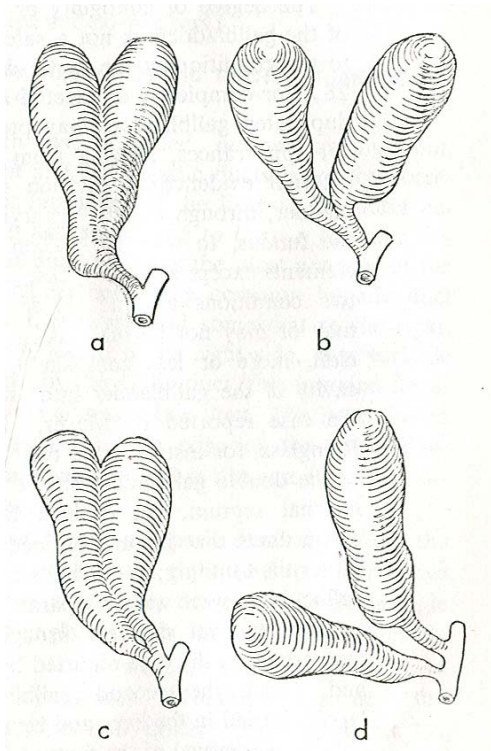
b) Stomach:

Congenital pyloric stenosis – Marked muscular thickening; projectile vomiting; surgery (pyloromyotomy)



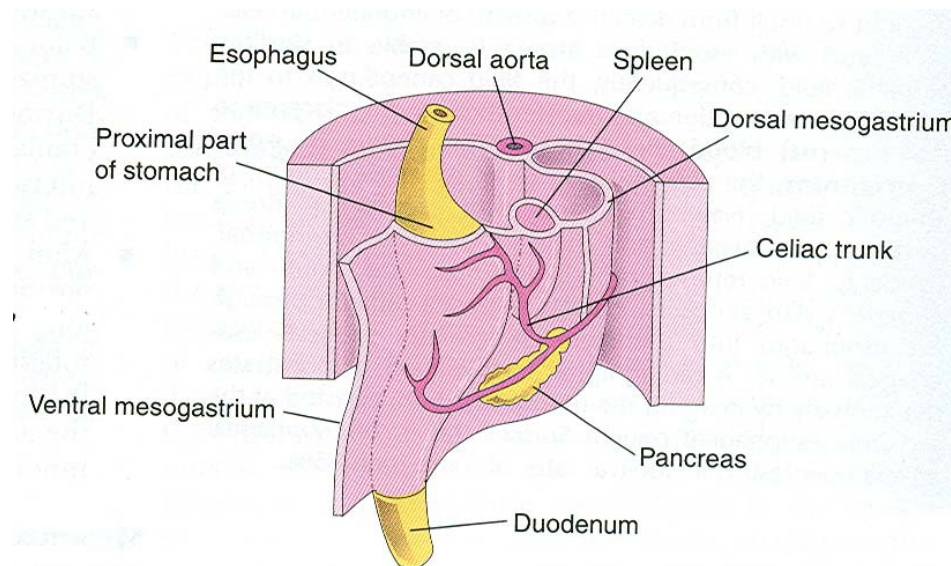
b) Gall bladder:

- i) Absence of cystic duct (Sessile Bladder)
- ii) Double gall bladder/ septate
- iii) Persistence of mesentery



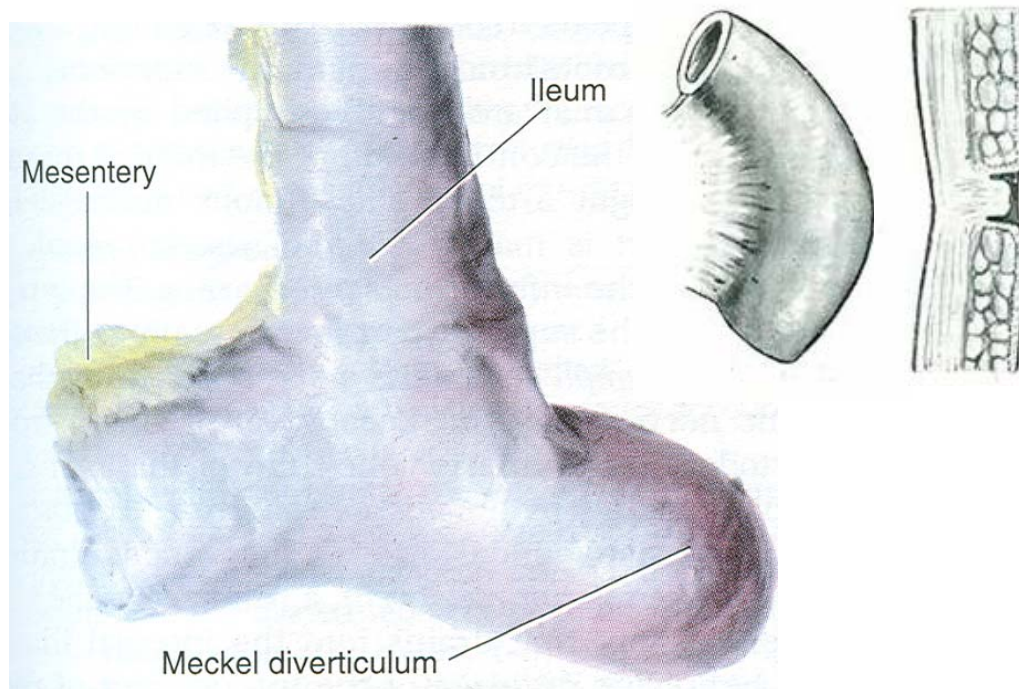
c) Spleen:

- i) Lobulated
- ii) Accessory

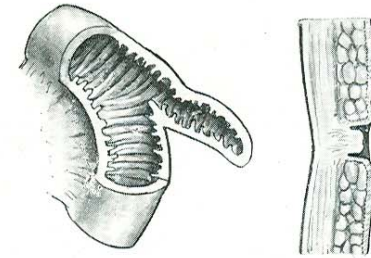


Midgut:

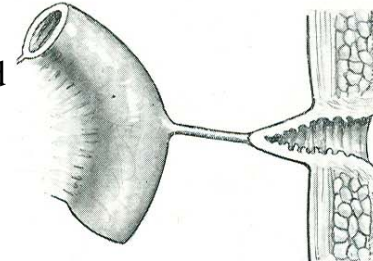
- i) Errors of rotation
- ii) Duodenal atresia/ stenosis
- iii) Meckel's diverticulum



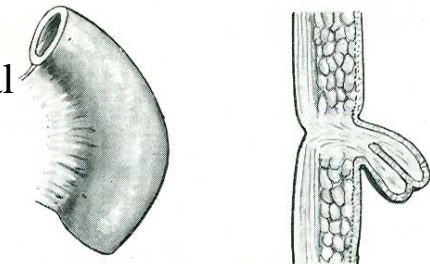
a) Meckel's
divericulum



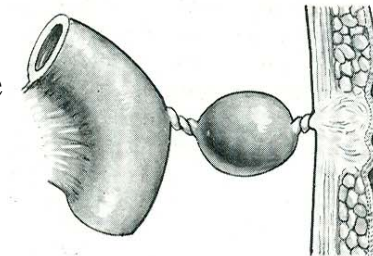
b) Vitelline cord
with umbilical
sinus



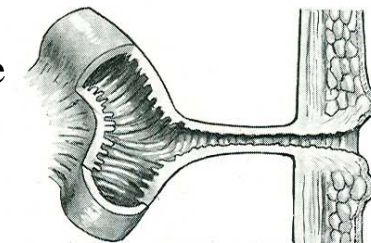
c) Umbilical
sinus



d) Vitelline
cyst



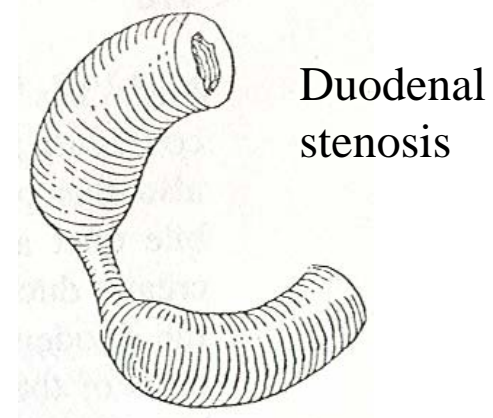
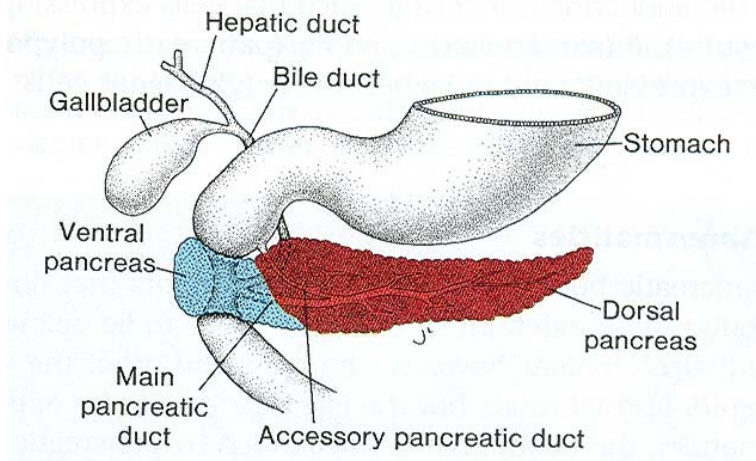
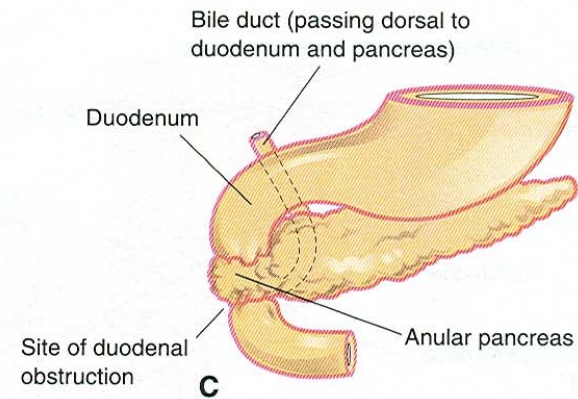
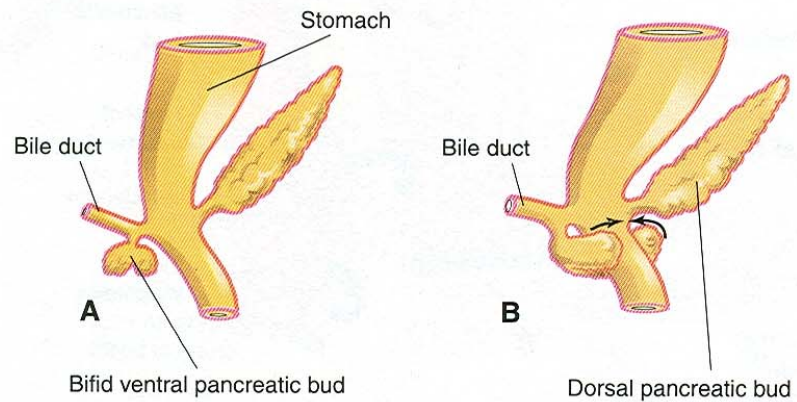
e) Vitelline
fistula



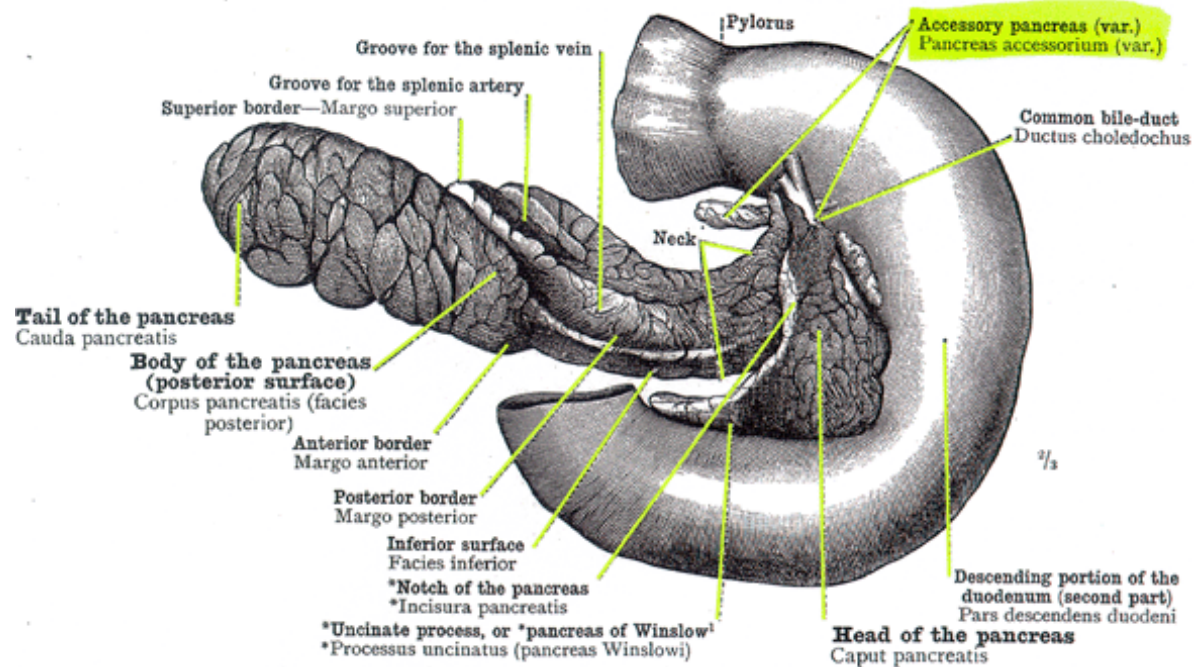
iv) Pancreas:

a) Annular

b) Divided



c) Accessory pancreas



v. Caecum

a) Anomalies of shape

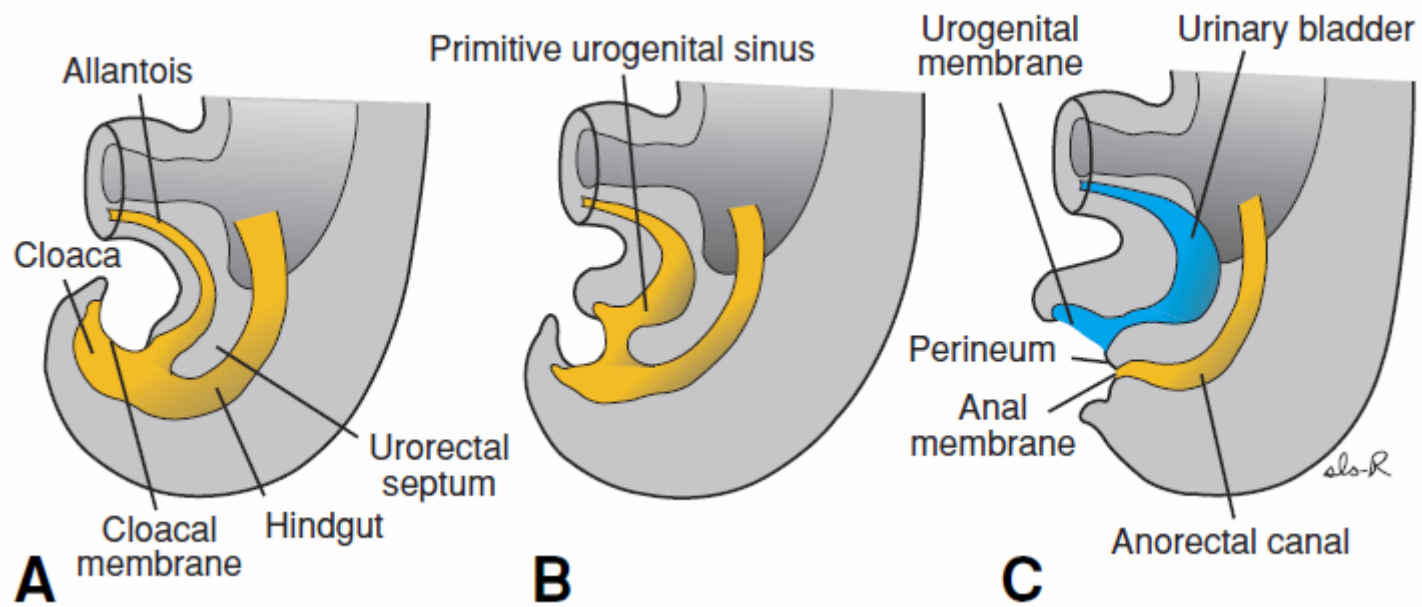
i) Conical (Infantile: 2%)

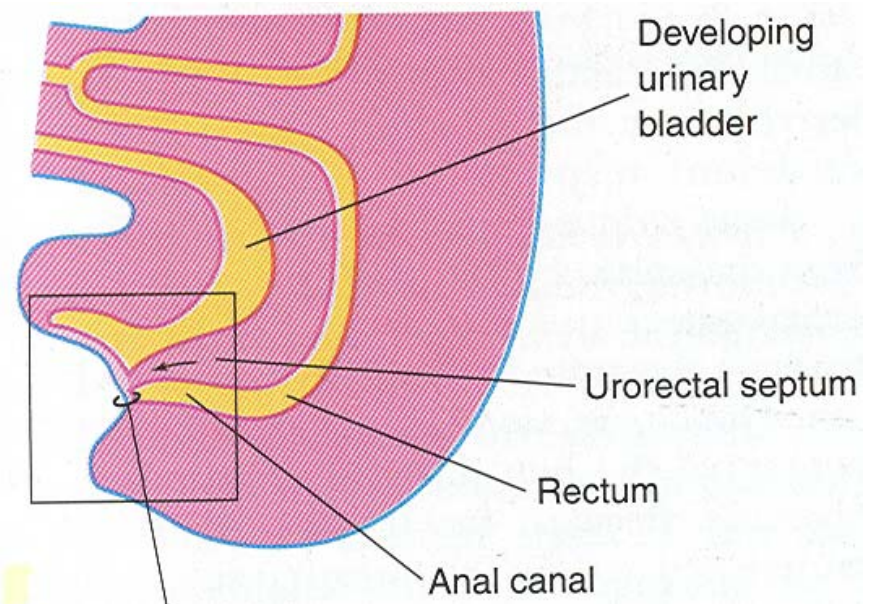
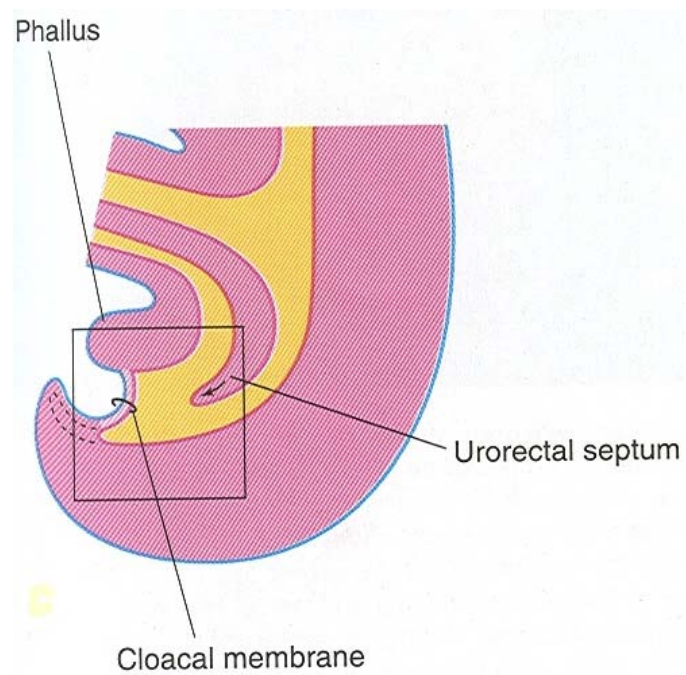
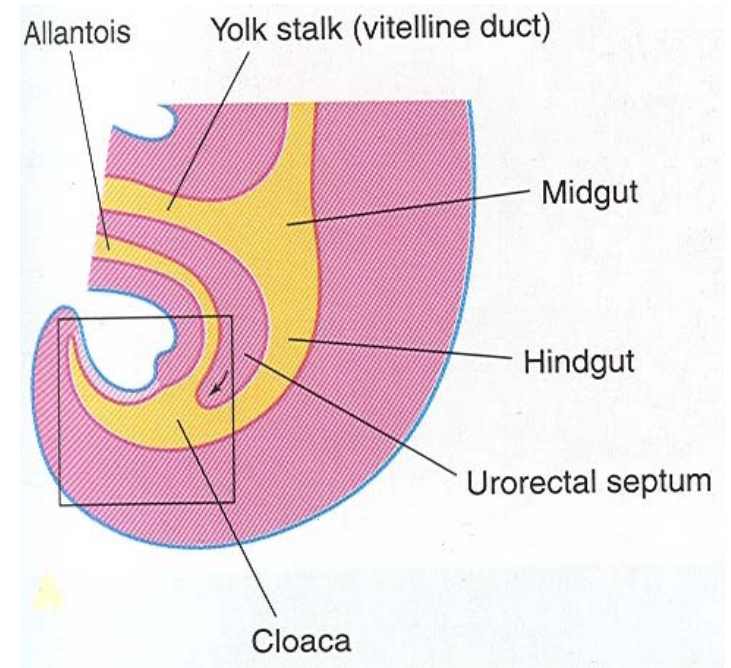
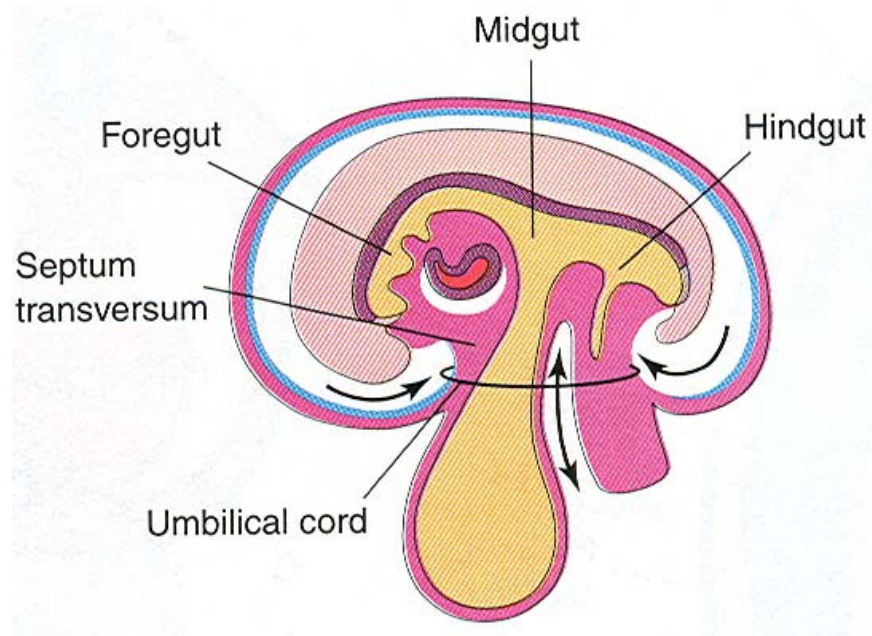
ii) Quadrate (3%)

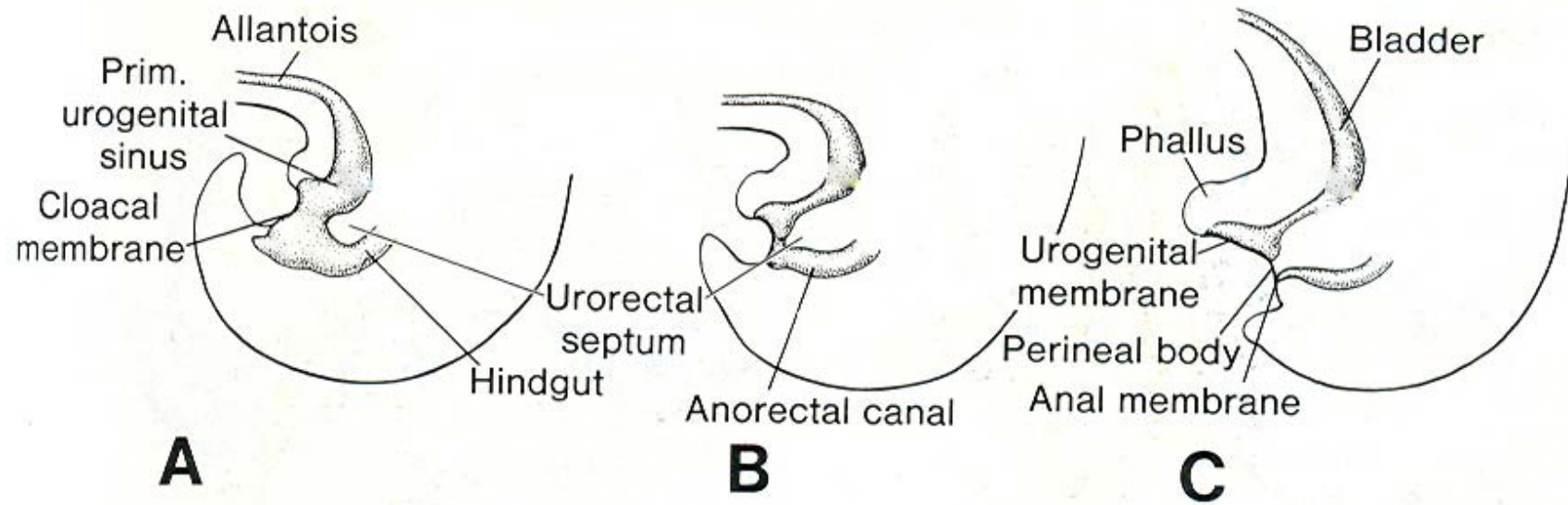
iii) Hyper position of appendix (4-5%)

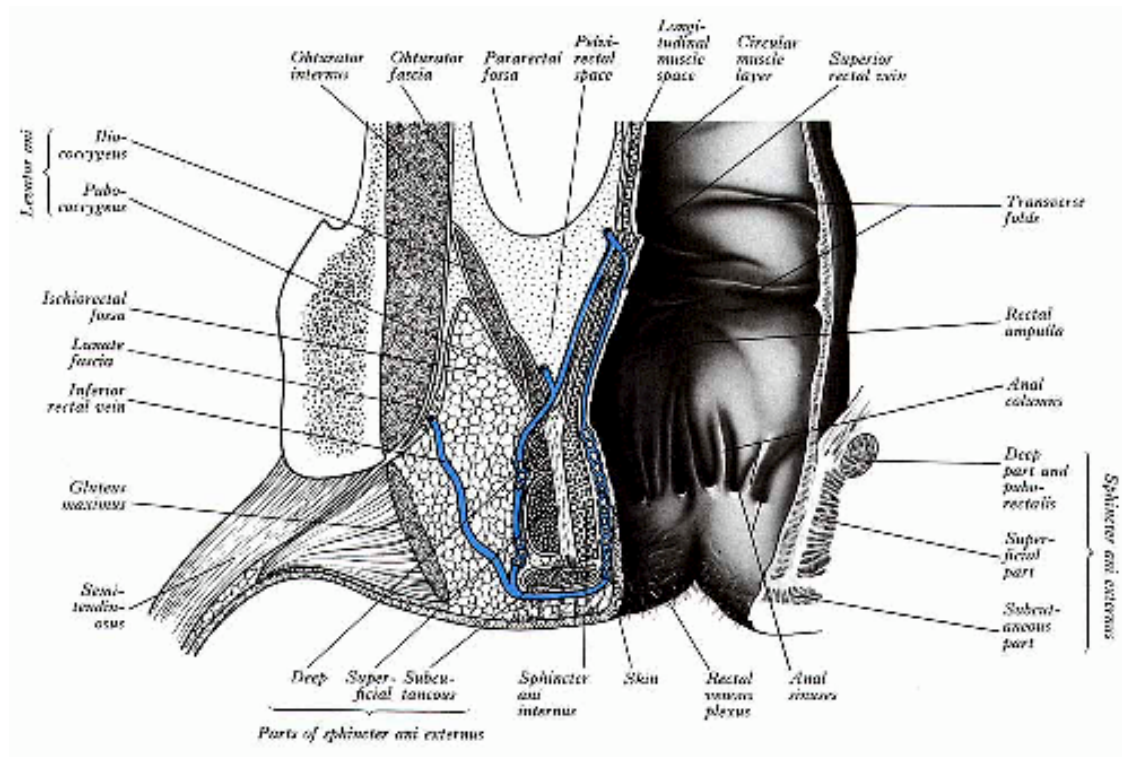
b) Anomalies of position

(errors of rotation)









Congenital anomalies of hindgut:

1. Imperforate anus
2. Fistulae:
 - a. Rectovesical
 - b. Rectourethral
 - c. Rectovaginal
- 3. Congenital megacolon(Hirschsprung disease).**
- 4. Rectoanal atresia**

Imperforate anus

