AN ANALYSIS OF GUT AND GENITO-URINARY ANOMALIES ENCOUNTERED IN EMERGENCY SURGERY

Dissertation submitted to

THE TAMILNADU Dr. M. G. R MEDICAL UNIVERSITY,CHENNAI

With partial fulfilment of the regulations

For the award of the Degree of

M.S.(General Surgery)

Branch I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

MARCH 2009

BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of **Dr. S.SKANDA** on **"AN ANALYSIS OF GUT AND GENITOURINARY ANOMALIES IN EMERGENCY SURGERY"** during his M.S. (General Surgery) course from May 2006 to March 2009 at the Government Kilpauk Medical College and Government Royapettah Hospital, Chennai.

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ACKNOWLEDGEMENTS

I am most pleased to acknowledge the Dean **Prof. Dr. M.DHANAPAL, M.D., D.M.** of Kilpauk Medical College and Hospital for the opportunity to conduct this study in theDepartment of Surgery, Kilpauk Medical College.

My deepest gratitude to my guide and mentor, **PROF.S UDAYAKUMAR, M.S.** Chief of Surgery Unit II who has inspired me immeasurably during my training as a postgraduate student.

I also acknowledge the invaluable advice and counseling received from **PROF.P.RAVI, M.S,** Head of the Department of General Surgery, Govt. Royapettah Hospital

I wish to express my personal appreciation to the Head of the Department of General Surgery, Kilpauk Medical College, **PROF G. GUNASEELAN,M.S.,** with whom I have been blessed to have an opportunity to work.

This study would not have been possible without the support of my Unit assistant professors **DR.T.S.JAYASHREE D.G.O, M.S,** and **DR.S.THIRUNAVUKKARASU M.S,** and to whom I owe my surgical training .

I wish to express my gratitude to my co post-graduates and my colleagues for their invaluable help in collection of patient data.

Lastly, I thank **MY PATIENTS** not only for their consent and co-operation towards the preparation of this study but also for the privilege of practicing our craft.

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INTRODUCTION

It is said that the abdomen is a Pandora's Box, a Black Box, a Box of Surprises and so on….the list is endless. The general surgeon has to have in his armamentarium not only a good command of anatomy, but also of surgical physiology and pathology in even the most normal of cases. Even when no surprise is anticipated, one field where the surgeon has to be most careful and cautious is while performing emergency operations. In the rarest of rare circumstances, the surgeon is stumped by certain variations of normal anatomy. However, the biggest and most fascinating challenges in adult emergency surgery come when one encounters a certain aberration in embryological development, forcing the surgeon to improvise on the spot. This particular study deals with genitourinary and gut associated embryological anomalies encountered by us in Royapettah Hospital, Kilpauk Medical College during the period between June 2006 and October 2008. The unusual aspect of this study is the emphasis on emergency surgery.

Another factor which is unique is that this study deals with adult patients that we have encountered in Royapettah Hospital, emphasizing the fact that a gut or genitourinary embryological anomaly is not within the purview of the pediatric surgeon alone. A good and competent general surgeon must be able to deal with and manage such cases with little mental preparation.

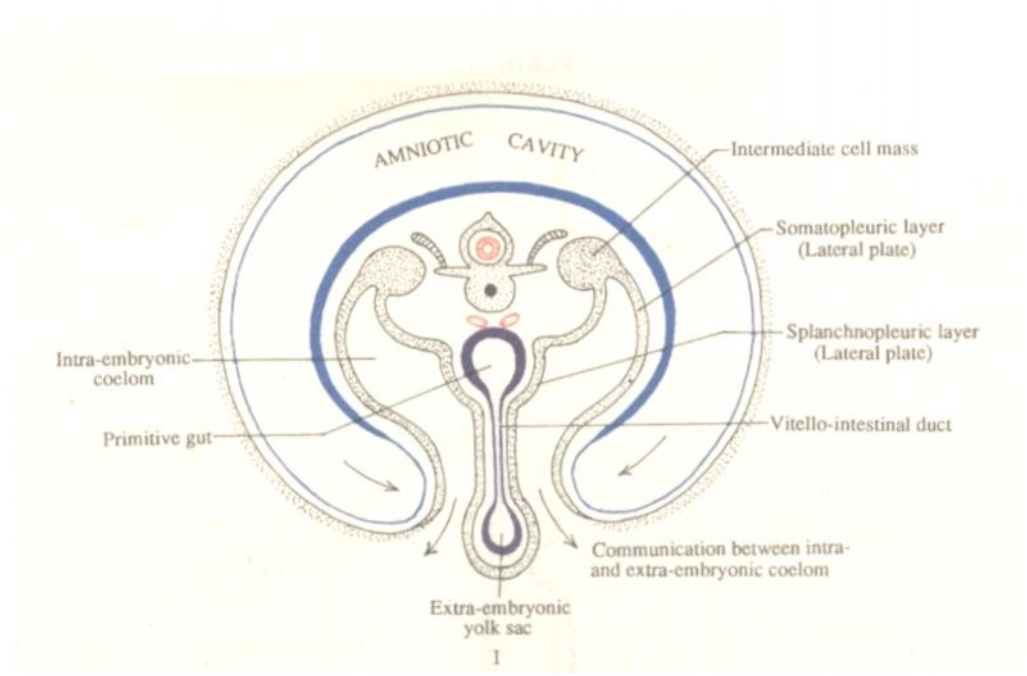
The spectrum in this study varies from the humble and ubiquitous Meckel's Diverticulum, which is invariably asymptomatic, to a rare case of Hirschsprung's disease in an adult male, which presented as intestinal obstruction. The surgeon dealing with the problem must be familiar with a whole gamut of procedures from a simple obstruction release, to doing a Ladd's procedure for gut malrotation! This study shows us the commonest embryological anomalies that we have encountered in the study period, their pattern of distribution, modes of presentation, and methods of management.

THE ALIMENTARY SYSTEM

The origin of the alimentary system is traced from the *trilaminar germ disc*, which, until the 6th week of intra- uterine life, is flattened and pear shaped. The cells occupying the central embryonic area grow more rapidly than those at the periphery. As a result, the embryonic area shows the development of head fold, tail fold and two lateral folds at the end of the 3rd week. These foldings convert the flattened germ disc into cylindrical embryo and help to establish the contour of the embryo.**(Plate I)**

The *cephalo – caudal folding* is mainly caused by the rapid longitudinal growth of the central nervous system, and the lateral foldings by the formation of the rapidly growing somites. During the process of embryonic folds the growth of the yolk sac stagnates. Eventually the amniotic cavity enlarges and surrounds the outer surface of the embryo so that the latter floats in amniotic fluid. The cephalo-caudal and lateral foldings converge on the ventral aspect of the embryo in such a manner that the amniotic membrane forms a tubular investment around the connecting stalk and converts it into the *umbilical cord.*

PLATES I AND II. THE LATERAL FOLDS OF THE EMBRYO



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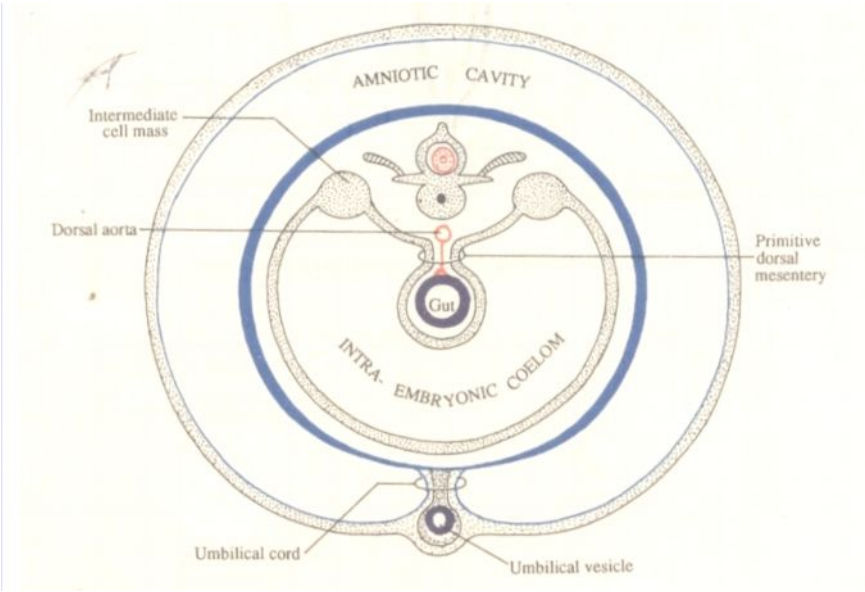


The yolk sac cavity on the other hand, is reduced in size and undergoes an hour glass contraction by the convergence of folds on the ventral surface of the embryo. The part of the yolk sac within the embryonic folds forms the *primitive gut.* The extra embryonic part of the yolk sac forms the *umbilical vesicle* which projects into the foetal surface of the placenta beneath the amniotic membrane. The umbilical vesicle is temporarily connected to the primitive gut by the *vitello-intestinal duct* which passes through the umbilical cord.**(Plate II)**

Head fold:

The germ disc bends ventrally (towards the yolk sac) around the cranial end of the notochord so that the forebrain vesicle occupies the cephalic extremity of the embryo. The part of the yolk sac contained within the head fold is known as the *fore gut,* which is initially a blind ectodermal tube and communicates caudally with the *midgut* through the *anterior intestinal portal.* Dorsal wall of the fore gut is supported by the notochord and the hind brain vesicle of the neural tube. Due to altered position of the

cephalic portion of the germ disc, the ventral wall of the foregut presents successively in cranio - caudal direction the following:

- The buccopharyngeal membrane
- Pericardial sac with the endothelial tube of primitive heart dorsal to the sac
- Septum transversum

The bucco-pharyngeal membrane lies at the bottom of a depression of surface ectoderm called *stomodaeum* or the primitive oral cavity. This is bounded on the cephalic side by the forebrain vesicle, and caudally by the bulging of the pericardial sac. Later the pericardial sac is displaced more caudally from the floor of the stomodeum by the ventral ends of the branchial or pharyngeal arches. The bucco-pharyngeal membrane ruptures by the 4^{th} week, thus the foregut communicates with the exterior through the stomodeum and the amniotic fluid gains entrance into the gut.

Prior to the formation of fold the pericardial sac lies on the dorsal wall of the yolk sac. The head fold brings the pericardial sac on the ventral wall of the foregut with a reversal of its surfaces. The foregut is separated from the pericardial sac by the angioblastic type of mesenchymal cells from which the heart develops, hence this area of the mesodermal cells is known as the *cardiogenic plate.*

The septum transversum is a mesodermal sheet which lies caudal to the pericardial sac and the heart and extends from the body wall to the ventral surface of the foregut. Dorsal to the septum and on each side of the gut, the coelomic duct passes through the *pleuro-peritoneal* canal. Subsequently the canal is closed on each side by the development of the *pleuro – peritoneal membrane*, which is gradually peeled off from the body wall by the down growth of the lung bud; thus a complete partition between the thorax and the abdomen is developed from the septum transversum. Caudal surface of the septum transversum is invaded by the hepatic bud which grows as an endodermal diverticulum from the junction of the fore and mid guts. The hepatic bud divides the septum into a cephalic horizontal portion known as *pars diaphragmatica,* and a caudal sagittal part called the

pars mesenterica. The former differentiates into an upper layer forming the fibrous pericardium, and a lower layer forming the *antero-median diaphragm.* The pars mesenterica forms the fibrous stroma, falciform and coronary ligaments of the liver, and the lesser omentum.

Tail fold:

Around the caudal end of the notochord and the neural tube the germ disc bends ventrally, enclosing a part of the yolk sac which forms the *hind gut.* The connecting stalk with the allanto enteric diverticulum is carried on to the ventral wall of the hind gut. The connecting stalk forms subsequently the umbilical cord. The distal part of the allantoic canal persists as the *urachus* which is connected to the apex of the developing urinary bladder. The proximal part of the canal incorporates with the hind gut. Caudal to the umbilical cord the ventral wall of the hind gut is formed by the bilaminar *cloacal membrane*, which lies at the bottom of a depression of surface ectoderm known as *ectodermal cloaca.* The latter is limited on each side by a raised margin, the *genital fold,* through which the secondary mesodermal cells migrate from the primitive streak to form the infra

umbilical part of the ventral body wall. The dorsal wall of the hind gut is supported by the notochord and the neural tube, and its bottom is occupied by the primitive streak and primitive node.

The allanto-enteric diverticulum divides the hind gut into pre and post allantoic parts. The *pre-allantoic part* forms a narrow tube and is continuous proximally with the midgut. The junction between the mid and hind guts is known as the *posterior intestinal portal.* The *post-allantoic part* of the hind gut dilates and forms the *endodermal cloaca.* The cloacal membrane gradually shifts to the ventro-caudal wall of the cloaca, partly due to the growth of the infra-umbilical part of the ventral body wall and partly by the regression of the primitive streak. At a later date, the cloacal membrane is divided by the caudal edge of the *uro-rectal septum* into a ventral *uro-genital membrane* and a dorsal *anal membrane.* Both membranes disappear at the beginning of the 5th week, and establish the formation of the uro-genital and anal orifices.**(Plate III)**

PLATE III. MEDIAN SAGITTAL SECTION (BEFORE HEAD AND TAIL FOLDS)

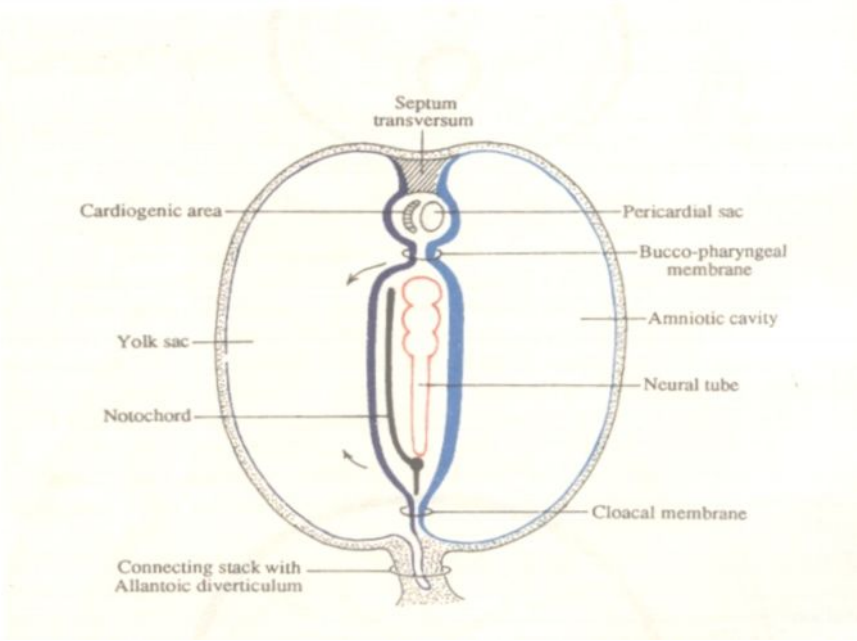
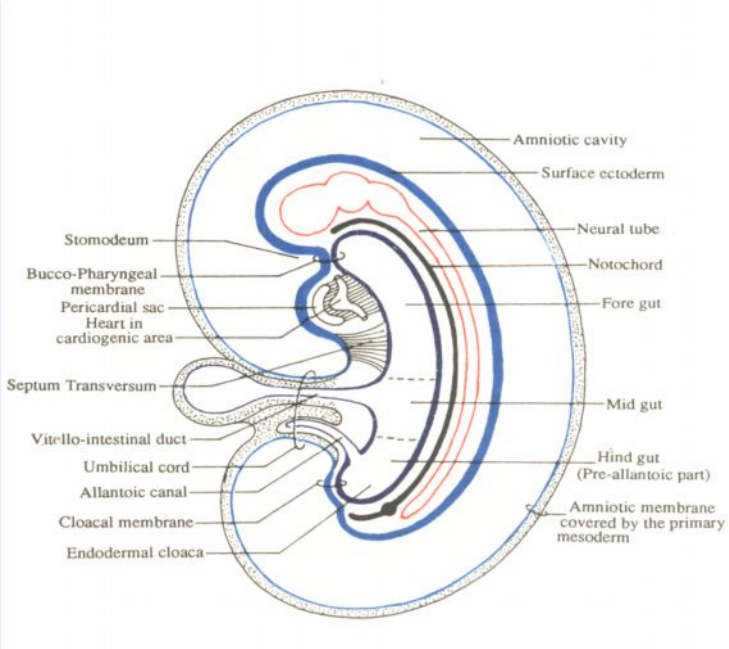


PLATE IV. MEDIAN SAGITTAL SECTION (AFTER HEAD AND TAIL FOLDS)



Lateral folds:

Along with the formation of the head and tail folds the lateral margins of the germ disc bend ventrally enclosing a part of the yolk sac, the *midgut.* The two lateral folds thus formed meet with the cephalo-caudal folds around the *primitive umbilical ring* of the ventral wall of the cylindrical embryo, where the layer of the *amnio-ectodermal junction* is reflected over the umbilical cord. The part of the yolk sac that lies outside the embryonic folds is ballooned out from the midgut as the *umbilical vesicle,* which is lodged beneath the amniotic membrane of the placenta. The umbilical vesicle is temporarily connected to the midgut by the *vitello-intestinal duct* which runs through the umbilical cord.**(Plate IV)**

Now the somatopleuric layer of the lateral plate mesoderm lines the inner aspect of the parietal body wall and the ventral surface of the intermediate cell mass of the embryo. The splanchno-pleuric layer invests the ventro-lateral surfaces of the primitive gut and is reflected dorsally as a bilaminar fold of the *dorsal mesentery of the gut.* The root of the dorsal

mesentery extends longitudinally along the ventral surface of the primitive vertebra and contains initially *two dorsal aortae* which have developed from the undifferentiated mesenchyme of this area. The somatopleure and splanchnopleure are continuous with each other at the root of the dorsal mesentery. The intermediate cell mass now lies in an explicable position for the subsequent development of the genital and urinary systems.

At first the intra-embryonic coelom communicates with the extra-embryonic coelom in the umbilical cord around the vitello-intestinal duct. Although the vitelline duct disappears early, the communication between intra and extra embryonic coelom persists for a while within the umbilical cord (5^{th} – 10^{th} week). During this period a part of the midgut loop herniates into the extra embryonic coelom forming *physiological hernia.*

DIFFERENTIATION IN THE ENDODERMAL LAYER

A detailed account of the endodermal development of the primitive three guts is discussed later. Only the gut derivatives are mentioned here.

Fore gut derivatives:

- Epithelial lining of the pharynx, oesophagus, stomach, duodenum upto ampulla of Vater and the mucous membrane of the tongue.
- Epithelial lining of the respiratory system, auditory tube and tympanic cavity
- Parenchyma of the tonsil, thyroid, parathyroid, thymus, liver and pancreas

Mid gut derivatives:

- Mucous membrane of the alimentary tract extending from the duodenum distal to the ampulla of Vater upto the junction the right two thirds and left one third of the transverse colon
- Meckel's diverticulum, when it exists, is derived from the persistent proximal part of the vitello-intestinal duct.

Hind gut derivatives:

- Mucous membrane of the digestive tube extending from the left third of the transverse colon upto the muco-cutaneous junction of the anal canal (pectinate line)
- Most of the mucous membrane of the urinary bladder and urethra, parenchyma of the prostate, bulbo-urethral or greater vestibular glands
- Epithelial lining of the vagina
- Primitive sex cells – from the dorsal wall of the hind gut

DEVELOPMENT OF THE ALIMENTARY SYSTEM

The alimentary system is developed from the endoderm of the definitive or secondary yolk sac. The primitive tubular gut extends in the median plane from the *buccopharyngeal membrane* at its cephalic end to the cloacal membrane at its caudal end. It is subdivided into three parts – the fore gut, mid and hind gut.

The Foregut is that part of the yolk sac which is contained within the head fold of the embryo. At the cephalic end, it is separated from the *stomodaeum* or the primitive mouth cavity by a bilaminar bucco-pharyngeal membrane which ruptures around the 4^{th} week of development. Thus the fore gut communicates with the exterior through the stomodeum. Caudally the fore gut is continuous with the mid gut, and the junction between them is known as the *anterior intestinal portal.* In adult, the position of the anterior intestinal portal roughly corresponds with the termination of the bile duct in the second part of the duodenum. Ventrally, at the future pharyngo-esophageal junction the foregut presents a median *laryngo – tracheal groove* which bulges

forward and then caudally to develop into the lower part of the respiratory passages including the lung buds. Therefore, the foregut may be divided into the *pre-laryngeal* or the cephalic part and the *post-laryngeal* or the caudal part. The cephalic part of the fore gut develops into the pharynx and a part of the floor of the definitive mouth cavity. With the development of the branchial arches and the pharyngeal pouches, the cephalic part serves as a birth place for the embryogenesis of the tongue, submandibular and sublingual glands, the palatine tonsils, the auditory tubes and the middle ear and the lower respiratory passages. The caudal part of the fore gut forms the oesophagus, stomach and proximal part of the duodenum (upto the termination of the Ampulla of Vater). The liver including biliary apparatus and the pancreas bud out from the caudal fore gut.

The Mid gut is embraced by the two lateral folds of the embryo, and extends from the *anterior to the posterior intestinal portals*; at the level of the latter it joins with the hind gut. The posterior intestinal portal

usually corresponds in adults with the junction of the proximal two-thirds and distal one-third of the transverse colon. In the early embryonic life, the midgut communicates ventrally with the extra-embryonic part of the yolk sac via the *vitello-intestinal duct*. Under normal conditions, the vitello-intestinal duct disappears entirely at about the 4^{th} or 5^{th} week. The development of a *caecal diverticulum* from the mid gut divides it into pre and post- caecal segments. The distal part of duodenum below the ampulla of Vater, the jejunum and ileum are developed from the *pre-caecal segment;* the post-caecal segment forms the caecum, the vermiform appendix, the ascending colon and the proximal two-thirds of the transverse colon.

The Hind gut is the part of the yolk sac which is contained within the tailfold of the embryo. Its caudal end is separated from the surface ectoderm by a bilaminar *cloacal membrane*. The ventral wall of the hind gut is drawn out as the *allanto-enteric diverticulum* which extends blindly up to the fetal end of the umbilical cord. The attachment of the allantoic diverticulum divides the hind gut into pre and post allantoic

parts. The pre-allantoic part gives rise to the distal one-third of the transverse colon, the descending and the sigmoid colon. The post-allantoic part is dilated to form the *endodermal cloaca* which is subsequently divided by the urorectal septum into a dorsal part, the primitive rectum and a ventral part, the primitive *urogenital sinus*. The cloacal membrane disappears somewhat later (about the 6^{th} week), after the rupture of the bucco-pharyngeal membrane. This establishes communications of the alimentary and the urogenital systems with the exterior. The endodermal cloaca develops into the rectum, proximal part of the anal canal, most of the mucus linings of the urinary bladder and the urethra.

The cephalic or pharyngeal part of the primitive gut is flanked by a layer of fused somatic and splanchnic mesoderm. Rest of the primitive gut in the abdomino-thoracic region is invested by the splanchnopleuric layer of the lateral pate mesoderm of the intra-embryonic coelom. It may be appreciated that while the endodermal layer forms the mucus lining of the gut and the structures derived from it, the musculature, stroma and the serous coat of the gut are derived

from the splachno pleuric layer of the lateral plate. Moreover the bilateral apposition of the splanchnopleuric layers after enclosing the gut forms the *primitive dorsal mesentery*, which suspends the gut from the dorsal body wall and conveys blood supply to it. The abdominal part of the foregut is supplied by the *celiac artery*, the midgut by the *superior mesenteric artery* and the hind gut by the *inferior mesenteric artery.*

CAUDAL PART (POST-LARYNGEAL) OF THE FORE GUT

Below the laryngo-tracheal groove the remaining part of the fore gut consists of oesophageal, gastric, and duodenal segments; two offshoots-the *liver* (including the biliary apparatus), and the *pancreas* are derived from the duodenal part of the fore gut. Although the spleen belongs to the haemopoietic tissue, it is described here because the spleen is derived from the mesenchyme of the dorsal mesogastrium.

Oesophagus

It is a narrow tube extending from the caudal margin of the commencement of the laryngeal diverticulum to the proximal end of the fusiform dilatation of

the rudimentary stomach. Dorsally it is suspended from the primordial vertebral bodies by a broad dorsal mesentery (*meso-oesophagus*), the root of which contains fused dorsal aortae. Ventrally, the oesophagus is separated in the upper part from the developing heart and the pericardial sac by the *laryngo-tracheal tube* and the *lung buds* which are the off-shoot of the fore gut; in the lower part of the ventral wall it is related with the septum transversum (Fig. 12.2). On each side of the oesophagus lies the *coelomic duct* (pleural passage) into which the corresponding lung bud invaginates. Therefore, the *thoracic mediastinum includes those structures which are contained within the ventral and dorsal meso-oesophagus.*

At first the oesophagus is short, but it elongates with the descent of the heart and caudal migration of the septum transversum. During elongation its lumen is obliterated for some time by the proliferation of the lining epithelium. The final restoration of the lumen takes place by a process of recanalisation or vacuolization due to the destruction of the central part of the epithelial plug.

Histogenesis of the Oesophagus

Initially the mucous membrane of the oesophagus is *ciliated columnar,* then it is changed into *simple columnar epithelium,* and finally is transformed into

the *stratified squamous epithelium* by a process of metaplasia. The splanchnic mesoderm around the oesophagus is differentiated to form its muscular and other coats. The muscles in the upper two-thirds are striated, whereas in the lower third they are unstriped.

Congenital anomalies of the oesophagus

- Tracheo-oesophageal fistula
- Oesophageal atresia
- Cardiospasm.

Tracheo-oesophageal fistula

Trachea is separated from oesophagus by the *tracheo-oesophageal septum,* which is a coronally oriented partition and extends caudo-cranially until it reaches the laryngeal inlet. The *tracheo-oesophageal fistula* consists of various types. Oesophagus communicates with the trachea at its bifurcation; this is due to failure of caudal growth of tracheo-oesophageal septum. Sometimes the laryngeal inlet is closed and the fistula at the tracheal bifurcation is the only communication. This takes place when the septum grows cranio-caudally in reverse direction. Rarely the oesophagus is

completely disconnected from the pharynx, when the septum grows obliquely caudal wards from the ventral to dorsal walls of fore gut.

Oesophageal atresia

When a segment of the oesophagus fails to recanalise, *atresia* results. Atresia may affect any part of the oesophagus, but is more frequent below the bifurcation of the trachea. The foetus is unable to swallow amniotic fluid, and *hydramnios* is associated with this anomaly.

Cardiospasm (achalasia)

This is a variety of neuro-muscular incoordination at the cardio-oesophageal junction, which fails to open properly when a peristaltic wave reaches. As a result, the part proximal to the junction dilates, although there is no mechanical obstruction. *Cardiospasm occurs due to congenital agenesis of the nerve cells* in the myenteric and submucous plexuses of the oesophageal wall.

Stomach

The stomach develops as a fusiform dilatation from the lower part of the foregut during the fourth or fifth week. Initially it is placed in the median plane, and its dorsal border grows more rapidly than the ventral border showing the formation of the rudimentary fundus and greater curvature. Eventually the pyloric part is pushed more ventrally, and this makes the ventral border concave which forms the future lesser curvature.

The *ventral border* of the stomach is suspended from the anterior body wall by the *ventral* mesogastrium which is derived from the septum transversum. The subsequent entrance of the hepatic bud within the ventral mesogastrium divides the latter into two parts-the portion between the stomach and the liver forms the lesser omentum, and the part between the liver and the body wall persists as the falciform and the coronary ligaments of the liver. The dorsal border of the stomach is attached to the posterior body wall by \h& dorsal mesogastrium which is a part of the dorsal mesentery of the primitive gut. The spleen is developed from the mesenchyme under cover of the left layer of the dorsal mesogastrium. The portion of the dorsal mesogastrium between the spleen and the stomach

forms the gastro-splenic ligament and that between the spleen and the dorsal body wall forms later the lieno-renal ligament.

Subsequently, the stomach undergoes rotation to the right through an angle of about 90° around a vertical axis, so that the original left surface becomes ventral and the right surface occupies the dorsal position. This provides an explanation why the left vagus nerve supplies the antero-superior surface, and the right vagus nerve appears on the postero-inferior surface of the stomach. The factors for the rotation of the stomach may be as follows:

(a) Rapid enlargement of the right lobe of liver allows it to occupy the right part of the abdomen, and the spleen gradually shifts on the left side.

(b) The omental bursa (lesser sac) extends to the left, dorsal to the stomach, and pushes the dorsal mesogastrium caudally beyond the stomach forming the folds of the rudimentary greater omentum.

(c) The apparent rotation of the stomach takes place due to an active growth process, and the dorsal mesogastrium gradually shifts to the left from the dorsal border until it reaches the adult position.

Histogenesis of the stomach

The gastric glands become apparent in the third month. The oxyntic cells and the zymogenic cells are differentiated during the fourth month, and the function of rennin starts probably in the fifth month.

Anomalies of the stomach

Congenital hypertrophic pyloric stenosis

Is a congenital defect of the stomach, where the circular muscle of the pyloric sphincter is hypertrophied, and a neuro-muscular inco-ordi-nation supervenes. The child suffers from progressive vomiting which is manifested between two weeks and two months of the post-natal life. The boys are more frequent victims of this anomaly than girls.

Duodenum

The duodenum is developed from two sources - *above and including the hepato- pancreatic ampulla it is developed from the terminal part of the fore gut; below the ampulla it is derived from the proximal part of the mid gut.*

The anterior intestinal portal (the junction of the fore-and midguts) corresponds with the termination of the hepato-pancreatic ampulla at the major duodenal papilla. The dual development of the duodenum is represented by the nature of the blood supply. The definitive duodenum is supplied by an anastomosis between the superior and the inferior pan-creatico-duodenal arteries; the former is a sub-branch of the celiac artery (artery of the fore gut), and latter is a branch of the superior mesenteric artery (artery of the mid gut).

The primitive duodenum presents a loop with a ventral convexity, and is placed in the median plane. The fore gut segment of its ventral convex margin is attached to the septum transversum by the ventral mesogastrium. Close to the anterior intestinal portal, the fore gut endoderm evaginates from the convex margin as a hepato-pancreatic bud, which extends head wards and ventrally within the ventral mesogastrium.

From this bud develop the liver, the biliary apparatus and the lower part of the head of the pancreas. The dorsal concave margin of the primitive duodenum is suspended from the dorsal body wall by the meso-duodenum. From this margin the dorsal pancreatic bud develops as an endodermal outgrowth of the fore gut, somewhat cephalic to the hepato-pancreatic bud. The dorsal pancreatic bud extends dorsally and slightly head-wards within the meso-duodenum, and develops into the entire pancreas except the lower part of its head.

Before the duodenum reaches the adult position, it passes through three successive phases -rotation, fixation, and axial rotation.

(a) Rotation

When the developing large gut (transverse colon) re-enters the abdominal cavity from the physiological umbilical hernia and undergoes subsequent rotation to the right, it pushes the loop of the primitive duodenum to the right side and presses the latter against the dorsal body wall. As a result, the ventral convex margin of the duodenum forms its lateral border and the original right surface becomes posterior.

(b) Fixation

The peritoneum covering the posterior surface of the rotated duodenum comes in contact with the parietal peritoneum of the dorsal body wall. Subsequently both layers of the peritoneum disappear by zygosis. Hence, most of the posterior surface of the definitive duodenum becomes non-peritoneal except the proximal 2.5 cm. of the first part.

(c) Axial rotation

Due to differences in the growth of the duodenal wall, the second part of duodenum undergoes axial rotation so that the hepato-pancreatic bud appears on its postero-medial aspect and the dorsal pancreatic bud shifts more ventrally. The axial rotation permits the head of the pancreas to meet with the rest of that organ and establishes intercommunication between the duct systems of the dorsal and the ventral pancreatic buds.

During the eighth week the lumen of the primitive duodenum is obliterated by the proliferation of the lining endodermal cells. Usually by the end of the third month the lumen is re-canalised.

Anomalies of the duodenum

Duodenal atresia (Plate VI)

On rare occasions the duodenal lumen fails to re-canalise, and the portion of the gut above the obstruction is distended. The atresia may affect any part of the duodenum, but is more frequent below the hepato-pancreatic ampulla.

The liver and biliary apparatus

The liver and its duct system develop during the fourth week as an endodermal diverticulum from the terminal part of the fore gut at the ventral border of the primitive duodenum. The hepatic bud thus formed grows ventrally and head wards within the ventral mesogastrium, and reaches the septum transversum where it divides into right and left branches. Each branch gives rise to clusters of liver cells, the hepatic cylinders, which eventually form two solid masses. These solid masses, initially of equal sizes, are the rudiments of the right and left lobes showing the bilateral symmetry of the liver. The cells of these masses differentiate into plates of

liver cells, which branch and anastomose with one another and form the parenchyma of the liver. The mesenchyme of the septum transversum persists as the fibro-areolar stroma of the liver. The growth of the liver plates within the septum transversum disturbs the longitudinal course of the vitelline and umbilical veins. Eventually these vessels break up into the capillary network and join secondarily with the hepatic sinusoids which develop in situ between the plates of the liver cells.

The original stalk of the hepatic diverticulum forms the bile duct, and its two branches persist as the right and the left hepatic ducts. Within the liver parenchyma the hepatic ducts branch repeatedly, acquire lumen as they grow, and arc converted into intra-hepatic part of the biliary passages. A cystic bud develops from the bile duct slightly caudal to the commencement of the hepatic ducts; this bud at first presents a solid outgrowth, which acquires lumen somewhat later and forms the gall bladder and the cystic duct. Another endodermal outgrowth, the ventral pancreatic bud, appears from the duodenal end of the bile duct. With the axial rotation of the second part of the duodenum, the bile duct diverticulum conveying the ventral pancreatic bud with it shifts from the original position

(the outer convex border) to the postero-medial aspect of the duodenum. Thus the adult position of the bile duct is established.

The growth of the liver in the early part of development is profuse, affecting both the lobes almost equally. The enlarged liver mass occupies most of the upper abdomen and projects caudally displacing the derivatives of the mid gut for a temporary period in the extra-embryonic part of the umbilical coelom. The portion of the ventral mesogastrium between the liver and the body wall is thinned out to form the falciform and the coronary ligaments of the liver. The part of the ventral mesogastrium between the liver and the stomach persists as the lesser omentum. The extension of the coelomic cavity cephalic to the liver separates the latter from the septum transversum which persists as the major component of the diaphragm. Thus the subphrenic recesses separate the liver from the diaphragm almost completely except the bare area of the liver where the primitive adhesion persists between them.

The extensive enlargement of the liver in the intra-uterine life is variously explained. The liver parenchyma gets abundant oxygenated blood, which stimulates its profuse growth. Moreover, the foetal liver is haematopoietic in function. The differentiation of the blood cells lakes place in several loci in

the interval between the liver cells and the wall of the hepatic sinusoids. The blood cells are set free into the sinusoids and thence into the foetal circulation. The walls of the sinusoids are lined by Kupffer's cells which are phagocytic. The haematopoietic function of the liver diminishes sufficiently in the last two months of pregnancy. This is associated with the progressive reduction of its size which mostly affects the left lobe. In the early part of development the weight of the liver forms about 10% of the body weight, whereas in the later part it comes down to about 5% of the body weight.

Histogenesis of the liver

At first the liver cells are arranged as portal lobules around the interlobular bile ductules, the radicles of portal vein and the branches of the hepatic artery. Then the liver cells are organised along the intra-lobular sinusoids and around the central veins (tributaries of the hepatic veins), showing the formation of the classical liver lobules. These alterations of the arrangements of the liver cells suggest that primarily the liver is exocrine in function, but in the later part of development it shows greater importance as an endocrine organ. The bile secretion usually starts from the fifth month.

Congenital Malformations of the Biliary Apparatus (Plate V)

Gall bladder

(a) Atretic gall bladder

When the cystic bud fails to canalise, the gall bladder may be rudimentary or even absent (agenesis).

(b) Septate gall bladder

Sometimes a mucous fold subdivides the interior of the gall bladder into two compartments. The fold may be longitudinal or transverse **(Plate V- A)**

(c) Double gall bladder

Sometimes two separate gall bladders arc connected by a single cystic duct or by separate cystic ducts. **(Plate V - B)**

(d) Intra-hepatic gall bladder

On rare occasions the gall bladder is completely buried within substance of the liver. **(Plate V - C)**

(e) Mobile or floating gall bladder

Frequently the gall bladder is suspended from the liver by a mesentery and enjoys greater range of movement.

(f) Hepato-cystic communications

Sometimes the intra-hepatic bile ductules pierce the bladder bed and open directly into the body of the gall bladder. Hence, after the surgical removal of the gall bladder a drainage tube is inserted, as a rule, close to the bladder bed.

(g) Hartmann's pouch

It is an occasional pouch arising from the neck of gall bladder and extends downwards and backwards. Whether this pouch is congenital or pathological, the matter remains undecided. **(Plate V - D)**

h) Absence of the cystic duct

In such condition, the bladder neck directly opens into the bile duct.

Bile Duct

(i) Atresia of the bile duct

This is due to failure of canalisation of the biliary passages. When the atresia affects the common bile duct, the gall bladder and the bile passages above the obstruction are distended. This condition is manifested by the persistent and progressive jaundice in a new born baby.

PLATE V. ANOMALIES OF THE BILIARY TREE

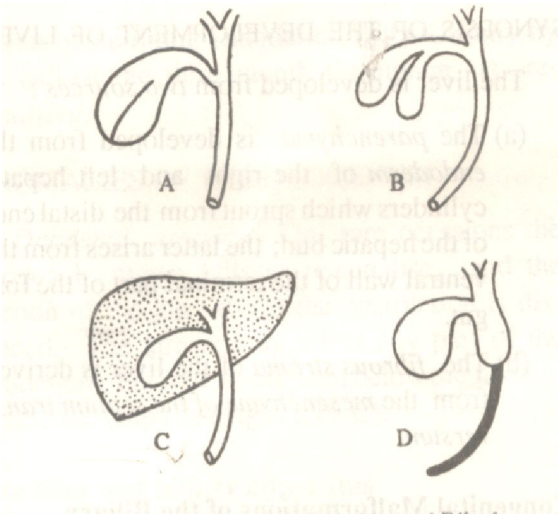
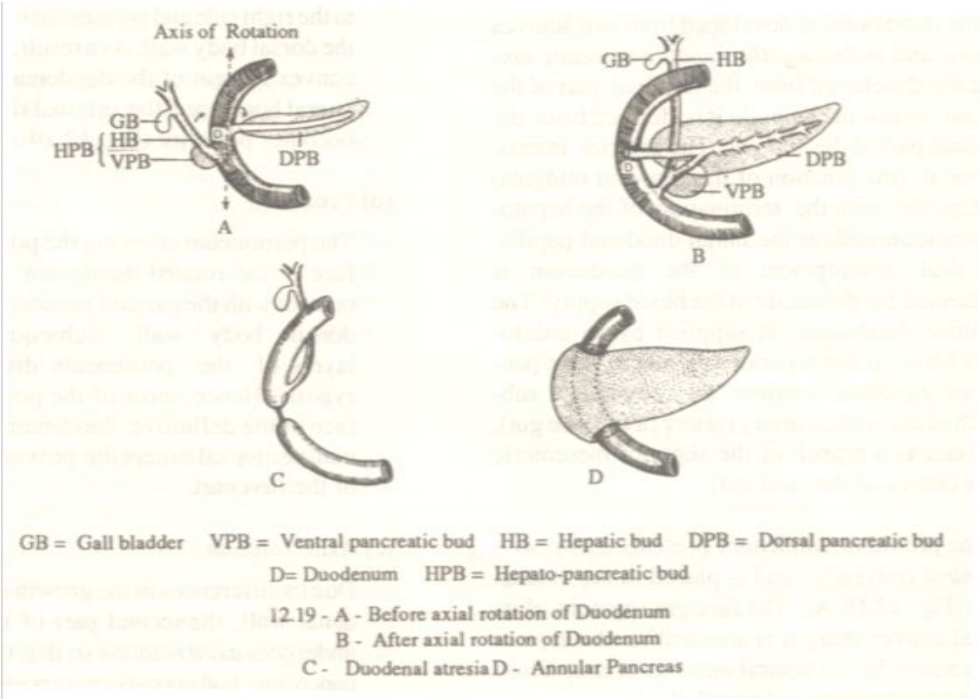


PLATE VI. ANOMALIES OF THE DUODENUM AND PANCREAS



GB = Gall bladder VPB = Ventral pancreatic bud HB = Hepatic bud DPB = Dorsal pancreatic bud
D= Duodenum HPB = Hepato-pancreatic bud

12.19 - A - Before axial rotation of Duodenum
B - After axial rotation of Duodenum
C - Duodenal atresia D - Annular Pancreas

The Pancreas

The pancreas owes its development from the endoderm of the duodenal segment of the fore gut. It develops in two parts - dorsal, and ventral.

The dorsal part arises as a diverticulum during the fourth week from the dorsal wall of the primitive duodenum slightly cephalic to the hepatic bud; it extends dorsally and somewhat head wards in the meso-duodenum and dorsal mesogastrium. The entire pancreas except the lower part of the head is derived from the dorsal part.

The ventral part develops as a diverticulum from the commencement of the primitive bile duct at the junction of the fore and mid guts. At first the ventral bud forms a bilobed structure which on subsequent fusion is converted into a single mass. Lower part of the head of the pancreas including the uncinate process develops from the ventral part.

When the second part of duodenum undergoes axial rotation, the ventral pancreatic bud (carrying the primitive bile duct with it) winds round the posterior surface of the duodenum to appear on the postero-medial aspect, where it meets with the dorsal pancreatic bud. The fusion of the two parts takes place in the seventh week.

Later a cross communication is established between the duct systems of the dorsal and the ventral parts . As a result the main pancreatic duct (duct of Wirsung) is developed from three sources - (i) from the distal part of the dorsal duct; (ii) from oblique cross-connection between the ducts of the two buds; (iii) from the proximal part of the primitive bile duct. The accessory pancreatic duct (duct of Santorini) develops from the proximal part of the duct of the dorsal bud; this duct either regresses or persists in rudimentary form.

The smaller ductules and the acini of the pancreas are derived from the repeated sprouting of the ducts of the dorsal and ventral pancreatic buds. The islet cells of Langerhans are developed during the third month from the acinar cells, which are detached from the walls of the alveoli and form independent colonies of islets.

As long as the pancreas grows dorsally in the meso-duodenum it is invested by the peritoneum on both right and left surfaces. With the rotation of the duodenum, the pancreas rotates to the right so that the original right surface (covered with peritoneum) now becomes posterior. Later the peritoneum disappears from the posterior surface except the tail of the pancreas; hence, the pancreas becomes a retroperitoneal organ.

Anomalies of the pancreas **(Plate VI)**

(a) Annular pancreas

The original bilobed structure of the ventral pancreatic bud sometimes fails to fuse to form a single mass. In such condition the two lobes grow in opposite directions around the duodenum and meet the dorsal pancreatic bud. The annular pancreas, thus formed, may produce duodenal obstruction.

(b)Accessory pancreatic tissue

Heterotopic nodules of pancreatic tissue may be found in the walls of duodenum, ***Meckel's diverticulum*** or gall bladder. A duodenal diverticulum is sometimes associated with the accessory pancreatic tissue when the latter is attached to the duodenal wall.

Spleen

The spleen is developed from mesoderm within the dorsal mesogastrium, where it appears at first as a number of lobules of the splenic tissue. These lobules join together to form a single splenic mass which projects under cover of the left layer of the dorsal mesogastrium. The presence of the splenic notches affecting the upper border of the adult organ indicates the lobulated development of the spleen. The appearance of the spleen in the dorsal mesogastrium divides the latter into the gastro-splenic ligament in front, and the lieno-renal ligament behind.

Anomalies of the spleen

Accessory nodules of the splenic tissue are occasionally present around the main organ, within the gastro-splenic ligament and the greater omentum, and very rarely in the left spermatic cord.

The Mid Gut

The primitive mid gut extends as a straight lube from the anterior to the posterior intestinal portals, and is suspended in the median plane from the dorsal body wall by the primitive dorsal mesentery which conveys the blood vessels of the mid gut, the superior mesenteric vessels. Unlike the fore gut, the mid gut presents no ventral mesentery. Initially the mid gut communicates ventrally with the extra-embryonic part of the yolk sac by the ***vitello-intestinal duct.***

As the development progresses, the mid gut grows in length more rapidly than the capacity of the abdominal cavity, and forms eventually a U-shaped ventral loop known as the primary intestinal loop which is suspended in the mid-sagittal plane by the elongation of the dorsal mesentery. *The vitello-intestinal duct is attached to the apex of the intestinal loop*; the superior mesenteric artery runs forwards through the dorsal mesentery midway between the proximal and the distal limbs of the U-shaped loop and gives rise to a number of pre-arterial and post-arterial branches. Therefore, the intestinal loop presents u cephalic limb or pre-arterial segment and the caudal limb or post-arterial segment. From the cephalic limb develop the lower part of the duodenum distal to the entrance of the bile duct,

the jejunum and most of the ileum. The caudal limb of the midgut gives rise to the terminal part of the ileum, the caecum and appendix, the ascending colon and proximal two-thirds of the transverse colon.

During the sixth week a caecal diverticulum develops from the anti-mesenteric border of the caudal limb close to the apex of the intestinal loop, and at about the same period the vitello-intestinal duct regresses completely under normal conditions. The proximal part of the caecal diverticulum enlarges to form the caecum, and its distal part persists as a narrow tube of the vermiform appendix. The dorsal end of the cephalic limb, the future duodeno-jejunal flexure, is anchored to the body wall by a mesenchymal condensation of the dorsal mesentery which is known as the superior retention band; this band persists in adult as the suspensory muscle of the duodenum or the ligament of Treitz. Similarly the dorsal end of the caudal limb, colic angle, is attached to the body wall by an inferior retention band which forms the phrenico-colic ligament in adult. The base of the mid gut loop is somewhat narrow and is known as the duodeno-colic isthmus.

Meanwhile the abdominal part of the coelomic cavity is largely occupied by the enlargements of the growing liver and the mesonephric kidneys, and the space left in the coelom becomes temporarily too small for the

accommodation of the progressively elongated loop of the mid gut. Hence it becomes obligatory for the intestinal loop to herniate into the extra-embryonic part of the coelomic cavity which remains patent advantageously during this period in the proximal part of the umbilical cord as the umbilical sac. Thus the mid gut loop appears as a content of the physiological umbilical hernia during the fifth week and remains in this position up to the tenth week.

Subsequent development of the mid gut may be conveniently divided into three stages.

First stage

When the U-shaped loop lodges in the umbilical sac, the cephalic limb of the mid gut (small gut) is subjected to a downward pressure by the caudal edge of the right lobe of the liver. Eventually the cephalic limb rotates downwards and to the right, and as a consequence the caudal limb (large gut) moves upwards and to the left. During the first stage, the intestinal loop rotates through an angle of about 90° around the axis of the superior mesenteric artery, and when viewed from the front the rotation is anticlockwise.

Second stage

During a stay of about five weeks (from the 5th week to the 10th week) in the physiological umbilical hernia, the right limb (previously cephalic limb) or small gut segment of the mid gut loop undergoes enormous elongation and is arranged in a series of coils. Meanwhile the caecal bulging exhibits its prominence in the left limb.

Progressive decrease in the dimensions of the liver and the mesonephric kidneys and the concomitant expansion of the abdominal part of the coelomic cavity, allow the herniated intestinal loop to re-enter the abdomen from the umbilical coelom. But the umbilical hernia cannot be reduced en masse, because the intestinal loop becomes much coiled and bulky by this lime and the umbilical ring (communication between intra- and extra-embryonic coelom) is too small to allow easy return of the intestinal loop. Withdrawal of the herniated mass, therefore, takes place in a definite order. The right limb or small gut segment of the intestinal loop is reduced first, because the bulky caecum cannot return so easily through the narrow umbilical ring, The caecum behaves like a neutral observer and takes a greater responsibility of directing the withdrawal of the gut in a sequential manner. Consequently, the coils of the small intestine occupy the right part

of the abdominal cavity. But the condition so happens that the right half of the abdomen fails to accommodate the huge aggregation of the intestinal coils. As a result the duodenal (proximal) part of the mid gut passes dorsal to the origin of the superior mesenteric artery and pushes the dorsal mesentery of the hind gut and the colic angle to the left side. Thus the coils of small gut occupy the dorsal, right and left parts of the abdominal cavity. The left limb of the mid gut (including the caecum and the associated colon) returns last in the abdomen. The caecum and the mid gut colon appear initially in the left part of the abdomen, and find to their utter surprise that the dorsal part of the abdomen is flooded with the coils of small gut. The caecum and the associated colon undergo a frantic search for their fixation to the dorsal body wall; they rotate upwards and to the right superficial to the small gut around the axis of the superior mesenteric artery and pass successively through the left iliac, umbilical, and sub-hepatic regions where the caecum persists for some time on the under surface of the right lobe of the liver. The portion of the colon which extends between the colic angle (where inferior retention band is attached) and the sub-hepatic region persists in adult as the transverse colon. The rotation of the midgut explains why the transverse

colon lies ventral to the superior mesenteric artery, whereas the third part of the duodenum passes dorsal to that artery.

The second stage of rotation of the mid gut shows sequential reduction of the intestinal loop from the physiological umbilical hernia at the end of the tenth week, until the caecum reaches the sub-hepatic region.

Third stage

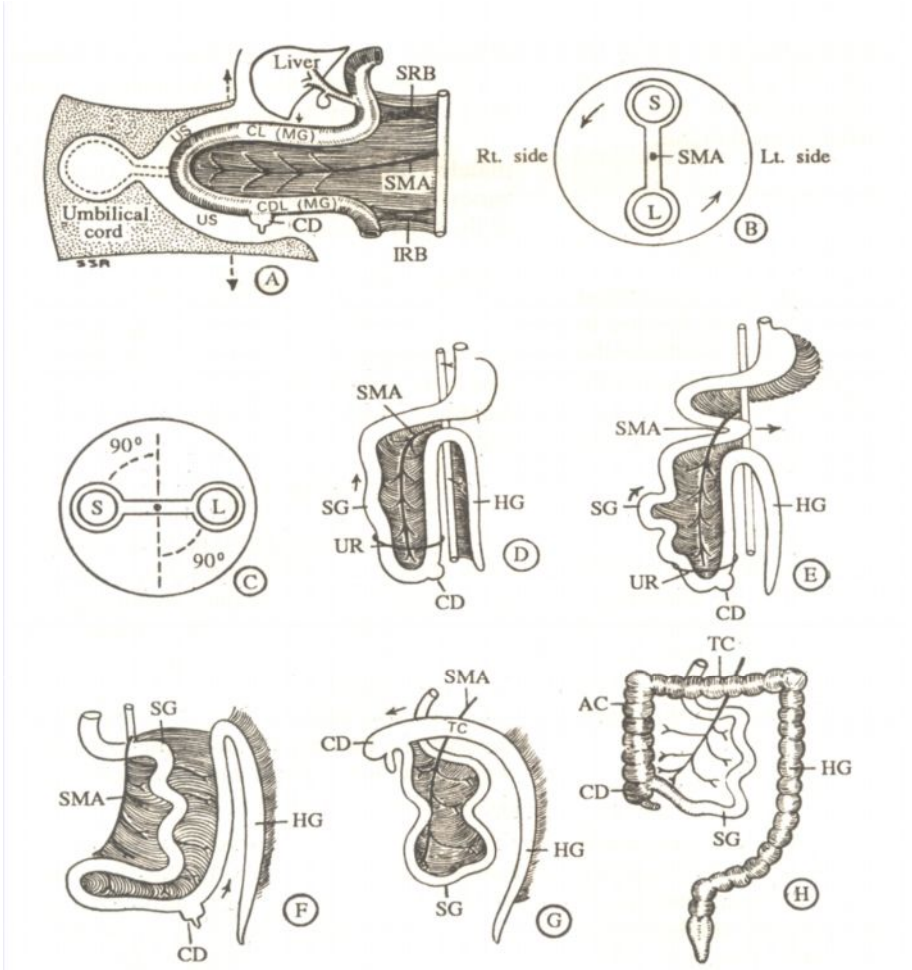
The caecum and the appendix grow caudally from the sub-hepatic region, pass through the right lumbar region, and finally reach the definitive position (normal) in the right iliac fossa. The portion of the colon thus formed, extending from the right iliac to the sub-hepatic regions, persists as the ascending colon.

The total range of rotation executed by the caecum and the appendix around the axis of the superior mesenteric artery is about 270° (three right angles). The first 90° of rotation takes place within the physiological umbilical hernia or even before that stage; the remaining 180° occurs within the abdomen after the intestinal loop is reduced from the umbilical coelom. When the rotation is complete, the derivatives of the midgut undergo a process of fixation. The dorsal mesentery of the ascending colon usually disappears and its posterior surface becomes non-peritoneal. Pre-arterial

segment of the dorsal mesentery of the midgut persists as the mesentery which suspends the jejunum and ileum from the posterior abdominal wall. Post-arterial segment of the dorsal mesentery persists in part to form the transverse mesocolon. The enlargement of the omental bursa (lesser sac) following the rotation of stomach allows a caudal extension of the infolded dorsal mesogastrium superficial to the transverse colon and its mesocolon. This infolded mesogastrium persists in adult as the greater omen-turn. Therefore, the third stage of development of the mid gut completes the rotation of the intestinal loop, and this is followed by the fixation or suspension of the different parts of the gut.

In order to reach the right iliac fossa (normal position,) the caecum and appendix undergo 270° rotation (three right angles) around the axis of the superior mesenteric artery. First90° rotation takes place within the physiological umbilical hernia; the remaining 180° occurs when the herniated, mass re-enters the abdomen. In the abdomen the caecum and appendix pass successively through the left iliac fossa, umbilical, sub-hepatic and right lumbar regions and reach the right iliac fossa. **(Plate VII)**

PLATE VII. ROTATION OF THE MIDGUT – DIFFERENT STAGES



C = Ascending colon CD = Caecal diverticulum CDL (MG)= Caudal limb (Mid gut)
CL (MG) = Cephalic limb (Mid gut) IRB = Inf. retention band SRB = Sup. retention band
G = Small gut SMA = Superior mesenteric artery TC = Transverse colon HG = Hind gut
UR = Umbilical ring US = Umbilical sac.

Fig. 12.2 1 - (A) Sagittal section of U-shaped mid gut in umbilical sac (before rotation)
(B) Coronal section of mid gut in umbilical sac (before rotation)
(C) Coronal section of mid gut loop (after initial 90° rotation)
(D) Small gut portion of U-shaped mid gut re-enters first from umbilical hernia
(E) Third part of duodenum grows to the left behind superior mesenteric artery
(F) Caecum enters the abdomen last and occupies the left side
(G) Caecum and Transverse colon rotate to the right in front of superior mesenteric artery
(H) Final position of the mid and hind guts

According to the shape, the adult caecum is classified under the following types :

(a) Foetal type (2%)

The caecum is conical and the appendix arises as a straight tube from its apex.

(b) Infantile type (3%)

The caecum assumes quadrate in form because two saccules of equal sizes develop on each side of the base of the appendix.

(c) Adult or normal type (90%)

The right saccule enlarges more than the left one, and the appendix is situated about 2 cm below the ileo-caecal junction.

(d) Exaggerated type (4% - 5%)

Here the right saccule enlarges and the left saccule atrophies. The appendix lies very close to the ileo-caecal junction.

Congenital Anomalies of the mid gut (Plate VIII)

Umbilical faecal fistula

When the vitello-intestinal duct remains completely patent, contents of the small gut arc discharged at the umbilicus.

Meckel's diverticulum (Plate IX)

Under normal conditions, the vitello intestinal duct disappears completely during the sixth week. In 2% human subjects its proximal part persists as the Meckel's diverticulum, which is attached to the anti-mesenteric border of the ileum about2 to 3 ft. proximal to the ileo-caecal junction. Usually the diverticulum is 2 inches long, and possesses the same calibre as that of the ileum. The tip of the diverticulum may be free or attached by a fibrous band to the umbilicus or to any other abdominal viscera.

The structural coat of the diverticulum is similar to that of the ileum. Occasionally, oxyntic cells secreting hydrochloric acid are found in the mucous membrane; the heterotopic pancreatic tissue may be present in its muscular coat.

Clinical manifestations:

- The vast majority of Meckel's diverticula are generally asymptomatic and are incidentally discovered on autopsy. The most common complication is generally bleeding, which occurs in 25-50% of patients who present with complications. The source of bleeding is a chronic acid induced ulcer in the ileum adjacent to a diverticulum that contains gastric mucosa.
- Another common complication is intestinal obstruction due to volvulus of the small bowel around a diverticulum associated with a fibrotic band attached to the anterior abdominal wall, or intussusception, or rarely, incarceration of the diverticulum in an inguinal hernia (Littre's hernia).
- Diverticulitis occurs in 10-20 % of symptomatic patients. This is more common in adult patients.

Diagnostic studies: **(Plate X)**

- Meckel's diverticula do not show up on routine X rays, ultrasound and CT scans. In children the single most useful investigation is scintigraphy with sodium ^{99m} Tc – pertechnate. This is preferentially taken up by the mucus secreting cells of the gastric mucosa and

ectopic gastric tissue of the diverticulum. The diagnostic sensitivity of this scan is 85% with a specificity of 95%. In adults, however, this sensitivity is much reduced. Sensitivity of this scan can be enhanced by the use of agents such as Cimetidine, Glucagon or Pentagastrin.

- In adults, when the scintigram is normal, a barium series must be performed.
- In patients with severe hemorrhage, angiogram may be useful.

Treatment:

The treatment for a symptomatic Meckel's should be prompt resection of the diverticulum or a segment of the ileum bearing the diverticulum. Resection can be performed either by stapling or sewing across the base of the diverticulum in a diagonal or transverse manner.

However, for an asymptomatic case, in children, during laparotomy, it is best to resect. In an adult some controversy exists. After a number of studies, it was concluded that is best to resect an incidental Meckel's upto the age of 80 years, so long as no additional conditions make the procedure hazardous.

Enterocystoma

Sometimes the vitello-intestinal duct is closed at both ends, but remains patent in the middle. The cyst thus formed lies beneath the umbilicus.

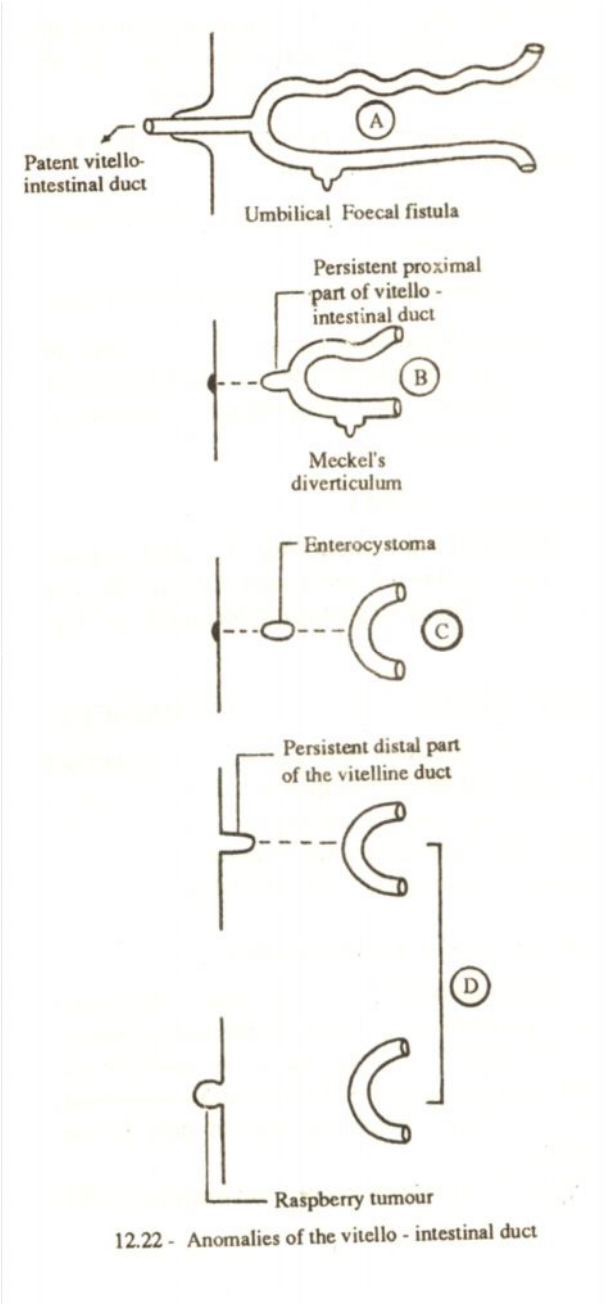
Raspberry tumour at the umbilicus

On rare occasion distal part of the vitelline duct persists, and the rest disappears. This distal part is evaginated from the umbilicus due to the increased intra-abdominal pressure, producing a raspberry red tumour.

Exomphalos (omphalocele)

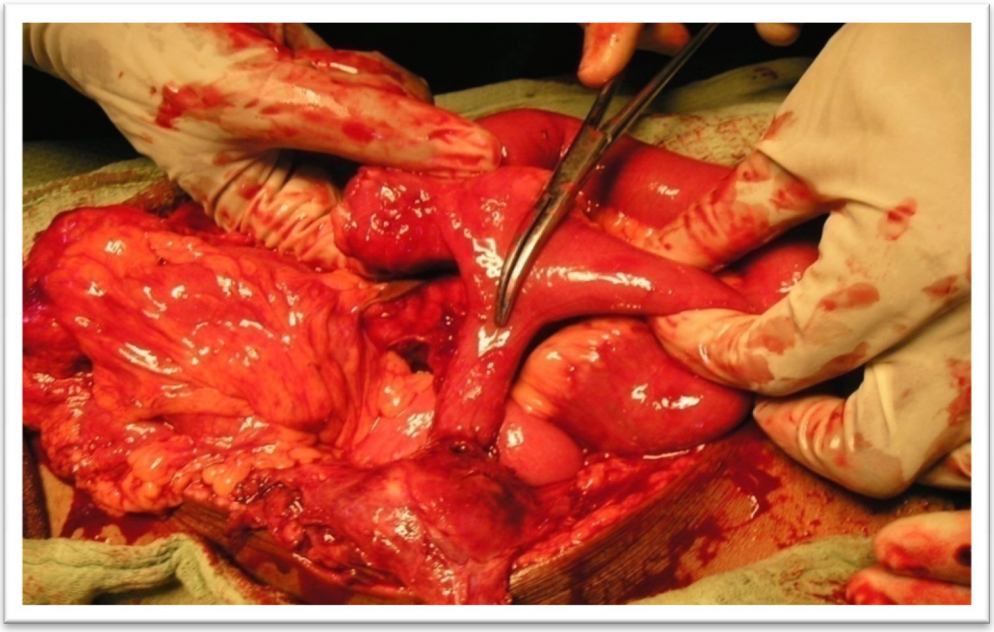
When the primitive mid gut loop fails to enter the abdominal cavity from the physiological umbilical hernia, a bulky mass is found in the umbilical cord at birth. This condition is known as exomphalos which is covered only by the amnion.

PLATE VIII. ANOMALIES OF THE MIDGUT



12.22 - Anomalies of the vitello - intestinal duct

PLATE VIII. AN INTRA – OP PHOTO OF A FULLY PATENT VITELLO-INTESTINAL DUCT



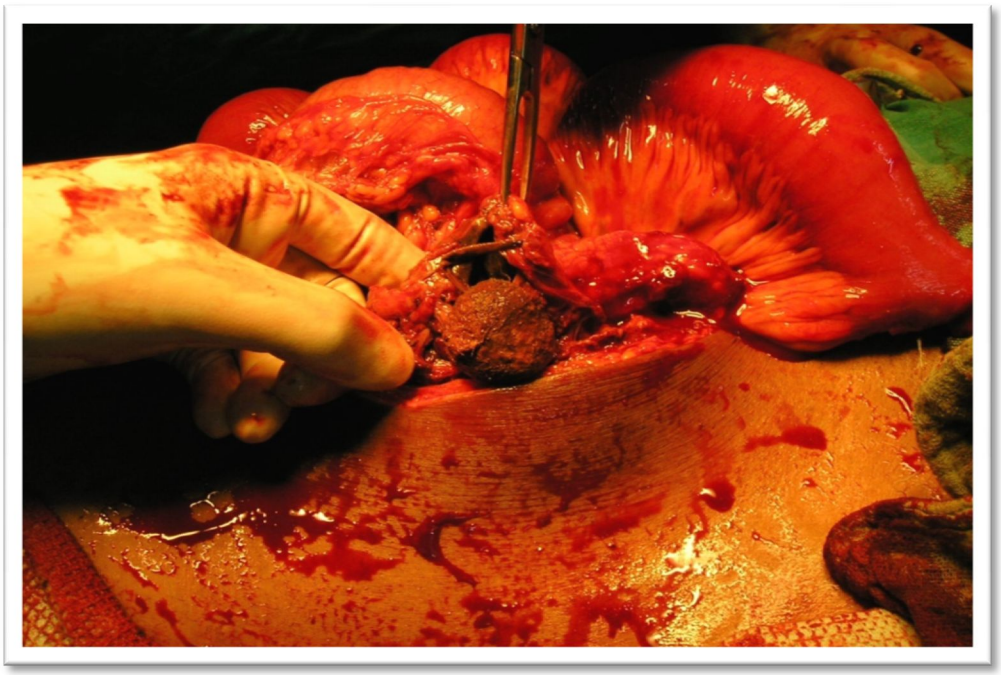


PLATE VIII. VITELLO INTESTINAL DUCT SHOWING A FECOLITH IN SITU

PLATE IX. INTRA OP PHOTO OF MECKEL'S DIVERTICULUM

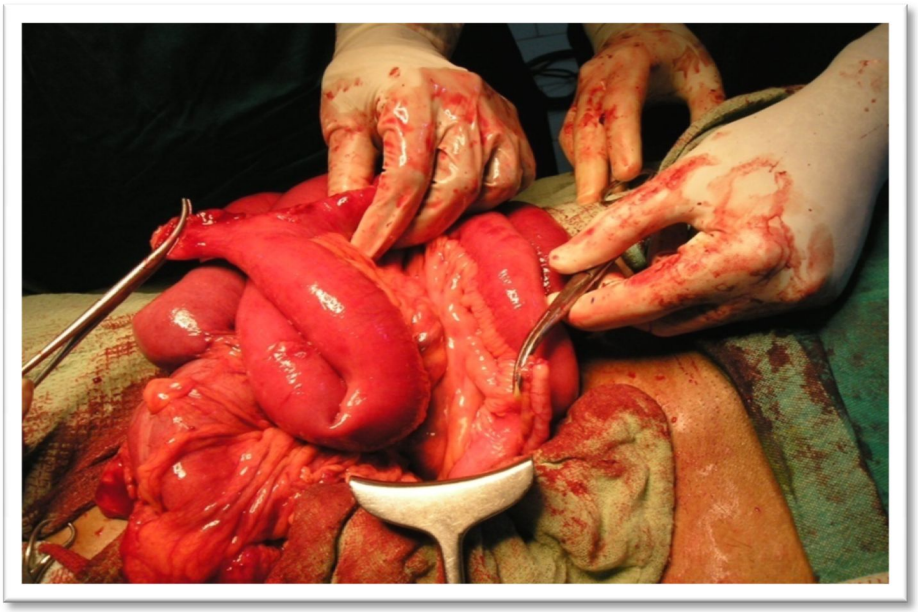


PLATE IX. MECKEL'S DIVERTICULUM BEING RESECTED

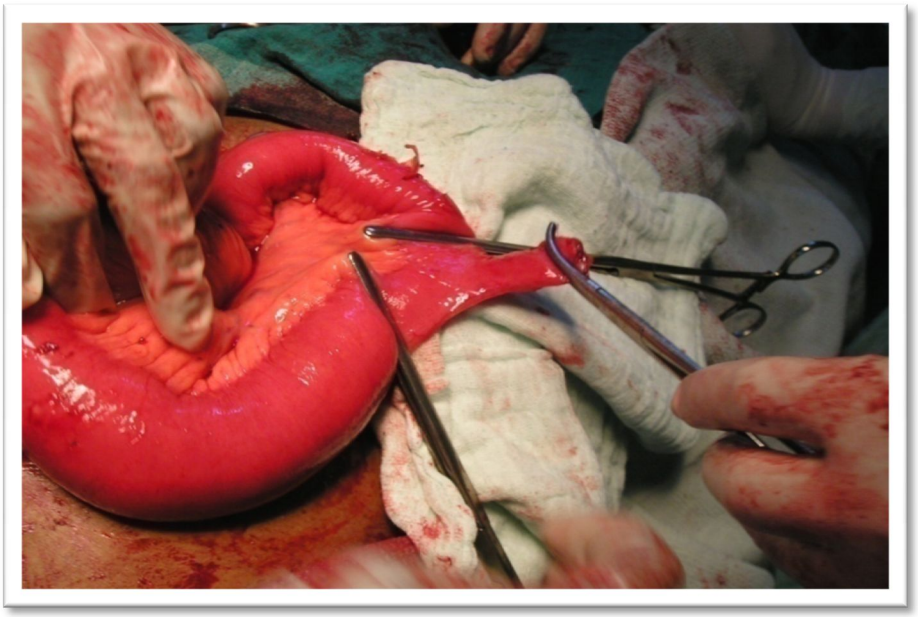


PLATE X. TECHNETIUM SCINTIGRAM SHOWING MECKEL'S DIVERTICULUM

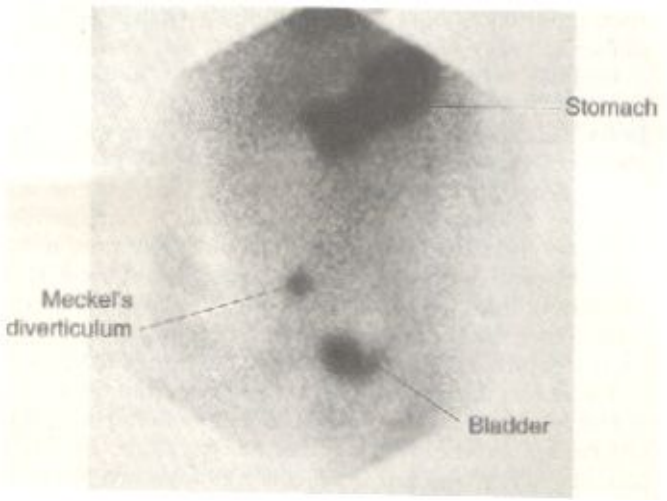
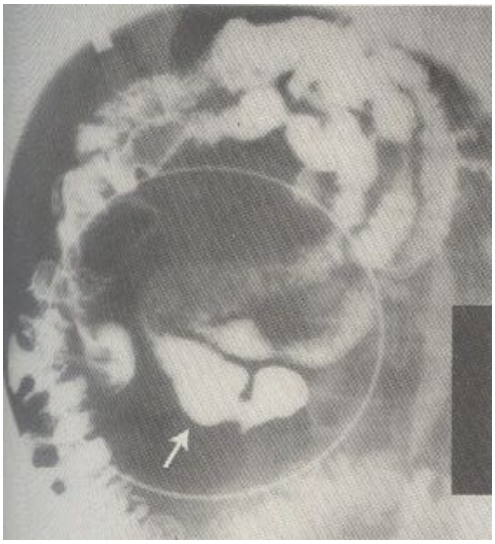


PLATE X. BARIUM FOLLOW THROUGH IN AN ASYMPTOMATIC MECKEL'S



Errors of rotation of the mid gut (Plate XI)

During the second stage

(a) Non-rotation of the mid gut :

Sometimes the umbilical ring remains dilated, and permits the reduction of the umbilical hernia en masse after initial 90" rotation. As a result the small gut occupies the right side of the abdomen, and the caecum with the associated colon is accommodated on the left side.

(b) Mal-rotation (reverse rotation) of the mid gut:

In this anomaly the caecum enters the abdomen first through the dilated umbilical ring and rotates upwards and to the right behind the origin of the superior mesenteric artery. Eventually the transverse colon extends behind the trunk of the superior mesenteric artery, and the third part of the duodenum lies in front of that vessel.

During the third stage

(c) ***Due to defects of fixation***

The caecum and the appendix may occupy the sub-hepatic, right lumbar, or the pelvic region.

PLATE XI. A CASE OF MID GUT MALROTATION

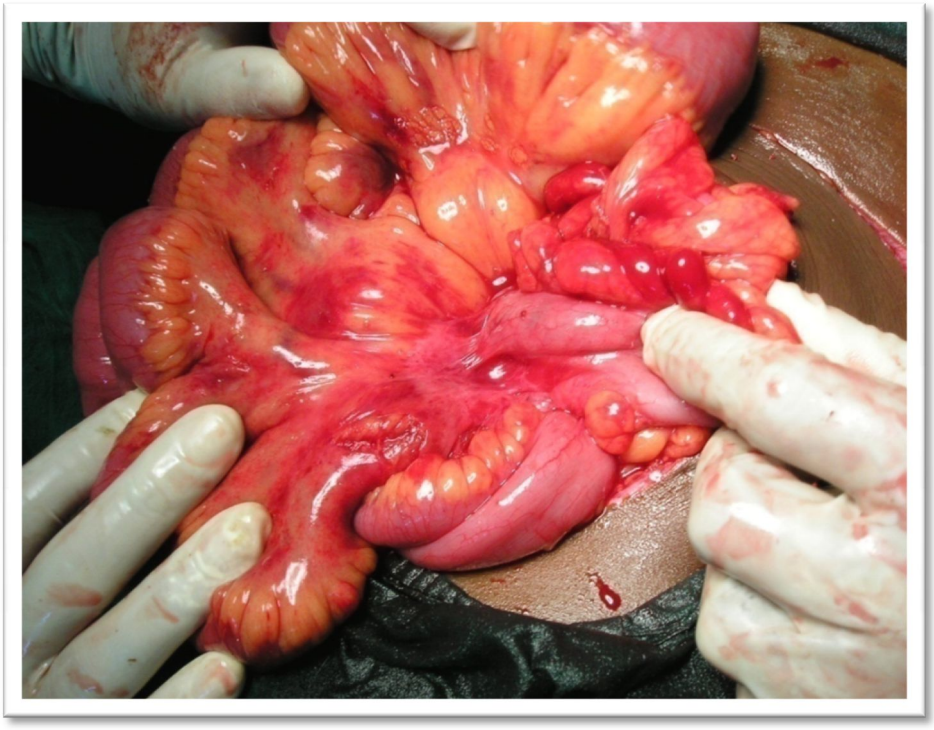
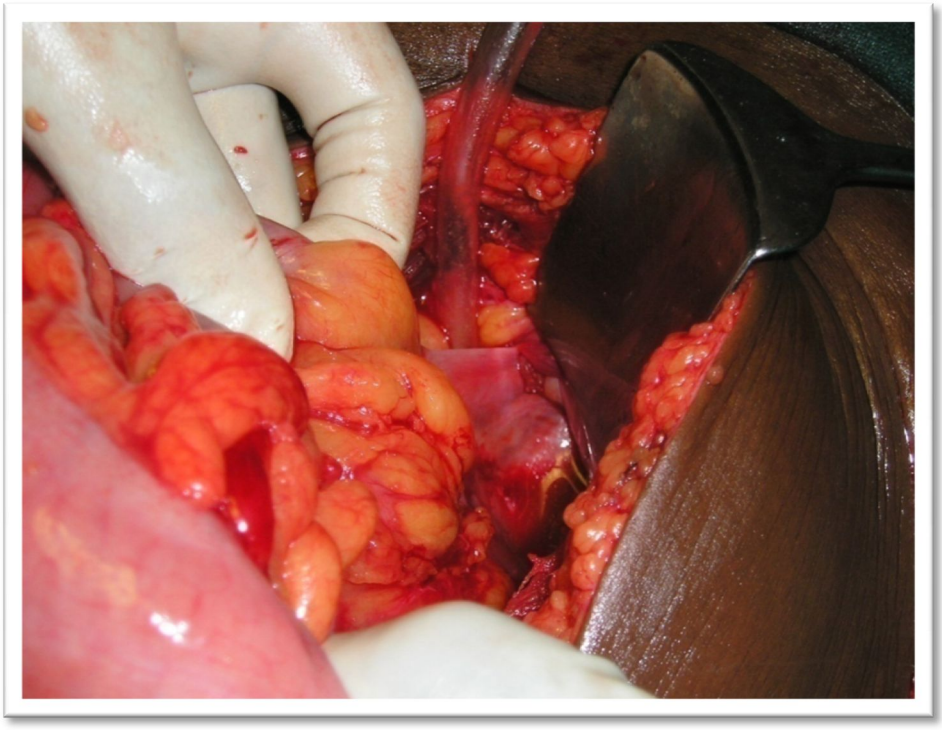


PLATE XI. MALROTATION DUE TO CONGENITAL BANDS



The Hind Gut

The hind gut is contained within the tail-fold of the embryo, and extends from the posterior intestinal portal to the cloacal membrane. Initially it is placed in the median plane and is suspended from the dorsal body wall by the primitive dorsal mesentery which conveys the inferior mesenteric vessels of the hind gut.

Caudally, the hind gut extends somewhat into the rudimentary tail as the post-anal gut or tail gut; subsequently the tail gut regresses. Ventrally, the hind gut is drawn out as a diverticulum, the allantois, which extends up to the foetal end of the umbilical cord. The allantois divides the hind gut into pre-allantoic and post-allantoic parts. The pre-allantoic part is narrow and tubular. The post-allantoic part dilates to form the endodermal cloaca. The ventral wall of the cloaca is initially formed by a bilaminar cloacal membrane, where the surface ectoderm and the endoderm come in close contact without the intervention of the mesoderm. At first the cloacal membrane is elongated and limited on each side by a raised margin, the genital fold. The mesodermal cells from the primitive streak migrate ventrally and head wards along the genital folds and appear within the genital tubercle (rudimentary phallus). These cells gradually accumulate in

the interval between the umbilical cord and the cloacal membrane, and form the infra-umbilical part of the anterior abdominal wall. Eventually the cloacal membrane shifts caudal wards and becomes shortened. It now lies at the bottom of the cloaca and forms a surface depression, the eclodermal cloaca. Subsequently, when the caudal edge of the urorectal septum fuses transversely with the cloacal membrane, the ectodermal cloaca is divided into an anal pit or proctodeum in the posterior part and a primary urethral groove in the anterior part. **(Plate XII A)**

Pre-Allantoic Part of the Hind Gut

It develops into the distal third of the transverse colon, the descending and the sigmoid colon. The proximal part of the hind gut is pushed to the left side during the rotation of the mid gut, and the inferior retention band which is attached to the colic angle (future left colic flexure) now forms the phrenico-colic ligament. The dorsal mesentery usually disappears in the descending colon, but persists in the sigmoid colon as the sigmoid mesocolon.

Post-allantoic part or endodermal cloaca **(Plate XII B)**

The endodermal cloaca gives rise to the development of the rectum, upper part of the anal canal, and most of the mucous lining of the urinary bladder and the urethra.

Development of Rectum and Anal canal (Plate XIII A)

The endodermal cloaca is divided by the urorectal septum into a ventral part, the primitive urogenital sinus and a dorsal part, the primitive rectum. The urorectal septum is a coronally oriented partition which consists of three elements -a vertical and two lateral. The vertical element also known as Toumeux fold grows caudally from the angle between the allantois and the hindgut. The lateral elements known as the folds of Rathke project inwards from each lateral wall of the cloaca. Each Rathke's fold contains a mesonephric (Wolffian) and a paramesonephric (Mullerian) duct, the former is placed lateral to the latter. These three elements fuse to form a composite urorectal septum which extends caudally towards the cloacal membrane. At first, a gap known as the cloacal duct appears between the caudal free edge of the urorectal septum and the cloacal membrane. Through this duct the primitive rectum and the urogenital sinus communicate temporarily with

each other. At about the seventh week, the cloacal duct is closed and the urorectal septum fuses with the cloacal membrane, dividing the latter into the urogenital membrane in front and the anal membrane behind. The transverse line of fusion between the septum and the cloacal membrane forms the future perineal body.

The ectodermal cloaca behind the perineal body presents a surface depression, the proctodeum or the anal pit. The backward growth of the perineal body separates the anal membrane further from the urogenital membrane. The underlying mesenchymal cells proliferate around the proctodeum, making the latter more deep. Finally the anal membrane ruptures and the rectum communicates with the exterior through the anal canal. The position of the anal membrane is represented in adult by the pectinate line of the anal canal (anal valves); some authors consider that Hilton's line of the anal canal represents the muco-cutaneous junction. Therefore, the anal canal is developed from two sources: upper part, from the endodermal cloaca, and the lower part, from the ectodermal proctodeum.

Congenital Anomalies of the Hind Gut (Plate XIII B)

Hirschsprung's disease (congenital mega colon)

It is a condition in which a segment of large gut (usually somewhat proximal to the anal canal) shows persistent narrowing since birth, due to the congenital agenesis of the nerve cells in the myenteric and sub mucous plexuses. The narrowed segment of the gut is devoid of parasympathetic innervation and fails to respond to waves of peristalsis. Eventually, the over-active sympathetic nerves induce a persistent spasm to the affected part of the gut. The gut proximal to the site of obstruction becomes necessarily distended for no fault of its own.

This anomaly may be treated surgically by resection of the constricted part of the gut, and subsequent mobilisation of the proximal dilated gut to the anal region.

Undivided cloaca

On rare occasions, the urorectal septum fails to develop completely, and the undivided cloaca opens to the exterior through a common perineal orifice after the rupture of the cloacal membrane. Both the genito-urinary and alimentary passages have a common perineal outlet. This is a normal

phenomenon in birds, but becomes an anomaly in man showing the signs of atavism.

Rectal fistula

(a) Recto-vesical fistula (high fistula)

When the three elements of the urorectal septum (a vertical and two lateral) fail to approximate, the cloacal duct persists at a higher level and the rectum communicates with the urinary bladder usually in the region of the internal trigone.

(b) Recto-urethral or recto- vaginal fistula (low fistula)

In this anomaly the vertical element of the urorectal septum grows normally, but the lateral folds (folds of Rathke) fail to grow inwards. As a result the cloacal duct persists in the lower part. In male, the rectum communicates with the urethra usually at the level of the colliculus seminalis of the prostatic urethra (recto urethral fistula). In female, the presence of the Mullerian ducts complicates the picture; the rectum communicates with the upper part of the vagina.

In both high and low fistulae the anal pits may be rudimentary or absent.

(c)Imperforate anus

This condition may be the primary defect, if secondary to the rectal fistulae. When primary, the imperforate anus may be caused by the following:

(a) Failure of the rupture of the anal membrane;

(b) Non-development of the ectodermal proctodeum ;

(c) Atresia of the lower part of the rectum.

(***d)Ectopic anus -***

Under normal conditions the perineal body grows backwards and separates the anal membrane from the urogenital membrane. When the backward growth of the perineal body is arrested, the anal and urogenital orifices lie close to each other. In female, the anal and vaginal orifices may be situated within the vestibule (cleft between the labia minora). In male, the anal orifice may appear at the base of the scrotum, in the intra-bulbar fossa of the spongy urethra, or at the coronal sulcus of the glans penis.

PLATE XII A. THE PRE ALLANTOIC HIND GUT

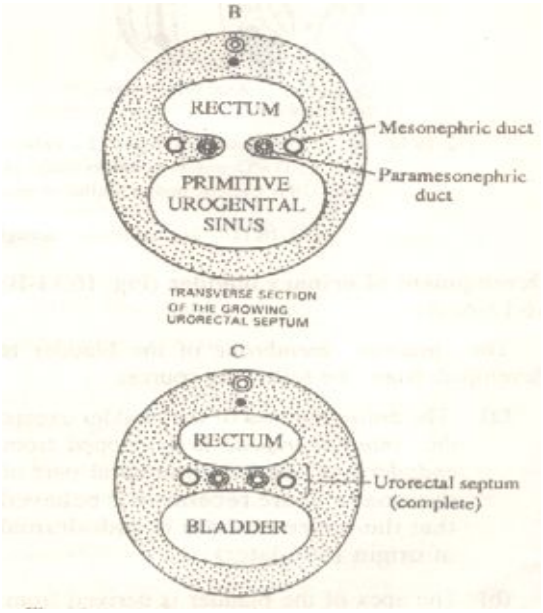


PLATE XII B. THE CLOACA

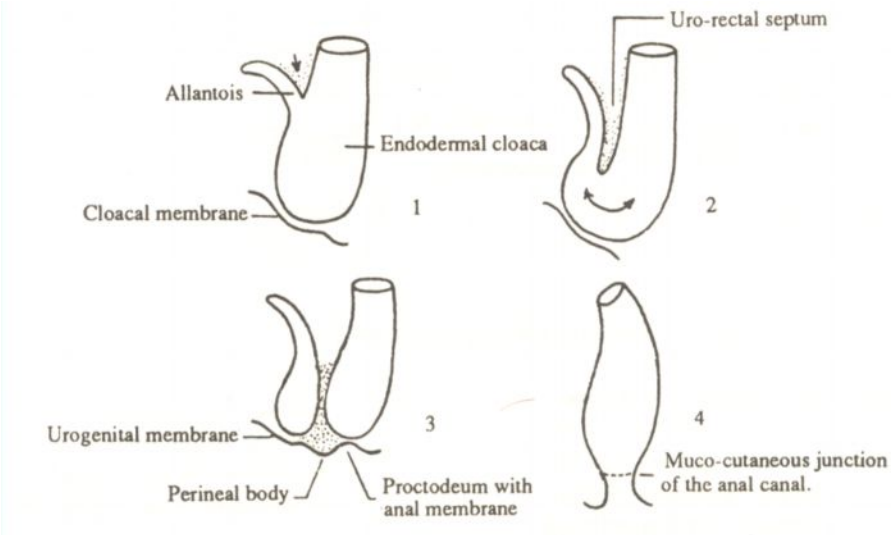


PLATE XIII A. THE RECTUM AND ANAL CANAL

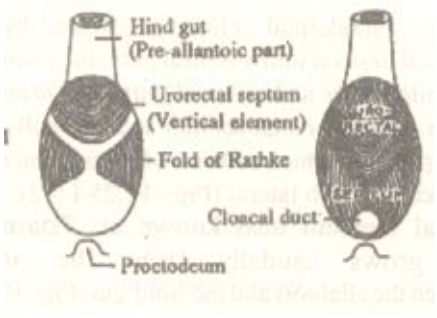
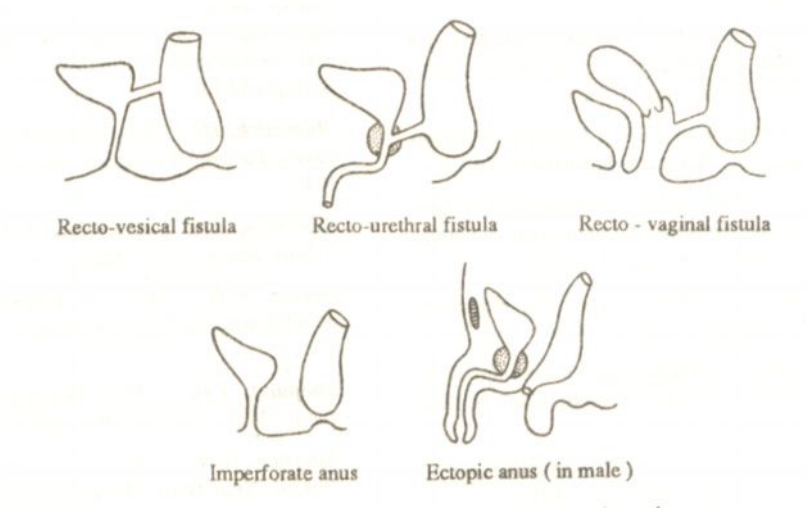


PLATE XIII B. HIND GUT ANOMALIES



Development of urinary bladder **(Plate XIV A)**

The mucous membrane of the bladder is developed from the following sources:

(a) *The entire mucosa of the bladder except the internal trigone* is developed from endoderm of the vesico-urethral part of the cloaca. More recently it is believed that the entire mucosa is endodermal in origin.

(b) *The apex of the bladder* is derived from absorption of the proximal part of the *allantoic diverticulum*. The distal part of the diverticulum is obliterated and the ***fibrosed wall persists as the urachus which extends from the apex of the bladder to the umbilicus. The fibrous cord of urachus is derived from the splanchnic mesoderm around the allantoic canal.***

(c) *The internal trigone of the bladder* is developed from mesoderm by the incorporation of *caudal parts of the mesonephric ducts*.

The mesonephric ducts open into the pelvic part of the urogenital sinus, and the common excretory ducts (the part of mesonephric duct between the ureteric bud and the cloaca) are blended with the dorsal wall of vesico-urethral part of the cloaca. Eventually the ureteric buds open directly into the bladder. Thereafter, the ureteric openings migrate head wards and laterally, probably due to the growth of the bladder wall; this increases the surface

area of the internal trigone. The mesodermal contribution of the trigone is not only confined to the bladder, but extends along the dorsal wall of the proximal part of the urethra. Currently it is thought that the distal portion of the mesonephric ducts and attached ureteric ducts become incorporated into the dorsal wall of the vesico-urethral part by a process of exstrophy or eversion. The exstrophy begins as the mouths of the common excretory ducts flare into a pair of expanded and flattened trumpet-like structures and blend into the bladder wall. The upper portion of this trumpet expands more rapidly than the lower part, so that the mouth of the narrow portion of the mesonephric duct appears to migrate interiorly along the posterior bladder wall. This process incorporates the commencement of the ureteric buds into the upper part of the bladder wall, and causes the mouths of the narrow part of the common excretory ducts to migrate inferiorly until they open in the pelvic part of urogenital sinus below the bladder neck as the ejaculatory ducts. Thus the triangular area of exstrophied wall of the caudal part of mesonephric ducts forms the trigone of the bladder. Subsequently, the mesodermal tissue of the trigone is overgrown by the endoderm from the surrounding bladder wall, and the mesodermal components of the mesonephric ducts persist as the trigonal muscle of ureters. This accounts for

the smooth and glistening mucosa of the internal trigone.

The musculature and other structures of the bladder are developed from the splanchnic layer of lateral plate mesoderm which surrounds the cloaca.

Developmental Anomalies

(1) Congenital recto-vesical fistula

This is due to incomplete development of the urorectal septum and the persistence of the cloacal duct.

(2) Congenital vesico-vaginal fistula

In this condition the Mullerian eminence projects into the vesico-urethral part of the cloaca, instead of urogenital sinus, and disintegrates thereafter, establishing communication between the bladder and the vagina.

(3) Urachal cyst

Sometimes the allantoic diverticulum is obliterated at the proximal and distal parts, but remains patent in the middle. This produces a cyst which may be palpable as a mid-line elevation in the infra-umbilical part of the anterior abdominal wall.

(4) Urachal fistula

The allantois may remain patent entirely and urine appears through the umbilicus. This is usually associated with congenital stenosis of the urethra. A more common variant of this is the ***Urachal sinus,*** where the sinus tract connects the umbilicus to the apex of the urinary bladder, and the tract is closed in entirety.

(Plate XIV B)

(5) Ectopia vesicae

Anterior wall of the bladder is deficient and the interior of the bladder is exposed to the infra-umbilical part of the anterior abdominal wall.

PLATE XIV A. THE DEVELOPMENT OF THE URINARY BLADDER

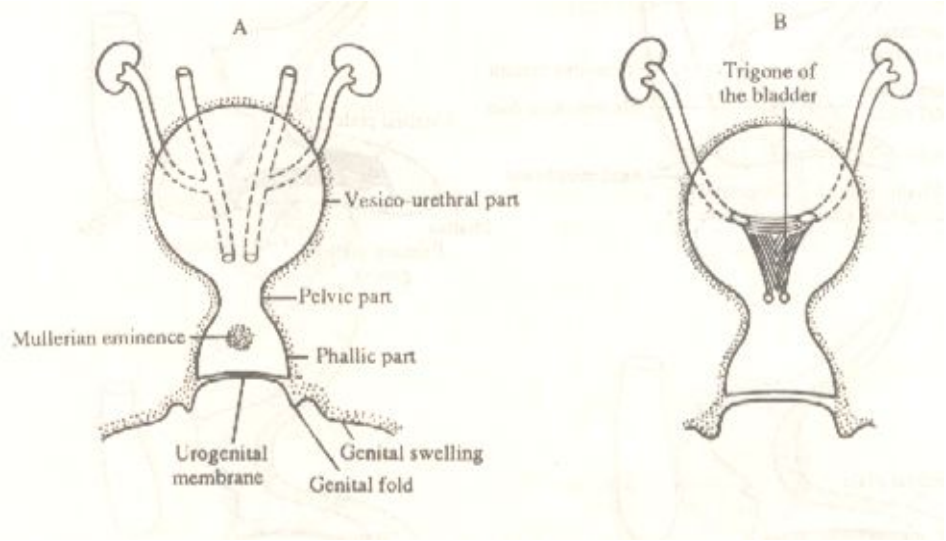


PLATE XIV B. AN INTRA OP PHOTO OF A URACHAL SINUS

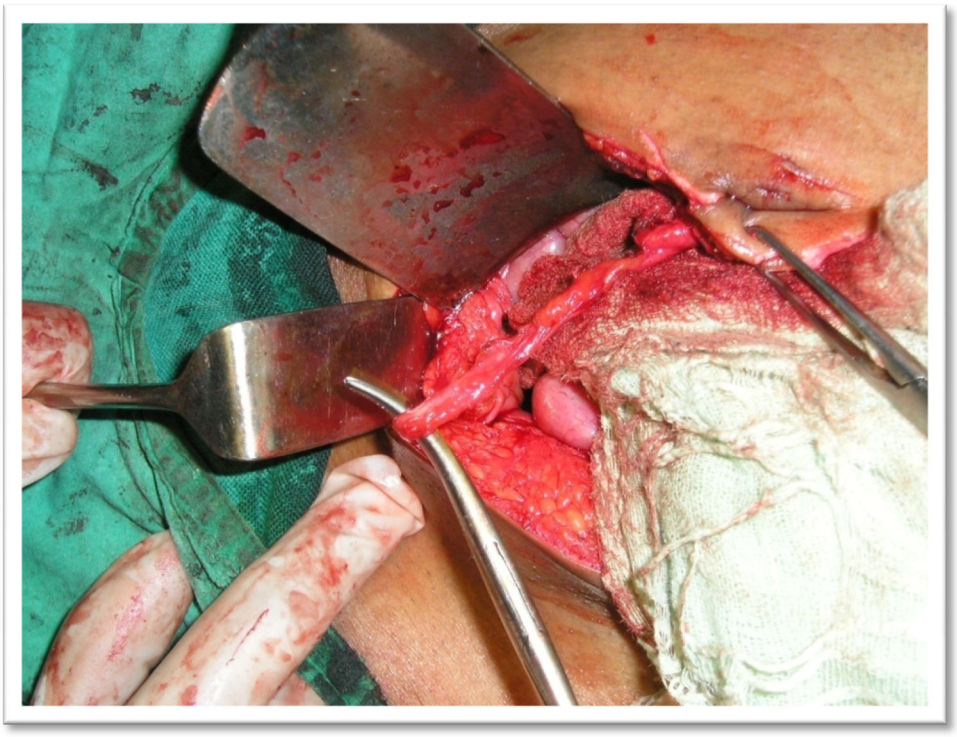


PLATE XV. A PLAIN X RAY OF INTESTINAL OBSTRUCTION DUE TO CONGENITAL BANDS

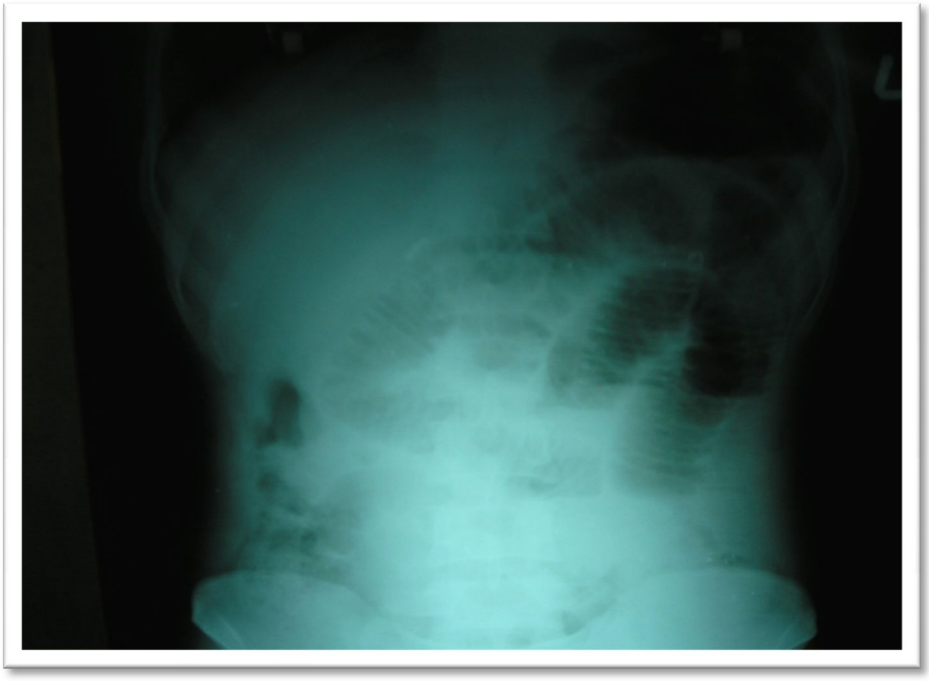
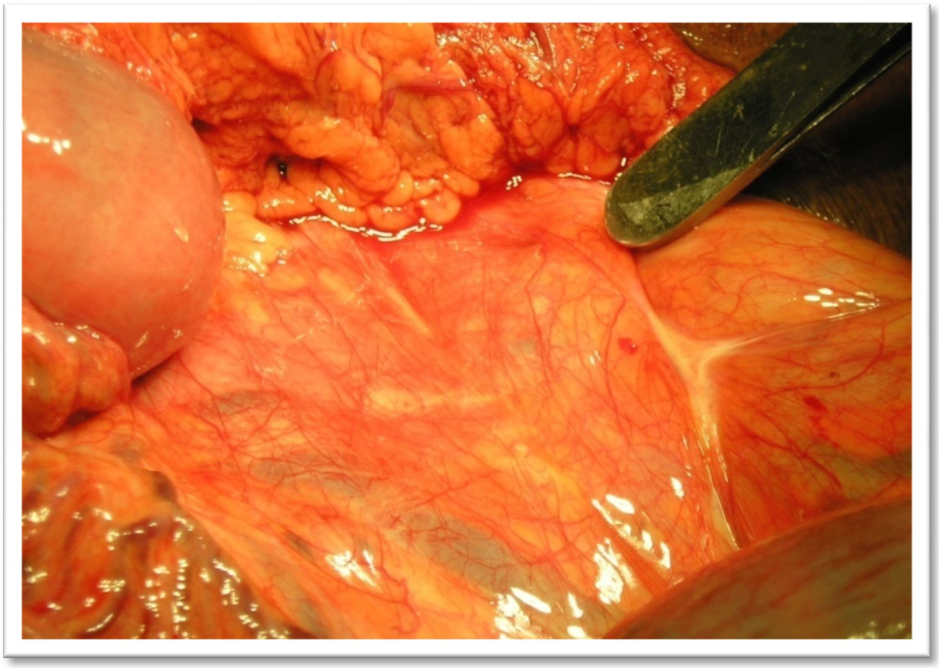


PLATE XV. INTRA OP PHOTO OF A MIDGUT BAND CAUSING INTESTINAL OBSTRUCTION



AIMS OF THE PRESENT STUDY

This study is a retrospective analysis of gut and genito-urinary anomalies encountered in abdominal emergency surgery from the period of July 2006 to October 2008 in a single institution.

The study aims to identify the following:

- To identify the most common embryological anomalies in adult emergency surgery
- To assess whether there is a difference between distribution of such anomalies between male and female patients
- To assess the most common modes of manifestation in such embryological gut/genitourinary anomalies

STUDY DETAILS

Type of study: Retrospective study

Number of cases: 28

Male: Female : 18 male: 10 female

Period of study: June 2006 to October 2008

Institution: Department of General Surgery, Government Royapettah Hospital, attached to Kilpauk Medical College

Type of analysis: Clinical data analysis

Inclusion criteria:

- Adult patients (above the age of 12 years)
- Abdominal emergency surgery (surgery for appendicectomy, and laparotomies included)

Exclusion criteria:

- Pediatric cases (below the age of 12 years)
- Elective surgeries and patients who were previously worked up suspecting such anomalies

Limitations:

- Being a study of embryological anomalies in emergency surgery, this study gives a general idea of the incidence of only those cases who present with complications, either resulting due to these anomalies or due to some other cause. Most patients in the general population do not present with too many problems, and the actual incidence of these anomalies is probably much higher. The true incidence in all these cases can be verified only by autopsy.
- Due to a limited number of cases, statistical analysis could not be done.

All patient details were meticulously recorded and details verified with the case sheets. All variables pertaining to patient details, presentation, pre-op investigations were recorded in preformed worksheet to ensure uniformity in recording and eliminating any bias.

Patient details and procedure done, with the anomaly detected are mentioned in the master chart.

MANAGEMENT PROTOCOL

1. Clinical evaluation
2. Investigations
    a. X ray chest PA (erect)
    b. X ray abdomen PA (supine)
    c. Ultrasound abdomen
    d. CT abdomen – plain and contrast
3. Surgical management
4. Complications during the immediate post – op period
5. Follow up in the late post op period and re-admissions if any
6. In case of asymptomatic patients where the anomaly was left alone, whether the patient suffered problems/ was operated later for a complication of the same anomaly

MODES OF PRESENTATION

In this study, it was seen that patients with documented gut embryological anomalies manifested with one of the following:

- Peritonitis – localized / generalized
- Intestinal obstruction
- Intra-abdominal/parietal wall abscesses
- Asymptomatic – discovered in the course of a laparotomy for some other reason

SURGICAL OPTIONS

Surgical options exercised varied according to each case. In cases where a Meckel's diverticulum was complicated or resected for some reason, a wedge resection was done. In cases where a congenital band was the cause of obstruction, the band was released. In one particular case where malrotation was a cause for obstruction, Ladd's procedure was proceeded with. Finally, in a documented case of Hirschsprung's disease in an adult male, a temporary sigmoid colostomy was done followed by an endorectal pull through 6 months later. However, in many cases where the anomaly was

asymptomatic and not the cause of the current problem, masterly inactivity was exercised.

POST OP PERIOD

Post op morbidity and mortality in the immediate 14 days were analyzed.

LONG TERM FOLLOW UP

All the patients were followed up for hospitalizations and/or problems related to the previous anomaly, if left un-operated in the first instance.

ANALYSIS OF THE STUDY

- A total number of 28 cases were taken up for analysis within the stipulated study period
- The number of male patients was 18 and the number of female patients was 10; the male – female ratio being 1.8: 1
- The total number of abdominal emergency surgeries in the study period was 921 (287 laparotomies + 624 appendicectomies)
- The age of the patients ranged from 14 years to 73 years.
- The mean age of manifestation was 30.8 years
- The median age of the selected study group was 27 years.
- The distribution of anomalies was as under:

	FOREGUT ANOMALIES	MIDGUT ANOMALIES	HINDGUT ANOMALIES	GENITO – URINARY ANOMALIES
MALE	**0**	**12**	**4**	**2**
FEMALE	**0**	**8**	**2**	**0**
TOTAL	**0**	***20***	***6***	***2***

- The various modes of presentation of such anomalies are as follows:

	PERITONITIS LOCALISED/DIFFUSE	INTESTINAL OBSTRUCTION	PARIETAL WALL ABSCESSES	ASYMPTOMATIC/INCIDENTAL FINDING
MALE	**3**	**6**	**3**	**6**
FEMALE	**1**	**4**	**0**	**5**
TOTAL	**4**	**10**	**3**	**11**

- The commonest anomalies are described in the table below:

	MECKEL'S DIVERTICULUM	ANOMALOUS BANDS	MALROTATION	URACHAL ANOMALIES	OTHERS
MALE	**6**	**6**	**1**	**2**	**3**
FEMALE	**4**	**5**	**1**	**0**	**0**
TOTAL	**10**	**11**	**2**	**2**	**3**

- The incidence of complicated Meckel's diverticulum is as under:

	SYMPTOMATIC	ASYMPTOMATIC/INCIDENTAL
MALE	**2**	**4**
FEMALE	**1**	**3**
TOTAL	**3**	**7**

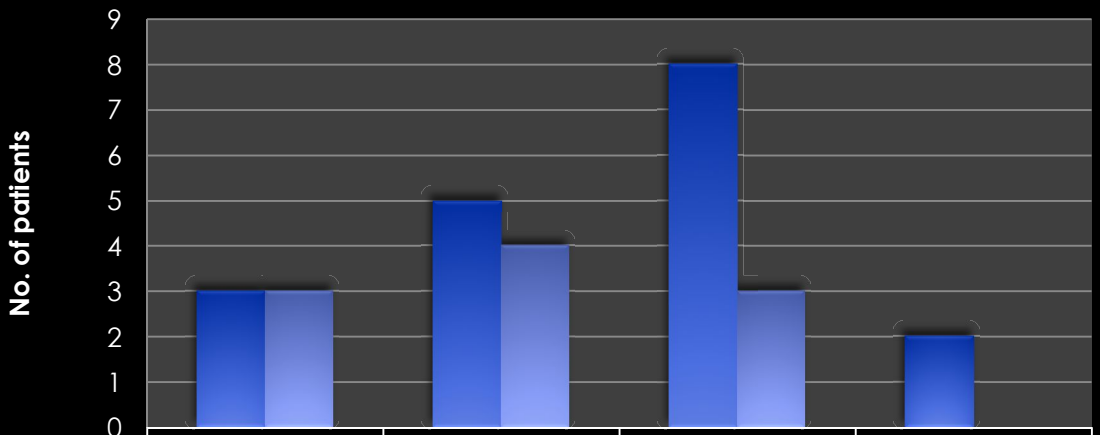
- A description of anomalous bands and their correlation to intestinal obstruction is described in the table:

	SYMPTOMATIC GUT BANDS	INCIDENTAL/ASYMPTOMATIC GUT BANDS
MALE	**4**	**2**
FEMALE	**3**	**2**
TOTAL	**7**	**4**

Based on the above data, the following were calculated:

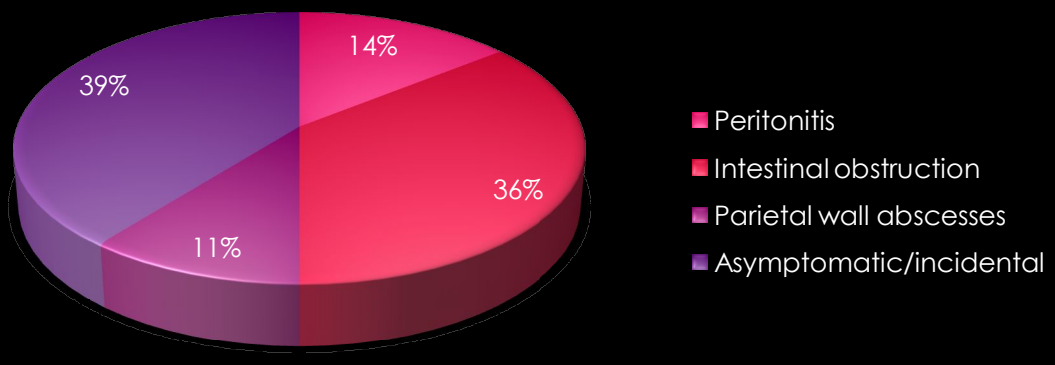
PARAMETER	OVERALL VALUE	VALUE IN MALE	VALUE IN FEMALE
INCIDENCE OF ANOMALIES	**3.04%**	**1.95%**	**1.08%**
INCIDENCE OF FOREGUT ANOMALIES	**0%**	**0%**	**0%**
INCIDENCE OF MIDGUT ANOMALIES	**2.17%**	**1.30%**	**0.86%**
INCIDENCE OF HINDGUT ANOMALIES	**0.65%**	**0.43%**	**0.21%**
INCIDENCE OF GENITO-URINARY ANOMALIES	**0.21%**	**0.21%**	**0%**
INCIDENCE OF MECKEL'S DIVERTICULUM	**1.08%**	**0.65%**	**0.43%**
PERCENTAGE OF COMPLICATED MECKEL'S	**30%**		
PERCENTAGE OF COMPLICATED GUT BANDS	**70%**		

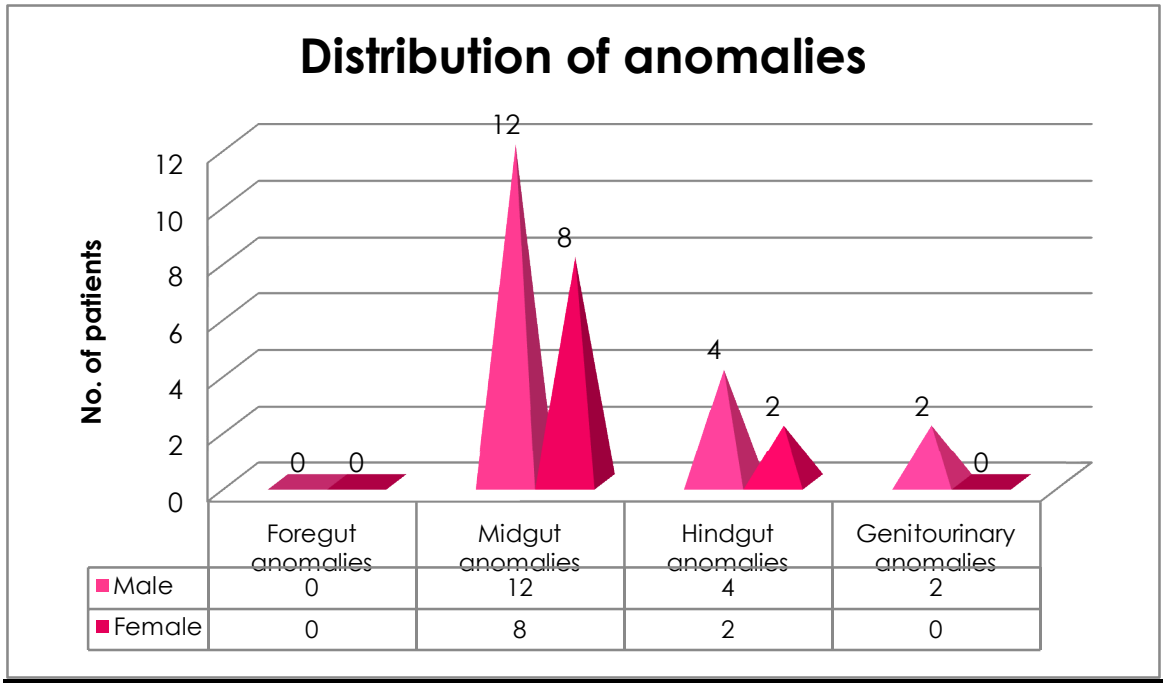
Agewise distribution of anomalies

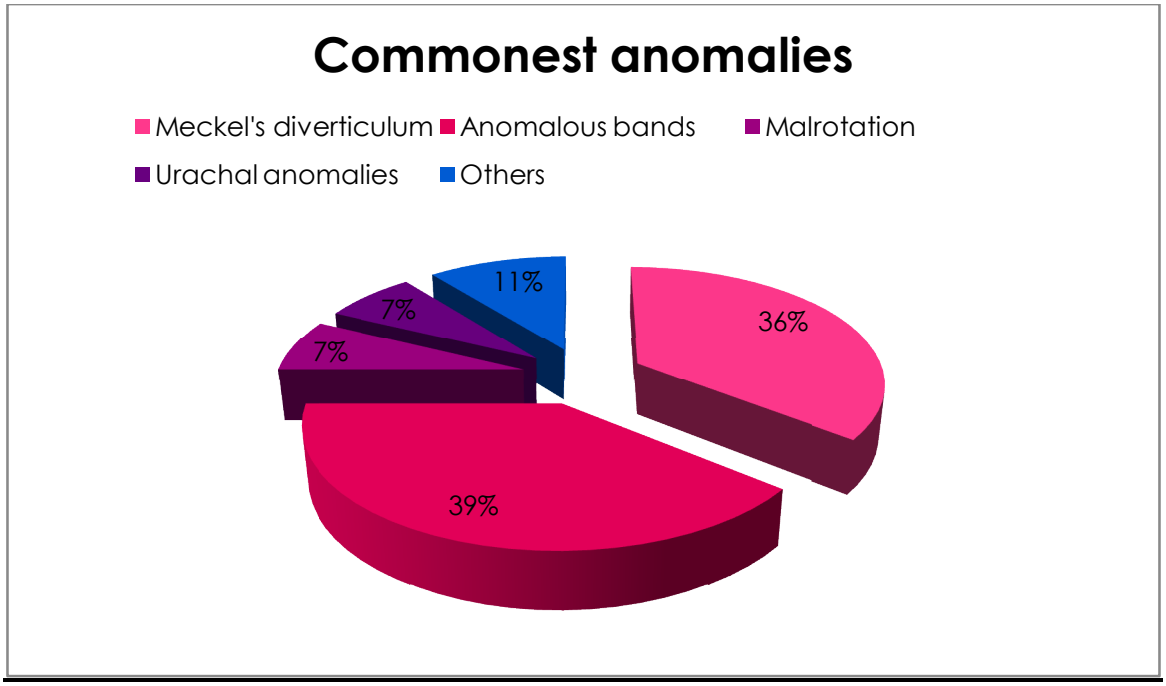


	12-20 years	21-30 years	31-40 years	> 40 years
Male	3	5	8	2
Female	3	4	3	0

Modes of presentation




Distribution of anomalies
No. of patients
12
10
8
6
4
2
0
12
8
4
2
2
0
0
0
Foregut anomalies
Midgut anomalies
Hindgut anomalies
Genitourinary anomalies
Male 0 12 4 2
Female 0 8 2 0


Commonest anomalies
Meckel's diverticulum
Anomalous bands
Malrotation
Urachal anomalies
Others
11%
7%
7%
36%
39%

SUMMARY OF FINDINGS

From the data mentioned earlier, the salient findings are made out:

- The incidence of gut and allied anomalies in this study was found to be around 3%.
- The number of males who presented with such anomalies out numbered females in a significant manner in the ratio 1.8:1. This suggests a strong tendency for such defects to be present in males. Similar studies done elsewhere have not shown any definite male predisposition, except in certain cases like Congenital Hypertrophic pyloric stenosis
- It was noted that most of these anomalies are asymptomatic, discovered during a laparotomy for some other reason. However, the incidence of intestinal obstruction in this series was quite high, being the next most common mode of manifestation.
- Among the anomalies, Meckel's diverticulum (36%) and anomalous peritoneal bands (39 %) were the most common, and with nearly equal incidence in the study.

- Most Meckel's diverticula were asymptomatic (70 %), while in the case of gut bands, the trend was reversed, with the possibility of a gut band being symptomatic being as high as 70%.
- The incidence of Meckel's diverticulum was found to be 1%, lower than the usual incidence of 2%. However, the rate mentioned in earlier series took into account the findings at autopsy. This being a study of only surgical patients, and specifically emergency surgeries, could possibly contribute to this lower rate encountered.
- The incidence of midgut anomalies was the highest. We encountered no foregut anomalies. This may be due to the fact that a foregut anomaly is likely to present earlier, and possibly rectified earlier.
- Similar studies do not exist for a satisfactory comparison. Other studies do not individually document incidence rates, except for the more common of these anomalies. Most of such cases are found in pediatric age group.

CONCLUSION

Though the number of cases encountered by the general surgeon is not very many, these gut anomalies pose an interesting problem in management. The situation is very simple when these embryological defects get complicated. But the dilemma lies in the management of asymptomatic cases – to resect or not to resect? Several studies conclude that when there are no further co-morbid factors influencing the outcome, it is better to remove these anomalies in lieu of anticipated problems. We also see that an anomalous band is more likely to cause a serious, life threatening problem than diverticula/duplications and the like. Another fact that stares at us is that the more distal the gut band, the more likely that it will stay asymptomatic and undiscovered for a longer period of time. In this series, most of the anomalies were dealt with at source. Those patients in whose case such defects were left alone were monitored. In the period of follow up, patients did not get readmitted or face further problems due to these.

A final lesson learnt from this study is that every patient is a textbook and that it is impossible to rule out congenital anomalies in any patient, merely by his/her age!

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PATIENT NAME: AGE: SEX:

INPATIENT NUMBER:

ADDRESS:

DATE OF ADMISSION:

BLOOD GROUP:

PRESENTING COMPLAINTS:

h/o Abdominal pain
h/o Abdominal distension
h/o Obstipation
h/o Fever
h/o Umbilical discharge

H/O PRESENT ILLNESS – Duration of symptoms and if there were any such problems earlier

PAST HISTORY:

PERSONAL HISTORY: If patient is a woman, any menstrual irregularities, h/o recurrent urinary infections

CLINICAL EXAMINATION

GENERAL EXAMINATION: Pallor/cyanosis/icterus/clubbing/fever/generalized edema/dyspnea/tachypnea

VITALS: Pulse
BP
Respiratory rate

SYSTEMIC EXAMINATION
CVS
RS

EXAMINATION OF THE ABDOMEN

- Whether all quadrants move with respiration
- Any visible mass
- Any visible gastric/intestinal peristalsis
- Abdominal distension
- Umbilicus – discharge if any
- Guarding/rigidity
- Free fluid/organomegaly

- Bowel sounds
- Abdominal girth
- Per – rectal examination – findings
- Hernial orifices
- Genito-urinary system

WORKING DIAGNOSIS

INVESTIGATIONS
- Complete blood count
- Blood grouping – cross matching
- Renal function tests
- X-ray chest PA (erect)
- X-ray abdomen PA
- Ultra sound abdomen
- CT abdomen (plain and contrast)

PRE OP INSTRUCTIONS:
- Nil per oral
- IV fluids
- Antibiotics
- Pulse charts
- Abdominal girth charts

INTRA OP:
- Operating surgeon
- Anaesthetist
- Anaesthesia given
- Any intra op problems encountered
- Surgery done
- Intra op finding
- If any incidental finding, whether the anomaly was corrected or not

POST OP:
- Complications if any

S.NO	NAME	AGE	SEX	IP NO	ANOMALY	PRESENTATION	LOCATION	SYMPTOMATIC
1	RAJENDRAN	37	M	876086	PATENT VID	PWA	MG	Y
2	RAMESH	35	M	877344	MALROTATION	I O	MG	Y
3	BOOPATHY	40	M	882554	LADD'S BAND	IO	HG	Y
4	RAMESH	27	M	884250	LADD'S BAND	INC.	HG	N
5	MOORTHY	32	M	885619	MIDGUT BAND	IO	MG	Y
6	SHANKAR	17	M	877102	HIRSCHSPRUNG'S	IO	HG	Y
7	GANESAN	18	M	870095	LANE'S ILEAL BAND	INC.	MG	N
8	HUSSAIN	26	M	868943	MECKEL'S	PERFORATION	MG	Y
9	BENJAMIN	15	M	835782	MECKEL'S	INC.	MG	N
10	MUTHULINGAM	73	M	836750	JEJ.DIVERTICULA	PERFORATION	MG	Y
11	STEVEN	37	M	840148	MECKEL'S	INC.	MG	N
12	CHINNAYYAN	35	M	848512	MECKEL'S	INC.	MG	N
13	JAYARAMAN	66	M	849188	LADD'S BAND	IO	HG	Y
14	PANDIARAJ	24	M	909606	MECKEL'S	PERFORATION	MG	Y
15	NARENDRAN	38	M	873966	MIDGUT BAND	IO	MG	Y
16	DANIEL THOMAS	27	M	882857	URACHAL SINUS	PWA	GU	Y
17	SATHYANARAYANAN	32	M	904660	MECKEL'S	INC.	MG	N
18	BHAGYARAJ	27	M	903204	URACHAL SINUS	PWA	GU	Y
19	MALA	35	F	891382	ILEAL INTUSSUSCEPTION DUE TO MIDGUT BAND	IO	MG	Y
20	GOVINDAMMA	23	F	884310	MIDGUT BAND	INC.	MG	N
21	CHITRA	21	F	830874	MECKEL'S	INC.	MG	N
22	GEETHA	19	F	833908	MECKEL'S	INC.	MG	N
23	DEENA	14	F	845400	MECKEL'S	PERFORATION	MG	Y
24	GAJALAKSHMI	27	F	849453	MIDGUT BAND	IO	MG	Y
25	VANMATHY	17	F	857402	MECKEL'S	INC.	MG	N
26	GEETHA	23	F	892157	MALROTATION	IO	MG	Y
27	THULASI	40	F	884617	LADD'S BAND	IO	HG	Y
28	BHAVANI	39	F	910264	LADD'S BAND	INC.	HG	N

***KEY: *IO* – INTESTINAL OBSTRUCTION; *INC* – INCIDENTAL; *PWA* – PARIETAL WALL ABSCESS; *FG* – FOREGUT; *MG* – MIDGUT; *HG* – HINDGUT; *GU*-GENITOURINARY**