DIVERTICULA OF THE GASTRO-INTESTINAL TRACT

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Terminology

The term 'diverticulum' comes from the Latin root 'diverto - I turn aside'. The '-culum' is a diminutive ending equivalent to the English '-icle' as in 'follicle' and 'cubicle'. English equivalent to the Latin 'diverticulum' is thus: 'diverticle' and means 'a small turning aside' - i.e., a pouch of limited size, as opposed to a diffuse pouching.

Classification

Choice lies between two classifications: a Pathological and an Anatomical one.

A. Pathological Classification

1. True Congenital Diverticula
   - (a) Meckelian
   - (b) Non-Meckelian

2. Acquired Diverticula
   - (a) Primary hernial protrusions of the mucous and submucous coats through a gap in the muscular coat.
   - (b) Secondary
     - i. Associated with diseases of the neighboring intestinal wall.
     - ii. Traction Diverticula
     - iii. Pseudo-Diverticula

B. Anatomical Classification: This is according to their level in the alimentary tract.

1. True Congenital Diverticula - same as under Pathological Classification.

2. Acquired Diverticula
   (a) Pharynx
   (b) Oesophagus
   (c) Stomach
   (d) Duodenum
   (e) Jejunum and ileum
   (f) Colon
   (g) Vermiform Appendix

The Pathological Classification is more to be desired, as there is a conformation to similar types, sharing a similar pathogenesis, irrespective of the portion of bowel in which they are found. In building up this classification, one is hampered by incomplete knowledge of the pathogenesis.

An Anatomical Classification is of supreme importance to the clinician as the influence of the diverticula upon the health of the individual is very largely dependent upon their situation within the alimentary tract.

The Anatomical basis will be followed in this thesis.

Review of cases of diverticula admitted to Winnipeg General Hospital from 1936 to 1945 inclusive, was made:-

Year	Total for the Year
1936	8
1937	10
1938	12
1939	16
1940	13
1941	14
1942	9
1943	7
1944	15
1945	15

CONGENITAL DIVERTICULA

Meckel's Diverticula

J. F. Meckel in 1822 was the first one to establish the etiology of the diverticulum which bears his name.

Occurrence

Meckel's diverticula constitute the vast majority of congenital cases. It occurs in approximately two percent of subjects.

Embryology

A Meckel's diverticulum is formed by a persistence of the intra-abdominal part of the vitello-intestinal duct, which normally becomes occluded and shrivels up during the seventh week of intra-uterine life.

The yolk sac of man contains no appreciable amount of actual yolk substance; because of its reduced state, it is frequently called the umbilical vesicle. From the nutritive standpoint, the human yolk sac is vestigial, yet is important in as much as its ento-dermal roof provides the epithelium for almost the entire digestive system. Also, blood cells and blood vessels arise early in the mesoderm of the sac proper and institute a vitelline circulation with the embryo.

In the youngest specimen known, the entoderm has not yet organized into a yolk sac (figure 1). Stages next older show a tiny vesicle lined with a simple layer of entoderm and surrounded by mesodermal cells.

Figure 1

When all the extra embryonic mesoderm is split by the coelom, it is the inner or splanchnic layer that covers the entodermal sac.

In embryos three weeks old, the roof of this vesicle consisting of taller entodermal cells, begins to fashion the fore-and-hind gut which are then connected by a slightly narrowed region to the yolk sac proper (figure 2). With the further growth of the embryo-body, there is progressive constriction of the embryo from the yolk sac (figure 3). This actual constriction is intensified when both embryo and yolk sac continue to enlarge, whereas their region of union

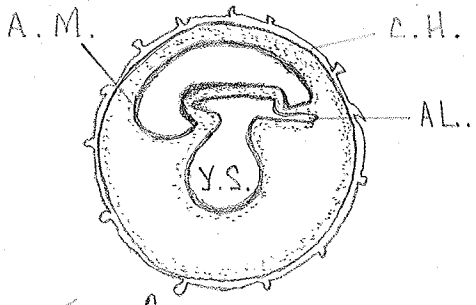

Fig. 2

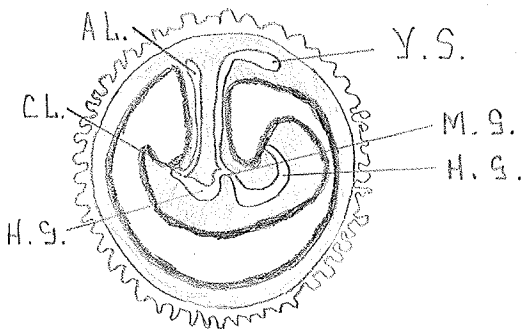

Fig. 3

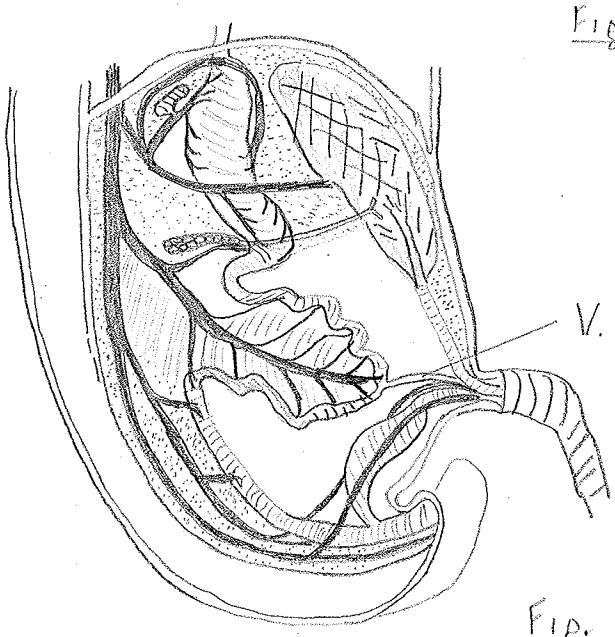

Fig. 4

Figures 2, 3 and 4:

These illustrate the formation of the vitello-intestinal duct.

In Figure 2, there is a wide communication between the yolk sac and the mid-gut, so that it is really one large cavity. The allantois is present at this stage.

In Figure 3, the vitello-intestinal duct has formed between the mid-gut and the yolk sac.

AL., indicates allantois;

AM., indicates Amnion;

CH., indicates chorion;

CL., indicates cloaca;

F.G., indicates fore-gut;

H.G., indicates hind-gut;

M.G., indicates mid-gut;

Y.S., indicates yolk sac.

Figure 4 illustrates the relationship of the vitello-intestinal duct to the intestinal tract and to the umbilicus. Meckel's diverticulum is formed by the persistence of this duct or parts of it.

lags. The slenderer connection does, however, elongate greatly to become the thread-like yolk stalk or vitello-intestinal duct which soon is incorporated into the umbilical cord (figure 4). The entodermal yolk stalk detaches from the gut by the end of the fifth week and soon degenerates.

The human yolk sac is a pear-shaped vesicle which attains an average size of about five millimeters by the middle of the second month. It subsequently shrinks somewhat and converts into a solid structure containing detritus. The sac usually persists throughout pregnancy and is frequently found in the after-birth between amnion and placenta.

A variation in the normal obliteration of the vitello-intestinal duct produces anomalies called Meckel's diverticulum of the ileum. This arises some two feet proximal to the ileo-colic valve and may be of a number of forms:

1. The most common form of the diverticulum is that of a conical, finger-like projection, opening at or near the free border of the ileum, the length varying one to six inches. Occasionally, a small secondary pouch opens from the main diverticulum (figure 5). Therefore, there may be diverticulosis of Meckel's diverticulum.

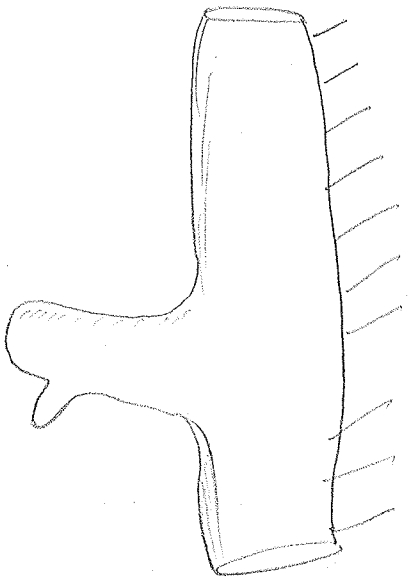

Fig. 5
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Meckel's Diverticulum with secondary pouch.

2. The duct may be completely obliterated with a cord remaining:

(a) Attached to the umbilicus;

(b) Hanging free in the abdominal cavity;

(c) Attached to some point within the abdominal cavity.

3. The duct may contain a cyst:

(a) At its central protion;

(b) In its distal portion with a sinus opening in the umbilicus.

4. The diverticulum may have its own mesenteriolum.

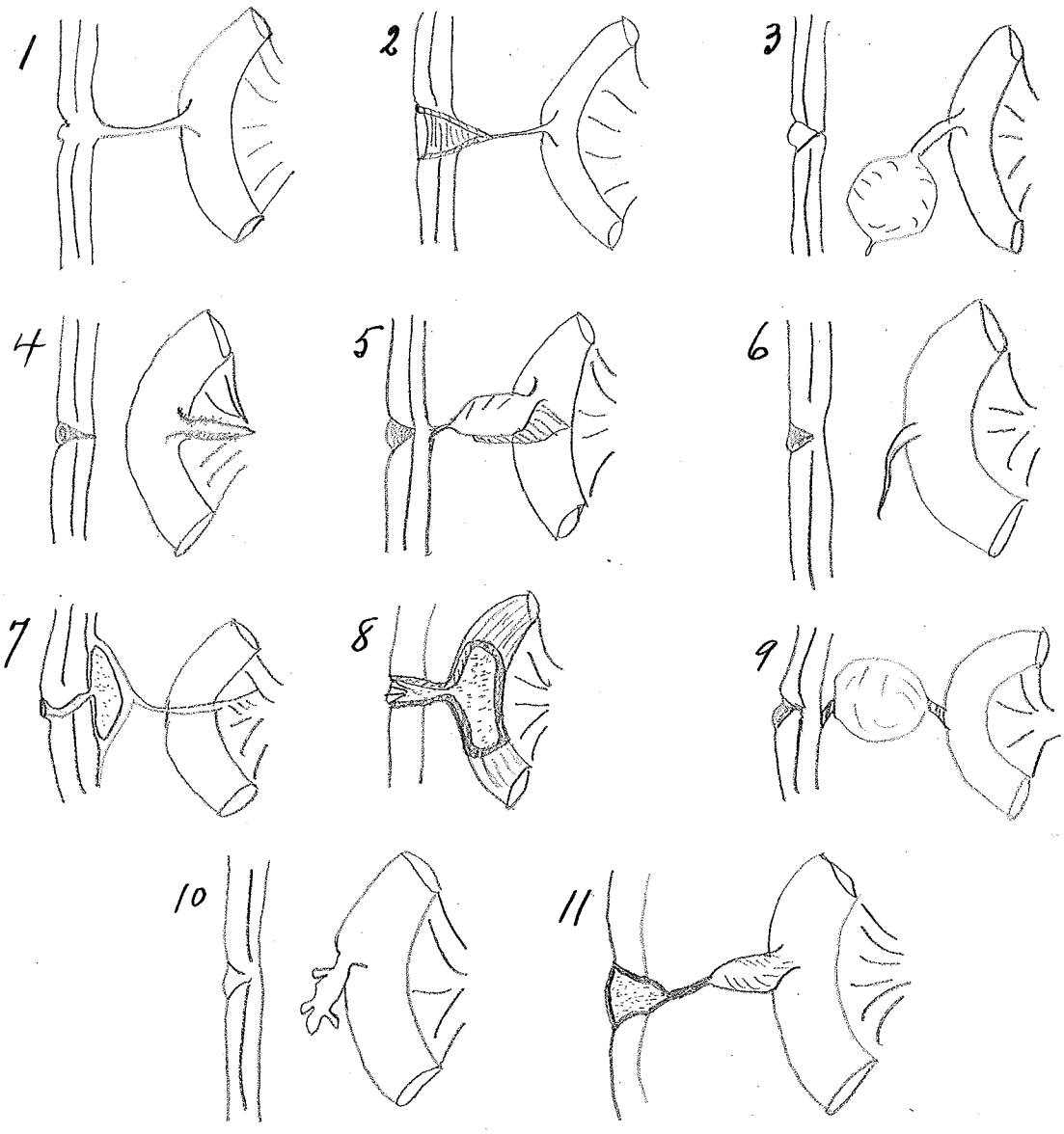
5. Still more rarely, it opens at the navel as a completely pervious duct through which intestinal contents escape; this condition constitutes a faecal umbilical fistula.

6. The peripheral remnant of the vitello-intestinal duct may show at the umbilicus itself as a moist rosette of membrane.

7. It may be as a closed cyst within the abdomen connected by a fibrous cord to either umbilicus, ileum or both.

Frequently, there is a narrowing of the gut lumen in the region of the diverticulum, congenital in origin or due to inflammation or to kinking.

Why and what causes such anomalies, one can only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DIAGRAMS ILLUSTRATING SOME OF THE VARIOUS ANATOMIC TYPES OF MECKEL'S DIVERTICULUM

1. Fibrous cord attached to the umbilicus from the ileum;
2. Persistent mucosa at the umbilicus attached to the ileum by a fibrous cord;
3. Cust hanging from the ileum by a twisted fibrous cord;
4. Blind tubular sac beneath the serosa of the mesentery of the ileum;
5. Blind tubular diverticulum attached to the umbilicus by a fibrous cord, having its own mesenteriolum;
6. Fibrous cord, remnant of the vitello-intestinal duct, hanging free from the ileum;
7. Cyst draining through the umbilicus, attached to the ileum by a fibrous cord;
8. Persistent patent vitello-intestinal duct, lined with mucosa, draining to the umbilicus from the ileum;
9. Cyst attached distally to the umbilicus and proximally to the ileum by a twisted fibrous cord;
10. Saccular diverticulum of the ileum showing diverticulosis of the diverticulum;
11. Saccular diverticulum of the ileum attached by a fibrous cord to a saccular area of mucosa in the umbilicus.

guess at.

Anatomy

Meckel's Diverticula are identical in anatomical structure with the ileum from which they arise. Their wall is composed of mucous membrane, muscularis mucosae, submucous tissue, a complete muscular coat with circular and longitudinal fibres, and a peritoneal coat.

Heterotopic tissues may be present in the Meckel's diverticulum in the form of pancreatic, biliary, duodenal, ileal, colonic and gastric tissue.

The most common type of heterotopic tissue found is gastric mucosa. Heterotopic tissues occur in approximately twenty-five percent of the cases of Meckel's diverticulum.

The gastric mucosa may form a localized tumour, usually near the tip of the diverticulum or may furnish a complete lining. The ectopic epithelium is a faithful reproduction of the structure of adult gastric mucosa, containing fully active oxyntic cells.

Pancreatic epithelium takes the form of a localized nodule (accessory pancreas) lying deep to the normal intestinal mucosa. It forms a yellowish-white tumour lying within the muscle coats and near the tip of the diverticulum. As in gastric heterotopia, adult pancreatic tissue is faithfully reproduced and islets of Langerhans may be found. One or more ducts may be

present which may open into the lumen of the diverticulum.

The factors responsible for epithelial heterotopia are not known. It is most probable that the condition arises from stimulation locally of the primitive entoderm to develop epithelium anomalous for a particular region. This is Car's view. Another view is the possibility of the phenomenon being due to fragments of tissue in the vitelline duct.

Pancreatic heterotopia is of no clinical significance by virtue of it being capable of producing pancreatic secretion, but, by forming a tumour mass at the tip of the diverticulum, may be responsible for the commencement of an intussusception. It may also be a factor in the causation of intestinal diverticula of the traction type.

It has been conclusively demonstrated that this gastric mucosa is capable of secreting hydrochloric acid and pepsin as does that in the stomach. It has been shown that it is acted on by secretory hormone that stimulates secretion in the mucosa of the stomach and that the secretion in the diverticulum occurs at the same time that it does in the stomach. The hydrochloric acid and pepsin secreted by the gastric mucosa in Meckel's diverticulum are probably responsible for the ulcerations that sometimes occur near the diverticulum.

Pathology

Meckel's diverticulum may be present throughout the

life of a person and yet cause no symptoms. It is always a source of great potential danger.

The following are the pathological conditions which may affect a Meckel's diverticulum or for which it is directly responsible:

1. Acute Inflammation: Diverticulitis;
2. Acute Intestinal Obstruction;
3. Peptic Ulceration;
4. Acute Intussusception;
5. New Growth.

1. Acute Inflammation: Diverticulitis

Symptoms and signs of acute diverticulitis are the same as in an acute appendicitis. It is important to search for a Meckel's diverticulum in cases diagnosed as acute appendicitis and in which the appendix is found at operation to be normal.

2. Acute Intestinal Obstruction

A Meckel's diverticulum may cause acute obstruction in a variety of ways: constriction and torsion are two of the ways. In all cases, it is essential to remove the diverticulum.

3. Peptic Ulceration

Gastric heterotopia frequently causes symptoms from the nature of its secretion and may be responsible for the formation of a peptic ulcer. Ulcer is identical

with peptic ulcer of the stomach in its morphology and capable of producing the same symptoms of pain, of bleeding, and of perforation.

The probable incidence of gastric heterotopia in Meckel's diverticula is about one in three thousand persons, so that symptoms arising from it are not so rare.

The ulcer is usually situated at the point of opening of the diverticulum into the ileum, i.e., at the point where the gastric mucosa ends and normal intestinal epithelium begins. It is doubtful if ulcer will occur without gastric mucosa being present. Severe hemorrhage may occur in gastric heterotipia of a Meckel's diverticulum in which no definite ulcer can be found in the specimen removed at operation but it is always possible for ulcer to be lower down the bowel.

Age Incidence

The ages at which peptic ulcer has given rise to symptoms show a wide range, though naturally most of the cases are in children.

Clinical Features

Pain is the predominant symptom. In a case of diverticulum with gastric heterotopia and ulcer, pain of a gastric ulcer type occurring in cycles and before meals with relief from food, was a notable feature.

Bleeding is of common occurrence. The haemorrhage may be alarming and exsanguinate the patient and it may

prove fatal. The haemorrhages are repeated at intervals. Diagnosis of intestinal polyp or angioma is usually made until a laparotomy is performed or perforation has occurred. A fairly massive painless intestinal haemorrhage in a child is very suspicious of Meckel's diverticulum. Perforation is a very common complication of peptic ulcer in Meckel's diverticula. It is usually free into the peritoneal cavity but may occur into the mesentery or into the colon.

Diagnosis

It is not possible to make a clinical diagnosis of peptic ulcer of Meckel's diverticulum but secondary aenemia associated with intestinal bleeding and with x-ray evidence of normal stomach and duodenum and colon should arouse suspicion of an ulcer in the ileum or in a Meckel's diverticulum.

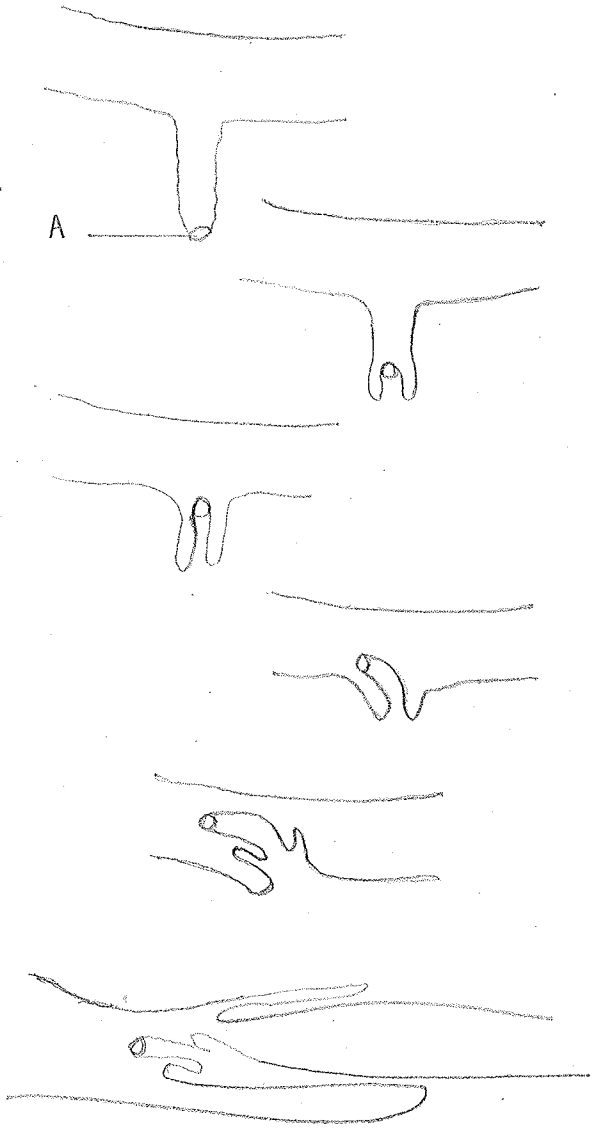
In differential diagnosis one has to consider Henoch's purpura, in which condition there will be bleeding elsewhere and alterations in the clotting and bleeding time. The presence of polypi, ulcers and haemangioma of the bowel can usually be proved by x-rays.

Treatment

Laparotomy and excision of the diverticulum.

4. Acute Intussusception

The exciting factor in cases of acute intussusception



Probable mode of development of the intussusception due to inversion of the diverticulum. A is the Pancreatic nodule.

initiated by a Meckel's diverticulum is usually a nodule of pancreatic or of gastric epithelium situated near the fundus. There seems to be little doubt that invagination of the diverticulum initiates the intussusception, and the cause of the invagination is the presence of a solid tumour of gastric or pancreatic epithelium.

There are usually signs and symptoms typical of acute intussusception. A history of haemorrhage frequently precedes the onset of the intussusception. Age incidence is from three months to forty-nine years. The treatment is operative and the diverticulum should be removed. Mortality is fifty-five percent.

5. New Growth

New growth arising in a Meckel's diverticulum is a great rarity. A simple polyp, argentaffinoma, medullary carcinoma, adenocarcinoma, and sarcoma have been reported.

Treatment

The treatment of Meckel's diverticulum is surgical. It must be directed toward relieving the symptoms produced by the diverticulum. If intussusception is present, it must be reduced surgically. If obstruction is present, it must be relieved. If a fistula is present, it must be closed. If a diverticulum is enclosed in a hernial sac, it must be reduced. If

recurrent pains are the symptoms, they must be relieved. All procedures must include excision of the diverticulum. When the diverticulum is excised, closure should be made accurately in a transverse plane which is perpendicular to the long axis of the bowel. If there is narrowing of the bowel, the simplest thing to do is to resect the segment in which the diverticulum is located.

Mortality is still high, around 9.3 percent, but can be greatly reduced by early diagnosis and by early surgery.

Mr. Roy H. Age 28
Admitted to Winnipeg General Hospital , August 18th, 1939 complaining of crampy abdominal pain for six days. He was seized with general abdominal pain; with onset of pain he felt desire to defecate which he could not do. Two days later after a bowel evacuation the pain went away for about six hours. The pain retunred in the afternoon and was more severe. His whole abdomen was tender. He kept on working. Pain and tenderness again disappeared. He was given an enema and felt much better. He took A.B.S.& C tablet, Milk of Magnesia, and Castor Oil and the pain returned. A large bowel movement followed next day. He was admitted to hospital six days after the pains started.

There was no nausea or vomiting.

On admission he still had crampy abdominal pain with general abdominal tenderness and slight rigidity of both recti muscles. White blood count was 19,940, temperature 99.2. Diagnosis of acute appendicitis was made. He was operated on August 19th. The appendix was normal and not removed. Meckel's Diverticulum was found, perforated and adherent to sigmoid above pelvic brim. The Diverticulum was removed and catheter purse stringed in the opening. Abdomen was drained and closed.

Pathological report: "Meckel's Diverticulum. Two irregular pieces of tissue 3 x 2 cms and 2 x 1 cms covered with pyogenic exudate. The larger has mucosa at one end, with a collapsed opening extending up 1.5 cms. Microscopic: acute inflammation."

Patient made a good recovery.

opening the bowel the diverticulum is seen to open into the lumen by a small pouting orifice. The opening and bulging mucosa together measures 1 cm. The hole in the ileum is immediately adjacent to the mouth of the Meckel's. It is seen to be a perforation of an ulcer 1 cm. in diameter with shelving edges and distinct thickening around its edge. The rest of the bowel mucosa is normal. The appendix is involved only in the general process. The stomach is normal. Smears of pus showed numerous mixed organisms including streptococci and B. Welchii.

Section through Meckel's diverticulum and perforated ulcer was examined. The diverticulum shows a gastric type of mucosa with very numerous parietal cells. This type of mucosa extends to the mouth of the diverticulum but the ileum shows a normal type of intestinal mucosa. The submucosa of the ileum is fibrosed and shows a chronic inflammatory reaction. The edge of the perforation shows much necrosis. The subserosa is also thickened and fibrous. This is an acute inflammatory exudate in the peritoneum which is very thick at the site of perforation.

This case is one of large Meckel's diverticulum in an infant. The mucosa is of gastric type with ulceration of ileal mucosa at their junction. Ulceration does not occur in the acid secreting mucosa but in the ileal mucosa next to it. Same is true in gastric ulcers. There was perforation of ulcer, with acute generalized peritonitis and fatal outcome.

There can be no doubt that the diverticulum, on account of its great size; its structure, which is identical with the intestine; and the fact that it gave rise to symptoms in infancy is of congenital origin.

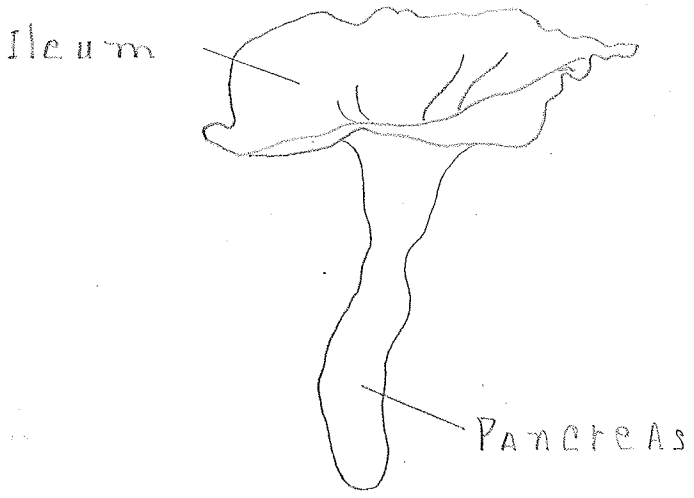
Etiology is congenital. Origin is a reduplication of the gut, which may be small or large.

2. Cyst-like Diverticula

It is a large cyst-like structure situated between the leaves of the mesentery. It appears independently of vessels and although always on the mesenteric border of the gut, the situation here has not the same significance as in acquired diverticula. Its shape suggests very clearly that it bears a relation to cysts in the intestinal wall. It is probable that cyst-like diverticula and true cysts of the intestinal wall share a common etiology. It is difficult, however, to determine their exact relation. A diverticulum may result from a cyst by acquiring a secondary communication with the bowel. Conversely, a cyst may be formed from a diverticulum by the sealing off of the latter's communication with the bowel.

3. Traction Diverticula Associated with Accessory Pancreas

A diverticulum may arise as a result of traction by an accessory pancreas.



Nauwerck's case was obtained from a man of 43. It was a funnel-shaped diverticulum nine centimeters long opening by a wide mouth from the anti-mesenteric border of the ileum and attached at its apex to an elongated mass of normal pancreatic tissue. The diverticulum was situated two centimeters above the ileocaecal valve; eighty centimeters from the ileo-caecal valve was a typical Meckel's diverticulum. In view of its shape and the presence of a "normal" Meckel's diverticulum, one may, I think, truthfully regard it as an example of a congenital traction diverticulum.

DIVERTICULA OF THE PHARYNX

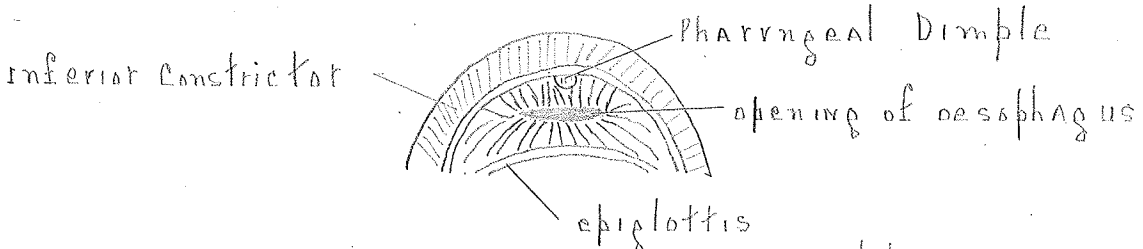
There are two types:

1. Congenital lateral diverticulum, which is really a blind internal branchial fistula opening into the fossa of Rosenmuller. Occasionally, such a fistula becomes greatly distended and food lodges within it. This condition is rare.

2. Pharyngeal Pouch: It commences in the posterior wall of the pharynx one centimeter above the commencement of the oesophagus. It should not be called oesophageal pouch. Pulsion diverticulum and pressure diverticulum are two synonyms.

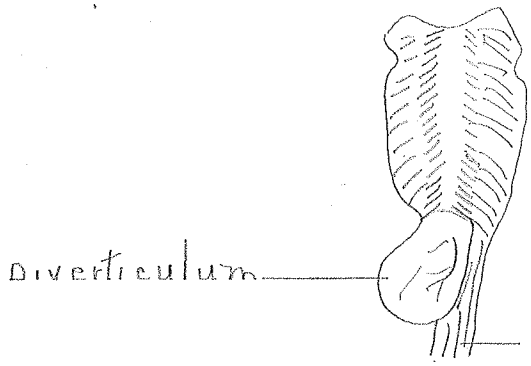
Etiology

The diverticulum commences at the pharyngeal dimple.



Showing the Phayngeal Dimple

After middle life, behind this dimple, some separation of the musculature of the inferior constrictor is apt to occur and the mucous membrane tends to herniate when a bolt of food is swallowed. So commences a pharyngeal pouch. As time goes on, the sac becomes larger and fills with food at every meal.

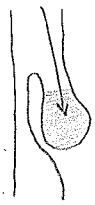


Pharyngeal diverticulum seen from Posterior Aspect

Unable to expand posteriorly because of the resistance of the spinal column, the pouch turns outwards, usually to the left, and obtrudes itself into the side wall of the neck.

Symptoms and Diagnosis

In about one-third of cases, the pharyngeal pouch is large enough to form a visible swelling. Sometimes the pouch can be seen to enlarge when the patient drinks water. The condition occurs principally in elderly men whose main complaint is dysphagia, for when the sac becomes full its lower part presses upon the oesophagus.



Showing how a pharyngeal diverticulum causes dysphagia

Regurgitation of undigested food often occurs. An irritable cough and gurgling noise in the neck also be symptoms. Dysphagia may eventually lead to progressive loss of weight and even extreme cachexia. The patient should be investigated by x-ray examination after the ingestion of a barium meal. Fundus of the pouch may at times be seen invading the superior mediastinum.

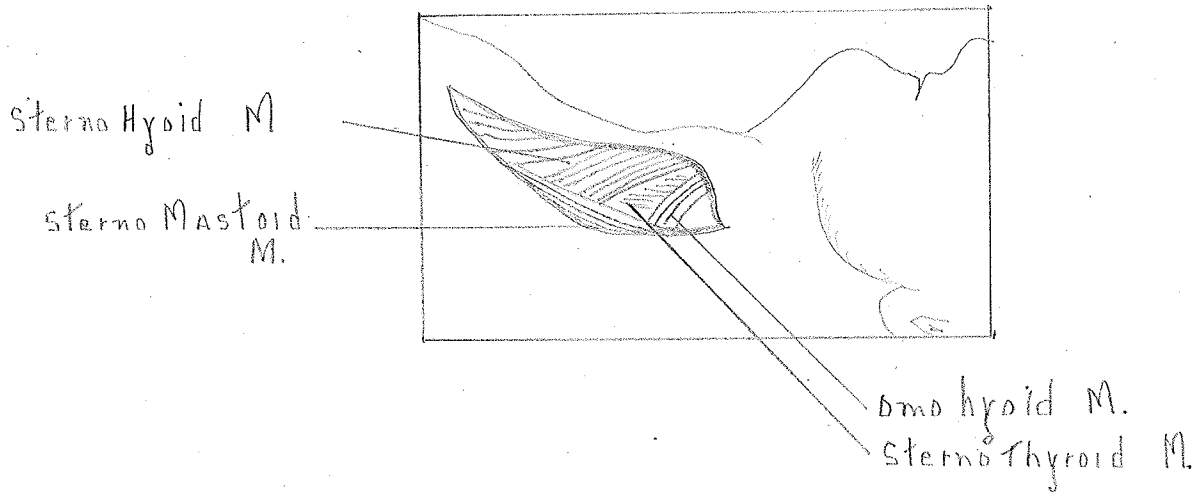
Treatment

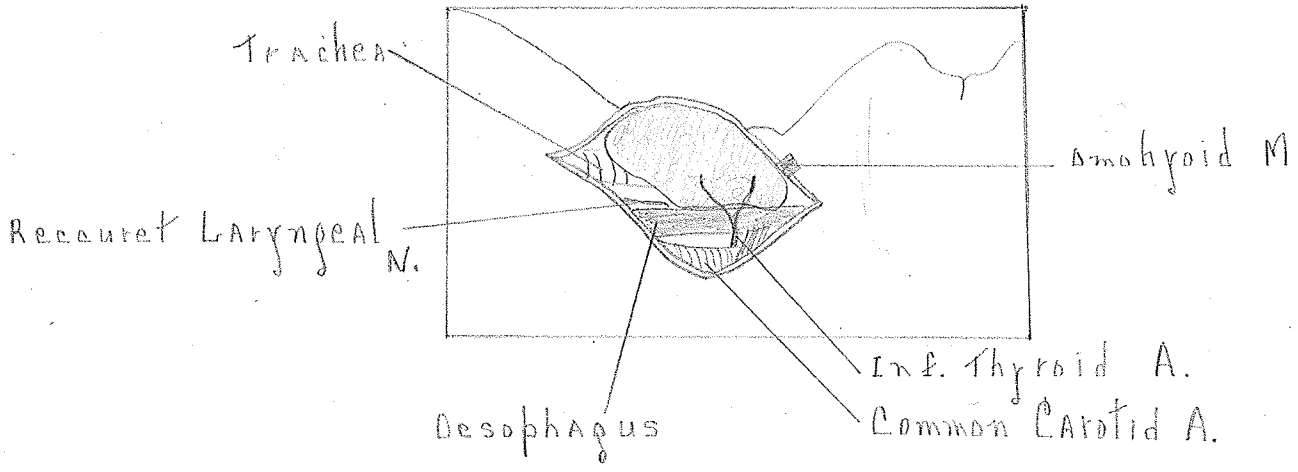
Treatment is surgical.

Operation

The incision should be adequate. It extends from the cricoid cartilage in front of the sterno-mastoid to just above the clavicle on the left side. A transverse incision extending from the middle of the neck at the level of the cricoid cartilage to the middle of the left sterno-mastoid muscle is seldom used. The neck is hyper-extended by means of a large sand bag under the shoulders. The sterno-mastoid muscle is defined and retracted. The pre-tracheal muscles are exposed and retracted medially. The pre-tracheal layer of the cervical fascia comes into view and is divided. The lateral lobe of the thyroid gland is dislodged and retracted to the right. In most cases, the inferior thyroid artery passing inwards from behind the common carotid artery towards the middle of the thyroid gland, will require ligature and division. Omohyoid muscle may need dividing. The oesophagus is found between the trachea and the

cervical vertebrae.





Good light is necessary. The oesophagus is exposed. A bougie may be passed into the sac from above through the mouth in order to make the sac more prominent or an oesophagoscope can be passed into the sac if necessary. The sac is carefully isolated by the division of adhesions between it and the wall of the oesophagus. One has to obtain complete isolation of the sac and the neck of the sac freed until its junction with the oesophagus can be clearly demonstrated.

One of three procedures can now be adopted:

1. The sac, when isolated, may be fixed to the deep structures of the neck so that the fundus is at a high level and food cannot any longer enter its cavity.
2. Two stage operation.
3. The sac may be removed at a single stage operation.

Operations 1 and 2 are employed in order to avoid the danger of infection and the development of serious septic cellulitis or mediastinitis. With use of Penicillin and Sulfa drugs, the single operation is preferred and is successful.

The neck of the sac at its junction with the oesophagus is secured by fine tissue forceps and the whole sac removed with scissors. Too much traction should not be exercised. The slit-like opening is now accurately closed with fine cat-gut and made water tight, the sutures being continuous. If the neck of the sac is very narrow, it can be ligatured, cut and antiseptic solution applied. Chromic or intestinal suture is used. The second row of sutures brings the muscular wall of the oesophagus over the sutured mucous membrane. Sulfathiazole crystals are placed in the wound. The wound in the neck is closed layer by layer. Drainage is not necessary very often.

Post-operative Care

Patient is fed for one week through a nasal tube passed into the stomach.

DIVERTICULA OF THE OESOPHAGUS

Diverticula of the oesophagus are divided into two types:

1. Pulsion
2. Traction

Pulsion Diverticula

Pulsion diverticula are found only in the region immediately above the diaphragm; to them the term Epiphrenal has been applied. The condition is rare. These diverticula are usually small in size but one as large as a human fist has been reported.

The etiology is not clear. In some of the specimens, the presence of a complete muscular coat would seem to indicate a congenital origin; while in others this layer is wanting and the diverticulum is a hernial protrusion of the mucosa through a gap in the muscular coat.

It is undisputed that increase in size results from accumulation of food contents and pressure acting on this mass from the oesophageal lumen. Diagnosis is assisted by complaint of dysphagia; vomiting of some food, such as rice or raisins, known to have been ingested some days previously; and often by the subjective feeling of pressure deep under the sternum. The condition may remain latent for years and it is only the onset of some complication which brings the patient to seek surgical aid. Radiology is essential to determine the exact

site and dimensions of the sac.

Complications

1. Cardiospasm
2. Diverticulitis: which may be of acute or chronic type.

Acute inflammation may cause sudden perforation leading to acute mediastinitis or a more chronic inflammatory permeation leading to diffuse mediastinal adhesions.

3. The other complications, such as ulceration, new growth, are very rare.

Treatment may be palliative or radical. Palliative measures consist in a bland diet and lavage of the sac may be attempted through the oesophagoscope. Radical measures may be necessary. Two operations are possible:
(a) Extirpation of sac;
(b) The formation of an anastomosis between the diverticulum and the stomach.
Both procedures carry a high mortality but the latter is the safer of the two.

Extirpation of the sac is especially indicated when the diverticulum lies in such a position as to make anastomosis difficult to per form. The trans-pleural approach is used. Section of the phrenic nerve is necessary.

The anastomosis operation is carried out from below

the diaphragm and if the sac can be readily exposed by this route it is to be preferred to the trans-pleural. Where massive adhesions make exact anatomical display almost impossible, the technique follows the same lines as that of oesophago-gastrostomy and is better divided into two stages. In the first stage, the lower oesophagus is exposed by the left trans-pleural route; the phrenic nerve is divided, the diaphragm split and a portion of the fundus drawn up inot the chest and sutured to the diverticulum. The wound is closed without drainage. Four weeks later a second thoracotomy is performed and the anastomosis completed.

Traction Diverticula

Small in size and rarely multiple, these diverticula are the result of chronic inflammatory change in the oesophageal wall. Probably the most common cause of this inflammation is tuberculous disease of the lymph nodes at the bifurcation of the trachea, but vertebral caries or pericarditis may be responsible. In some cases, portions of ingested food may become lodged in the lumen and so act as a tension factor which contributes to increase in size. The dimensions of these diverticula vary from that of a hazel nut to that of a walnut. They are usually placed on the anterior wall of the oesophagus and the apex of the diverticulum is directed upwards.

Diagnosis

Traction diverticula are seldom diagnosed during life but at times they assume considerable importance and may even be the cause of fatal illness. Diagnosis can only be made after perforation and this complication is rarely seen. The accidental inclusion of a sharp fragment of bone in the sac may predispose to it. Perforation into the mediastinum will produce a rapidly spreading fatal infection unless the ground has been prepared by a previous inflammation, when localized abscess offers some hope of treatment. Perforation into the bronchial tree - the more usual event - may cause immediate suffocation as the contents are aspirated; or an acute infection of the lung may result, followed in favourable cases by the formation of a localized abscess. The expectoration of ingested material is certain evidence of this catastrophe.

Treatment

Treatment is indicated only when complications are present. Mediastinal perforation and infection should be dealt with by open operation and drainage, supplemented with Penicillin and Sulfa drugs pre and post-operatively.

The treatment of those cases of bronchial perforation which survive to present a picture of chronic abscess of the lung, consists of a suitable drainage of pulmonary infection, a plastic operation to close the fistula and

finally a second plastic operation to heal the defect in the lung. All this is supplemented with Penicillin and Sulfa drugs pre and post-operatively.

Mr. J.L. - Age 86

Admitted April 25th, 1945

Entrance complaints were:

Shortness of breath for 10 years.
Bringing up of food, which he had eaten one half to two hours before for 4 months.
Coughing up blood for 4 months .
Cramps in his legs on exertion for 2 to 3 years.

This was a very old man, speaking no English and could not give any detailed information. He was treated for congestive heart failure.

His examination showed other findings but one interestong one. X-ray showed diverticula in the lower third of the oesophagus, but no obstruction. The upper gastro-intestinal tract was negative.

He was discharged on May 23rd, 1945, as improved.

Regurgitation of food one half hour to two hours after meals was no doubt caused by diverticula of the lower third of oesophagus.

DIVERTICULA OF THE STOMACH

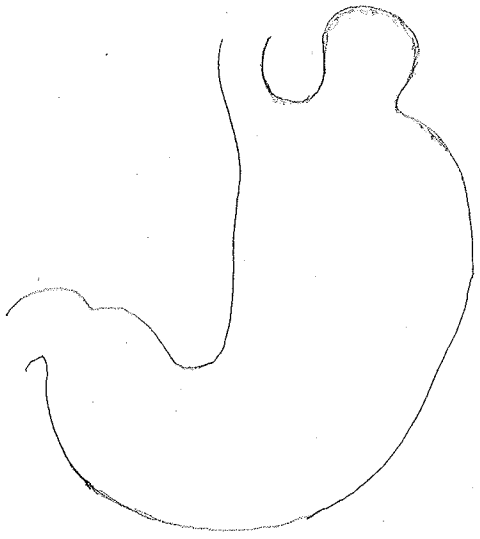
Helmout, in 1804, reported the first diverticulum. True diverticula of the stomach are rare, only second last to jejuno-ileum. They are more common in males and usually are found in fifth to sixth decades of life. Diverticula elsewhere in the digestive tract may be associated.

Classification

Included are not only true diverticula whether congenital or acquired, but also diverticulous formation resulting from intrinsic lesions of the stomach, such as ulcer.

1. True Diverticula

There is pouching which includes all coats of the gastric wall without definite evidence that organic disease was the causative factor. These are probably congenital.



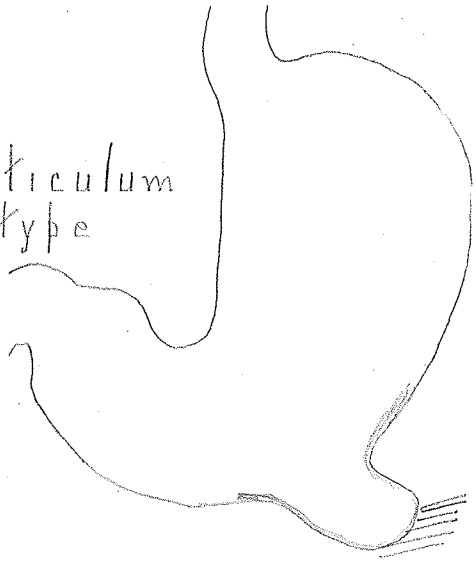
true diverticulum at cardiac end of stomach. the mural elements are intact but thinned out.

2. Acquired True Diverticula

All coats of the wall are present but there is evidence that some disease was instrumental in causing the pouching.

(a) Pulsion type: these result from intragastric localized pressure.

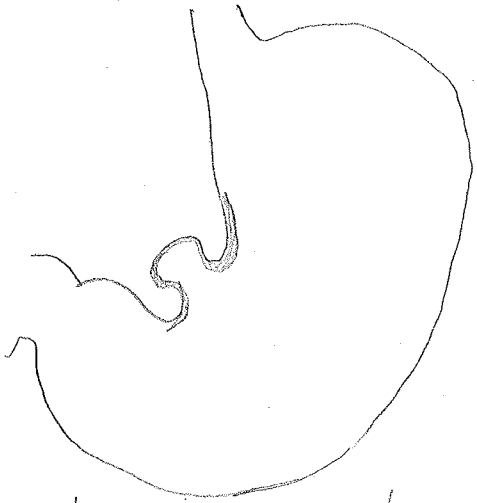
(b) Traction type: these are incident to extra gastric adhesions.



Acquired true diverticulum of traction type

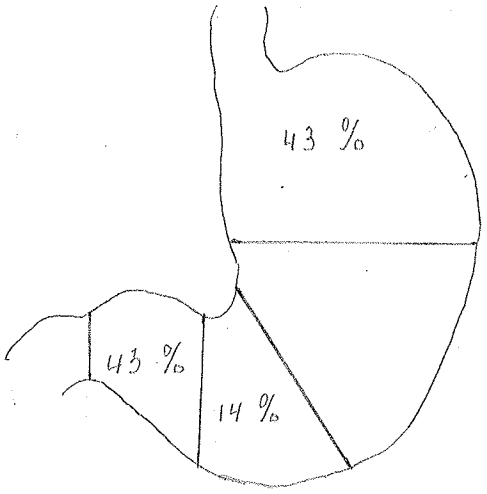
3. False Diverticula

These are diverticulous formations in which there is a break in the gastric wall incident to some disease which has invaded the wall of the viscus.



False diverticulum due to wall weakened by ulcer

Situations of Gastric Diverticula



Etiology and Pathogenesis

The etiology of true (congenital) diverticula is unknown, but the relative weakness of the musculature at the cardia would account for diverticula in this situation. The etiology of the acquired type of diverticula usually is obvious, in as much as they often are attributable to adhesions to the gall bladder, pancreas, spleen, and so forth. The false type, apparently, is the result of weakening of the gastric wall by an inflammatory reaction, ulceration or neoplasm. Intragastric pressure or progressive ulcerating processes, under such circumstances, could produce pouching at the site of involvement. In congenital and acquired true diverticula, the mural elements are intact but thinned out.

Symptoms

These are never characteristic. Epigastric pain

is most common and is relieved or aggravated by food. Nausea and vomiting, cardiospasm and melena with secondary anemia, are other symptoms. Deep epigastric tenderness is occasionally found.

Diagnosis

As there are no characteristic symptoms, diverticula cannot be recognized without the aid of roentgenology. X-ray photograph will show a tubular process with a blind end when filled with barium meal.

Complications

Haemorrhage is the commonest complication.

Treatment

1. Exclusion of all other possibilities, such as ulcer, carcinoma, cholecystic disease, or functional gastric disorder.
2. Treat medically if the symptoms are mild: treatment consists of a bland, smooth diet and administration of alkalis one or two hours after meals.
3. Post-ural drainage for diverticula at the cardia.
4. If medical regime fails or if there is evidence of an associated lesion, operation would be the most appropriate procedure. All cases in which the lower two-thirds of the stomach is involved should be operated on, because of the frequency of mistaken diagnosis of diverticula in this region.

Operative Procedure

1. For the tubular form of diverticulum, amputation by means of crushing clamps, reasonably close to the wall of the stomach, with closure and inversion of the stump is all that is necessary.

2. For the more globular type and when communications with the stomach are large, care must be taken in inversion of the stump, since the wall is much thinner than the wall of the stomach itself.

3. If secondary changes, such as an ulcer or malignant process, have taken place in the diverticulum, it may be necessary to excise a portion of the gastric wall, with the diverticulum to make certain that all secondary evidence of disease has been removed.

DIVERTICULA OF THE DUODENUM

Introduction

Chomel's case, published in 1710 is the first reference to diverticula of the duodenum to be found in the literature. Case in 1913 is credited with the first roentgenologic diagnosis of duodenal diverticulum verified on operation.

Classification of Duodenal Diverticula

1. True congenital, i.e., a diverticulum present from birth and identical in structure with the intestine from which it rises - is a condition of exceptional rarity in the duodenum.
2. Acquired hernial (or primary) type.
3. Secondary - with ulcer
   - traction.

Incidence

The percentage incidence is 0.75 percent of routine radiographs at King's College Hospital from 1925 to 1937 inclusive. The acquired hernial type is the most common.

Radiological Appearance

Duodenal diverticula are seen as smooth, barium filled pockets persisting at a three hour or six hour radiograph. They are usually round in outline, but may yield a mushroom-shaped or pear-shaped contour, especially those situated at or near the duodeno-jejunal flexure. Unless large and well defined, a pocket in the first part of the duodenum

associated with an ulcer will not be revealed. The duodenum may show marked deformity, but no definite diverticulum. Furthermore, the pockets secondary to ulcer are in most cases shallow and wide-mouthed and there is no retention of barium in them. They empty with the duodenum. The oblique position for examination is of importance.

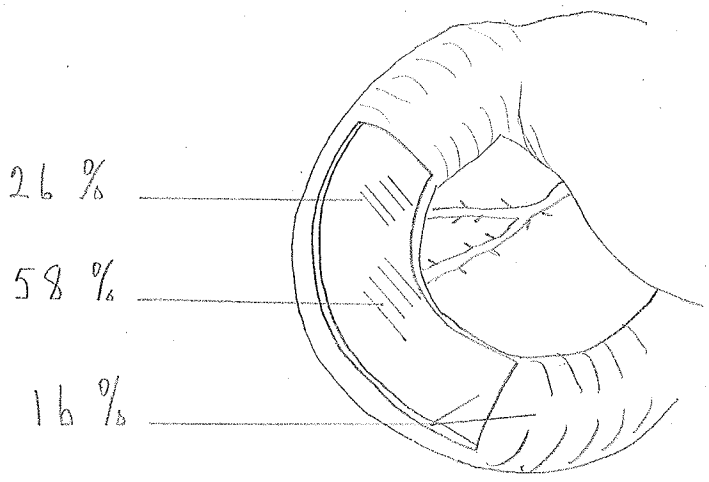
Traction diverticula are rare and difficult to distinguish radiologically from other types. The most frequent sites for traction diverticula are in the gall bladder region and at the flexure, where occasionally the intestine may become caught up by peri-gastric fibrosis around an ulcer of the lesser curvature of the stomach.

Small ulcer crater in the first part; dilated common bile duct or anatomical variation of the opening of the bile and pancreatic ducts into the duodenum; anatomical variation in the duodenum; shadows due to gall stones and pancreatic stones; ileus; differences in shape; abnormal position; the enlarged bulb due to an annular pancreas, of duodenum are some of the pitfalls to be watched for in x-ray diagnosis. The most important procedure in x-ray diagnosis is the screening operation - palpation of the duodenum under observation.

Primary Acquired Diverticula of the Duodenum

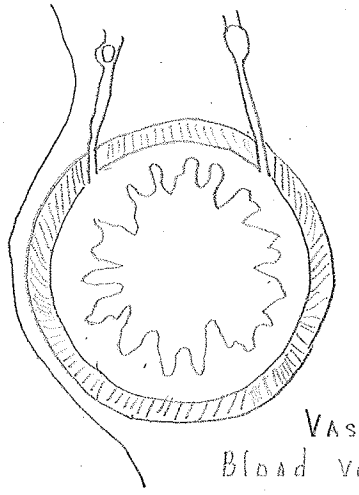
The average age of patients is fifty-six years, more common in women than in men, the proportion five to two.

By far the greatest number of primary acquired diverticula occur in the second part of the duodenum in close relation to the entrance of the common bile and pancreatic ducts. Two diverticula may be present. More than two are rare.



The diverticula are all necessarily related to the pancreas lying either behind it or partly buried in its substance. All diverticula arise from the concave surface of the bowel, at or near the ampulla of Vater. Diverticula arising in relation to the entrance of the common bile duct are called perivaterien. The concave side has the weak areas in the bowel occasioned by the passage of the common bile and pancreatic ducts and the blood vessels. The passage of the ducts through the duodenal wall occasions a considerable gap in the muscle fibres. The longitudinal coat is most affected, for in the angle between the tracks of the pancreatic and bile ducts a well defined circular coat exists, whereas the divarication of the muscle fibres of the longitudinal coat is practically

continuous. The weakness continues for some distance below the entrance of the ducts.



Vascular Supply of duodenum.
Blood vessels entering bowel wall.

Pathogenesis

The diverticula are hernial protrusions of the bowel lining beginning at a weak spot in the muscularis. The muscular coat is grossly deficient, sometimes to the extent of complete absence. A typical primary duodenal diverticulum is a thin-walled sac opening into the bowel by a wide mouth. It is rounded in shape. The edges of its mouth are well defined and may be in continuity with one of the plicae inside the intestine. The lining is smooth and there may be a complete peritoneal covering. In all the cases there is a close relation either to one of the ducts piercing the duodenal wall or to a blood vessel. The wall is thin. They are not larger than a golf ball. In microscopic examination of a section taken from the fundus shows no muscle. Mucous membrane is thinned and Brunner's glands are absent.

The formation of diverticula depends on two factors:

(a) Locus minoris resistentiae;

(b) Pressure inside the cavity.

Areas of diminished resistance are furnished by the piercing of its muscular coat by the bile and pancreatic ducts and by the blood vessels. The pressure inside the duodenum depends on: (a) the contents; and (b) the contraction of the muscular wall. The contents are fluid. Irregular, incoordinated tonic contractions of the circular and longitudinal coats increase the intra-duodenal pressure and an explanation of the mode of origin of these primary diverticula becomes possible.

The mucous membrane of the duodenum is forced into the interval occasioned by the passage through the duodenal wall either of the ducts or the blood vessels by the circular contractions of the duodenal muscle. In the early stages, the mucous membrane will recover its position, but once started there will be a certainty of recurrence, and repitition of the event over a number of months or even years will result in a stretching of the interval between the muscle fibres and its permanent occupation by a wedge-shaped pouch of mucous membrane. The same process occurs in jejunum and colon. Once the projection of mucous membrane is established, the formation of a typical pouch is inevitable, if the patient lives long enough.

Duodenal diverticula occur with diverticulosis of the colon; visceroptosis; peptic ulceration; gall stones and

cholecystitis; and chronic appendicitis. If there is a definite relationship, it is not certain. One must bear in mind that these patients are x-rayed because of gastro-intestinal symptoms and the associated diverticula are only accidentally discovered and may receive more emphasis than is in strict accordance with the facts.

The Ulcer Diverticulum

Ulcer diverticula form a distinct group and are found readily at operation. Each diverticulum consists of all four layers of the normal duodenum. Its peritoneal surface is free and smooth. Its mouth is well defined, and bounded by a definite ridge of mucous membrane. The lining is, in most cases, smooth and free from well defined rugae. There is no inflammatory process in its wall. The associated ulcer lies at some distance from its edge and not in the wall or floor.

Pathogenesis

The diverticula are produced by contraction of scar tissue of the healed ulcer. The duodenum is shortened so that the wall of the duodenum unoccupied by the ulcers has been puckered up and ballooned outwards by intraduodenal pressure to form diverticula. The actual shortening may be as much as 2.6 centimeters. Pressure plays a part as shown by absence of rugae and by thining of the muscular coat. It is probable that the persistent spasm of muscle associated with ulcer plays a part by helping to create the deformities,

which are subsequently fixed by the scar contraction.

Traction Diverticula

These are caused by the contraction of scar tissue adherent to the wall of the duodenum. Most commonly, gall bladder would be adherent and the wall of the duodenum is pulled out to form a small pouch.

Pseudo-Diverticula and Unclassified Cases

These are perforations of the duodenal wall into the peritoneal cavity giving rise to a diverticulum, the walls of which are formed by fibrous tissue. Communication between the duodenum and the gall bladder may also be regarded as a pseudo-diverticulum. Pseudo-diverticulum may be associated with a growth.

The Clinical Aspect

The majority of primary diverticula probablu give rise to no symptoms at all and their radiographical discovery is accidental, the examination being undertaken to investigate pain arising from other causes. They may contribute to dyspepsia and give rise to serious symptoms only on rare occasions.

What are the Criteria by which Diverticula are held responsible for symptoms?

They may be described under two heads:

1. The absence of any other lesion such as diverticulosis of the colon, peptic ulcer, visceroptosis, gall bladder

disease and other diseases of the liver, and chronic appendicitis, have to be excluded.

2. Direct radiological evidence

(a) Size of the diverticulum: a large diverticulum with prolonged retention may be regarded definitely as a factor in the causation of the symptoms. Retention of six hours or more is to be regarded as "pathological".

(b) Tenderness on palpation under the screen is of no value.

(c) Situation of the diverticulum: diverticula arising in third part of the duodenum and peri-vaterien only rarely cause symptoms. Those situated near the duodeno-jejunal flexure are liable to produce symptoms due to pressure.

Symptomatology

There is no characteristic symptom complex but few groups are as follows:

(a) Flatulent dyspepsia: the dyspepsia is of a vague type lasting over many years, the chief feature being flatulence, pronounced borborygmi, and a sense of oppression in the epigastrium after meals. This is due to retention of duodenal contents. It is indistinguishable from chronic cholecystitis and gets worse as the patient grows older.

(b) Epigastric pain - ulcer type; this is due to diverticulitis.

(c) Partial duodenal obstruction: attacks of pain of colicky type situated in the left lumbar region.

(d) Acute diverticulitis and perforation.

(e) Acute duodenal obstruction.

(f) Obstructive jaundice due to:

i. Ascending infection of the common bile duct secondary to diverticulitis;

ii. Constriction of the lower end of the common bile duct by an area of pancreatitis secondary to inflammation of the diverticulum.

iii. Obstruction of the common bile duct by pressure of a diverticulum upon the lower end.

(g) Pancreatitis.

Secondary Diverticula

Diverticula due to ulcer and traction diverticula are not likely to cause symptoms on their own account. These are in all cases due to the underlying cause, and must be treated accordingly.

Operative Technique

This may be difficult and dangerous, especially if

the diverticulum is posterior, close to common bile duct or buried in the pancreas. Injury is to be avoided. The operative procedure falls under two heads:

(a) The exposure of the diverticulum;

(b) The method of dealing with the diverticulum.

Exposure may be transduodenal or mobilizing duodenum and exposing its posterior surface.

Diverticulum should be removed and defect closed in layers. If there is narrowing of duodenum, a posterior gastro-jejunostomy should be done.

Diverticulum could be suspended by the fundus to keep it draining and prevent retention. Another procedure is to invert it into the duodenum with closure of the neck.

Mrs. R. Age 49 Admitted to Winnipeg General Hospital May 8, 1941

In 1934 she began having pain in right dorso-lumbar region. Pain came on in attacks of two to three weeks, occurring at intervals of two to three months. No particular rhythym or sequence of pain. No relation to food. Pain is worse at night and relieved by vomiting. Vomiting approximately once or twice a week during an attack. No jaundice. Had gaseous eructations after meals.

In 1940-41, attacks of pain more frequent and more severe. Had three or four attacks of pain requiring morphine.

In February 1941, pain became more continuous and still located in right dorso-lumbar region; no radiation. Patient became confined to bed. Pain was markedly increased by movement. No relation to food. Began losing weight; vomiting everything she ate. Pain was relieved by vomiting.

In March 1941, cholecystectomy was done in Kenora, Ontario.

In May 1941, she began to have epigastric pain - described as steady burning pain in left side of wound - radiating down below umbilicus. No pain in back now. Pain comes on one hour after food, lasts one hour - relieved by vomiting as a rule. Also relieved by milk. Has been vomiting for last four days.

Admitted to hospital May 8, 1941.

Review of systems, family and personal history are essentially of no importance.

Past Illnesses

1926 Appendectomy and ventral suspension

1926 Inguinal herniotomy

1927 Caesarian section for placenta praevia

1928 Removal of duodenal diverticulum? or gastro-enterostomy

1929 Artificial menopause with deep x-ray

Marital History: Para iv Gravida vii; one miscarriage; all instrumental deliveries.

Examination: except for tenderness across epigastrium was negative.

A tentative diagnosis on admission was:

(a) stoma ulcer;

(b) Distortion of stomach through adhesions causing retention.

May 12, 1941: Barium series was done and reported as follows:

Stomach - if there is an enterostomy stoma it is not functioning. No lesion could be demonstrated in the stomach.

Duodenum - The duodenal bulb is negative. In the third portion of the duodenum there is a large diverticulum which is tender on palpation and multiple diverticula varying in size in the descending and transverse portion of the duodenum.

5 hours - There is a trace of barium in the stomach. The diverticula are filled with barium. The head of the meal is in the terminal ileum.

Summary - There is no evidence of an enterostomy stoma. Multiple diverticula of the duodenum. Nine hours after ingestion of the barium meal the stomach is empty. There are some traces of barium in the epigastrium which is evidently in the diverticula. The head of the meal is in the ascending colon.

While in hospital, she was put on sippy diet with antispasmodics with no improvement in symptoms. As long as she was on milk, she did not vomit but could not eat solid food without nausea.

May 29, 1941: Duodenal drainage showed pus present.

May 23, 1941: Icterus Index was 7.

Occult blood in stools was found in one out of four specimens.

Urinalysis and blood examinations were normal.

She was operated on and resection of diverticulum of third part of duodenum done. Pathological report: "A diverticulum 3 centimeters in diameter. The mucosa is congenital, wall is two millimeters thick. Micro: The musoca is congested and contains a moderate number of chronic inflammatory cells. The submucosa shows moderate fibrosis and the musculosa is thinned and the arterioles thickened. Diagnosis: Duodenal diverticulum with evidence of past inflammation.

She was discharged from the hospital on June 16, 1941, with some separation of the skin incision. She was instructed to do dressings at home and to increase diet gradually avoiding coarse foods.

There are no follow up notes on her case.

Mr. J. T. - aged 70, was admitted to the Winnipeg General Hospital on June 11, 1940, complaining of attacks of epigastric pain and vomiting for twenty years. Each attack started as epigastric discomfort, gradually increased and was relieved by vomiting and taking of soda bicarbonate. Food increased the pain and lean meat and crust of bread either started the attack or made the pain worse. Pain radiated straight through to his back. No haematemesis or tarry stools. Review of systems - all negative.

The physical examination was essentially negative except for jaundice. X-rays show no abnormality in stomach or first part of duodenum. Second and third parts of the duodenum had large barium filled diverticula. At the end of five hours the diverticula of the duodenum retain barium. Gall bladder did not visualize with Graham's method.

His icterus index was 37 on June 12 and on June 19th icterus index was only 7.

There was no occult blood in the stools. On admission haemoglobin was 85, red blood cells 4,400,000, color index .9, and a normal stained film. Blood Wassermann reaction was negative. White blood cells were 18,700 on admission. Urinalysis was negative.

He was discharged on July 4th. Diagnosis on discharge was "Diverticula of and and 3rd part of duodenum, with acute diverticulitis, producing obstructive jaundice.

He recovered on medical treatment and was discharged without any surgery attempted.

DIVERTICULA OF THE JEJUNUM AND ILEUM

This condition is very rare. In eleven thousand three hundred and sixty-two barium examinations at King's College Hospital, seven cases only were found (0.06 percent). The first complete description of the condition appears to have been made in 1844 by Sir Astly Cooper who found numerous pouches of the jejunum at a post mortem upon a man of sixty-five years of age. The average age at discovery is fifty-five, the extremes being thirty-eight and seventy-five. The condition is more common in males.

Morbid Anatomy

The diverticula may be single or multiple. Most diverticula of the small intestine are of the acquired or mucous membrane hernia type, similar to those affecting the duodenum; similar also to those which are of such frequent occurrence in the colon: that is, all are hernias of mucous membrane. There is no difference in morphology between the single diverticulum and the multiple cases. The site of herniation through the wall of the intestine corresponds with the passage through it of the blood vessels. The diverticula are on the mesenteric aspect of the intestine. They do not, however, push between the leaves of the mesentery unless they are of large size or unless they fuse with a fellow

from the opposite side of the mesentery. In the case of the diverticulum on the anti-mesenteric side of the intestine, the opening corresponds with a blood vessel of unusual size. The diverticula may carry with them, during their development, a layer of muscle fibres from the muscular coat of the bowel. This may form a thin but complete muscular coat for diverticula of small size. In the larger diverticula the fundus is completely devoid of a true muscle coat. The muscle is limited to walls at the commencement of the diverticulum.

The increase in size of the diverticulum is at the expense of the mucosa, submucosa and serous coat.

At the mouth of the diverticulum there is some evidence that the muscle coat has hypertrophied, so that in the specimens a firm ridge is produced, rather like a sphincter. In the larger diverticula, there may be hypertrophy of the muscularis mucosae to compensate for the absence of a true coat. There is often hypertrophy of the muscularis of the affected part of the bowel.

The ileum is relatively immune and is affected only in severe cases in which diverticulosis has started in the jejunum and spread steadily down the bowel until the ileum is reached and similarly affected. In these cases, the diverticula are always largest and most closely set together in the upper part of the jejunum.

Pathogenesis

Two factors must be held responsible for their development:

(a) The presence of a weak area in the bowel wall - a locus minoris resistentiae;

(b) A pulsion force acting from within the bowel which starts the process of herniation.

A. The Locus Minoris Resistentiae

The weak areas in the muscle coat of the bowel are the gaps caused by the entrance of the blood vessels.

B. The Pulsion Force

Under normal conditions of contraction, a prolonged increased rise in pressure does not occur in the jejunum. But, if there is an irregular contraction of the muscle layers, so that one set of fibres are relaxed, a considerable alteration occurs from the normal both in the local pressure produced inside the lumen and the resistance of the wall to such pressure. In a peristaltic wave, the vascular gaps are narrowed and may be temporarily obliterated in the contracting portion. The wave of peristalsis is preceded by a wave of relaxation. In this situation, the vascular channels gape to their utmost, and constitute a very weak area in the bowel wall. Under normal conditions, however, there is no tendency for the mucous membrane to herniate in these areas, as the contents of the bowel are hurried

along and there can, therefore, be none but a temporary rise of pressure inside the lumen of the bowel. If persistent irregular contraction of the bowel is present, then there may be considerable local increase in pressure. If fixed spasm is occurring in two segments of the intestine, with the intervening segments relaxed, the contents are prevented from passing along the bowel, the pressure in the relaxed portion is increased and the contents are being squeezed against the mucous membrane, which in turn is forced against the muscle coat and tends to push it into any interval between the muscle fibres that it can find. The vascular channels, gaping to their utmost in the relaxed muscle wall, offer to the mucous membrane a locus minoris resistentiae. The nature of the contents of the bowel is an important factor and diverticula are most common in the uppermost part of the small intestine, the duodenum and jejunum, where the contents are less fluid than in the lower part of the small intestine, in which pulsion diverticula are extremely rare. The same is true of the large bowel. As a result of the combination of increased pressure and relaxation of the muscular wall, a wedge of mucous membrane is driven into the gap. After many repititions of this process, a permanent hernia of the mucous membrane will result. This hernia, so formed, will tend to increase in size purely as a result of

pressure from within the gut. It is impossible to estimate how long it takes for a diverticulum to develop. Thus, we see that the ultimate cause of the formation of hernial diverticula lies in abnormal and incoordinated contraction of the muscle coat, and in support of this, the jypertrophic changes in the muscle coat may be recalled anew. Whether this incoordination of muscle action is of neurogenic or myogenic origin, will be discussed in the section dealing with diverticula of the large intestine.

Symptoms

There are no characteristic symptoms. The two outstanding symptoms are:

(a) Vague abdominal discomfort, sometimes pain, particularly after meals;

(b) Flatulence with borborygmus. Occasional occurrence of attacks of vomiting, symptoms of chronic obstruction and bleeding from the bowel may be added.

The flatulence is probably due to retention of bowel contents in the diverticula. The dull ache to the same cause or due to ballooning out of the pouches under tension shortly after partaking of a meal. Colicky pain may be due to intestinal obstruction by a large pouch. As with duodenal diverticula, great caution must be exercised in attributing symptoms to diverticula of

small size before all other possible causes are eliminated.

Diagnosis

The diagnosis of jejunal and ileal diverticula can be made only by x-ray examination and then only when the diverticula are of considerable size or contain a residue of barium after the jejunum has emptied.

Complications

The chief of these are:

(a) Perforation

(b) Acute diverticulitis

(c) Acute obstruction

(d) Volvulus of the jejunum

Perforation is often mistaken for perforated duodenal ulcer.

Acute diverticulitis comes with acute central abdominal pain, rigidity and tenderness of both recti. Laparotomy usually reveals a mass the size of a man's fist towards the centre of the abdomen. This is often mistaken for acute appendicitis.

Rupture of acutely inflamed diverticulum would lead to acute localized or generalized peritonitis.

Acute obstruction is usually due to acute inflammation.

Volvulus of the jejunum may be a result of interlocking of two diverticula.

Treatment

The treatment of choice is resection of the affected portion of the bowel followed by end-to-end or by lateral anastomosis.

the appendix, we find an exception to the general rule that the diagnosis of intestinal diverticula depends upon x-ray examination.

The average age incidence is only forty-two and equally divided between the two sexes.

Morbid Anatomy

Diverticula may be single or multiple. Multiple are more common. They may be associated with acute or with chronic appendicitis. The situation of the diverticula is not regular. Diverticula are most common on the mesenteric border but are found on the anti-mesenteric border. The distal portion of the appendix is more commonly affected. The wall of the diverticula contains no muscle. There is a gap in the muscle coat of the appendix, through which the mucous membrane has herniated to form diverticula. Microscopic examination shows inflammatory fibrosis in the muscle coat where gaps occurred.

Pathogenesis

1. The Relation of the Diverticula to the Muscular Coat

   There are two types of diverticula:

   (a) Hernial pouches of mucous membrane forced through a gap in the muscle coat;

   (b) Distended pockets of the mucous membrane over which the muscle will eventually

atrophy, so that a complete diverticulum, visible from the peritoneal aspect, is formed.

2. Inflammatory Changes in the Appendix Wall

All cases show inflammatory change. There is no retention of faecal material that might predispose to the onset of inflammatory changes such as occur with diverticulosis of the colon. Chronic inflammation has aided and possibly been mainly responsible for, the formation of diverticula in one or more of the following ways:

(a) By causing obstruction to the lumen of the appendix, the obstruction being somewhere in the proximal half of the lumen. The stenosis in all cases is marked but not complete.

(b) Chronic inflammatory changes may act by causing weak areas in the muscular coat, through which may occur herniation of the mucous membrane. The sequence of events is probably this: the abundant lymphoid tissue normally present in the appendix is infective and granulation tissue appears, which invades the submucous coat and finally the muscularis, leaving a weakened scar.

(c) Chronic inflammation may be the cause of persistent spasm in the muscular coat, the

s significance of which is discussed subsequently.

3. The Situation of the Diverticula in Relation to the Circumference of the Appendix Wall

The most favoured site for the development of diverticula is along the concavity of the appendix, along which the vessels of the meso-appendix enter. The next most frequent site is on the opposite margin, that is, at the anti-mesenteric border.

The gaps caused in the musculature by the entry of the blood vessels are similar in anatomical picture to those of the small intestine. The number of final branches arising from the appendicular arteries depends upon the length of the appendix, varying between four and twelve. Immediately before reaching the appendix, each branch divides into two, which pierce the muscle coat, somewhat obliquely, on either side of the mesenteric line. The relation to the entry of vessels of diverticula at the convex margin of the appendix is not so well marked. Before the branches of the appendicular artery enter the concave aspect of the appendix, small branches are given off, which pass on either side around the appendix underneath the serous coat and enter the muscle layer on the convex margin.

4. Changes in the Mucous Membrane affecting the Lumen

The mucosa is thrown into folds due to shortening of the muscular coat. The muscle coat is very much

thicker than in the normal. This increase in thickness may be due to at least one of three causes:

(a) Contraction of the muscle and fixation in contraction;

(b) Hypertrophy;

(c) Inflammatory oedema.

The pouching of the mucous membrane, which may lead to diverticula, is due to shortening of the muscle coat as a result of persistent contraction. Such spastic conditions of the musculature may result from lack of neuro-muscular co-ordination, such as seems to form diverticula in the small and large bowel or may have a more local origin in chronic inflammation of the appendix wall.

Summary

From a study of these observations, it would appear that in not all the cases are the mechanics of production the same and at least two types of diverticula may be distinguished:

1. A hernial protrusion of the mucous membrane through a gap in the musculature, occasioned in most cases by the passage of a blood-vessel and in some as a result of local degeneration of the muscle.
2. Distension of sacculations in the mucosa, with subsequent thinning from atrophy of the muscularis covering them.

The first type may arise purely as a result of partial obstruction to the lumen.

The second type is indirectly due to spasm of the muscularis with tortuosity and migration of the lumen. Subsequently, in these cases, obstructive effects are produced so that the mucous membrane may be forced into gaps in the musculature. This diverticula of the appendix may be due to one of two exciting causes:

(a) Passive distension;

(b) Irregular muscular action.

The predisposing causes are:

(a) The presence of gaps in the muscular coat through which the vessels enter;

(b) Weakening of the muscular coat due to chronic inflammation.

Diagnosis and Treatment

There are no symptoms directly referable to the presence of diverticula, so that it is not possible to diagnose the condition prior to operation. Only rarely do diverticula show up on x-ray examination.

The treatment is appendicectomy.

Unusual forms and Pseudo-Diverticula

Two forms may be distinguished:

(a) Where the narrowing of the lumen occurs close to the tip of the appendix and the distal pocket is somewhat distended and its wall a little thickened. There is no "turning aside".

(b) A mucocele of the appendix.

DIVERTICULA OF THE LARGE INTESTINE

Diverticula of congenital origin are excessively rare. Most of the diverticula are herniations of the mucous membrane through gaps in the muscle coat. They possess little or no muscle in their walls.

The chief source of cases of diverticulosis of the large bowel is derived from a study of x-ray records, the rest from post mortems. Barium enema is the only way to demonstrate diverticula. It has been estimated that between five and seven percent of the general population over forty years of age have diverticula of the large bowel. The condition is slightly more common in women than in men. The average age is fifty-nine years. The average duration of symptoms is seven and a half years.

Morbid Anatomy

A typical uncomplicated acquired diverticulum of the large bowel forms a rounded hemispherical swelling standing out from the peritoneal surface.

Distribution

The diverticula are always multiple and are most common in the sigmoid colon. Rectum is often not affected. The diverticula vary in size and shape in the different parts of the colon. It may be little larger than a pea. In the early stages, it is funnel-shaped,

its mouth being its widest part, but once the muscle coat has been completely penetrated, the pressure within the intestinal lumen distends the body of the sac and transforms it into a globular swelling. It is only when chronic inflammatory changes occur in the wall of the bowel surrounding the mouth of the diverticulum that the latter becomes narrowed. The long axis of the diverticula tends to be almost at right angles to the wall of the intestine. Diverticula of the transverse and ascending colon are usually similar in size and shape to those in the sigmoid but occasionally much larger ones are found in these situations. The points at which the diverticula emerge are governed by two anatomical factors: the arrangement of the longitudinal muscle fibres and the point of entry of the blood vessels through the muscle coat.

1. The Muscle Coat

There are three taeniae coli and between them the longitudinal muscle coat is very thin or entirely absent.

2. The Blood Vessels

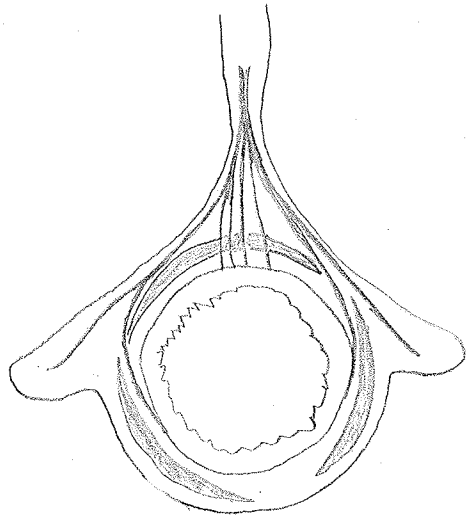
The vascular supply is similar in arrangement to that of the small bowel, in that a series of arcades are formed from which the terminal vessels are given off to supply the gut. Here the similarity ceases. The

arrangement is the same for all parts of the large intestine except the caecum.

Shortly before the terminal vessels from the arterial arcades reach the intestinal wall, they divide into two sets of branches:

(a) Small branches, which continue in the direction of the parent vessels and pass at the intestinal wall in the region of the taenia mesenterica, running at right angles to the bowel wall;

(b) Larger vessels, which pass obliquely to either side of the wall of the gut, running from the mesentery under the peritoneum as far as the mesenteric side of the two remaining muscular tinae. Similar branches given off from the terminal vessel pass into the appendices epiploicae of each side.



Relation of vessels to the wall of the colon.

Origin and Development of Diverticula of the Colon

The sites of election for the formation of diverticula in the sigmoid colon are in two rows immediately to the mesenteric side of the anterior and posterio-lateral taeniae coli. The points at which the diverticula emerge correspond exactly with the point of entry of the vessels, and also correspond with the situations of the appendices epiploicae. Thus, in obese people, the diverticula are completely obscured by fat.

The diverticula are herniations of the mucous membrane of the bowel through a gap in the musculature. The muscular coat of the bowel is continued into the wall of the diverticulum, but gets progressively thinner and is atrophic. The gaps in the muscle coat are caused by the entry of the blood vessels. There is no evidence of generalized atrophy of the muscular wall of the bowel.

The Radiological Aspect

An examination of the colon after a barium meal will nearly always show up diverticulosis, if present, but the more certain method is by use of a barium enema, in which the diverticula are filled out under pressure. It is the best practise to confirm by a barium enema a diagnosis of diverticulosis of the colon made by means of barium meal. The earliest suspicion

of diverticulosis of the bowel from x-ray standpoint is an alteration in the indentations: instead of being placed exactly opposite one another and of uniform depth, they are spaced at irregular intervals. These early changes are due to persistent spasm of the circular fibres. It is really a pre-diverticular state. If the cause of this spastic colon is relieved or disappears, no diverticula will develop.

The next stage is more irregularity of indentations with haustra narrow and wedge-shaped, the apex of the wedge being acute. Final stage is development of small pin head diverticula. This is the "saw edge" stage and each tooth of the "saw edge" is a diverticulum. Next is increase in size and becomes club-shaped. The final stage in diverticulosis is the transformation to the typical flask shape. Delayed emptying occurs after evacuation in all diverticula due to narrowness of communication and to absence of muscle in their walls. Stasis of faecal material within a diverticulum is shown by a thin film around the contents. If only that part of diverticulum next to the bowel is filled, a "new moon" appearance is obtained, the rest being black.

Double contrast enema may be used. After administration and evacuation of barium enema, air is injected slowly into the colon. X-rays taken and double contrast is obtained between the air infiltration and

the barium residue.

The diverticula show more plainly and the chief use of this method is in the diagnosis of inflammatory conditions of the colon.

The x-ray appearances briefly are:

(a) Spasticity of the colon, with irregular indentations between the haustra;

(b) Irregularity in the shape of haustra;

(c) Pin head protrusions of mucous membrane;

(d) The "saw edge" colon, with wedge-shaped diverticula;

(e) Club-shaped diverticula;

(f) Flash-shaped diverticula;

(g) Evidence of faecal retention.

Distribution

Sigmoid is affected in 75 percent of cases. It seems that diverticulosis usually begins in the sigmoid and spreads proximally in most instances. The situations in one hundred and seventy-nine cases reported by Edwards are as follows:

Position	Number
Sigmoid Only	94
Descending colon only	3
Sigmoid and descending colon	38
Sigmoid and descending colon and transverse colon	18
Sigmoid and descending colon and ascending colon	13
Transverse colon only	4
Ascending colon and caecum only	9
Total	179

Pathogenesis

The diverticula are herniations of the mucous membrane and the submucous coat of the colon through the muscular coat. The condition is acquired and not congenital. The herniations are all of pulsion type. Here again two conditions must be present:

(a) Locus minoris resistentiae;

(b) Increased pressure from within the gut.

The areas of least resistance are gaps in the wall of the bowel occasioned by the passage of the blood vessels. There is no evidence to show any congenital or acquired structural abnormality of the bowel wall. The increased pressure is two fold:

(a) The pressure of contents;

(b) The contraction of the muscular wall.

The two are interdependent. The pressure exerted by faecal or gaseous contents is never great and alone does not cause diverticulosis. The contraction of the muscular coat has to be an abnormal one. Diverticula result from regular spasm of the bowel muscles maintained over a long period of time. Thus, we see, the mechanics involved in the formation of hernial diverticula are similar for all intestinal herniae.

Etiology

What causes the irregular behaviour of the intestinal wall?

These may be:

1. The Neuromuscular Tract

   (a) Factors of central origin - such as physical reactions;

   (b) The neuromuscular junction - that is, some abnormality of Auerbach's plexus;

   (c) Stimulation of the plain muscle of the gut - direct stimulation of muscle by the contents of the bowel.

2. Factors not directly associated with the Bowel

Endocrine disturbance, avitaminosis and chemical changes in the blood have been suggested.

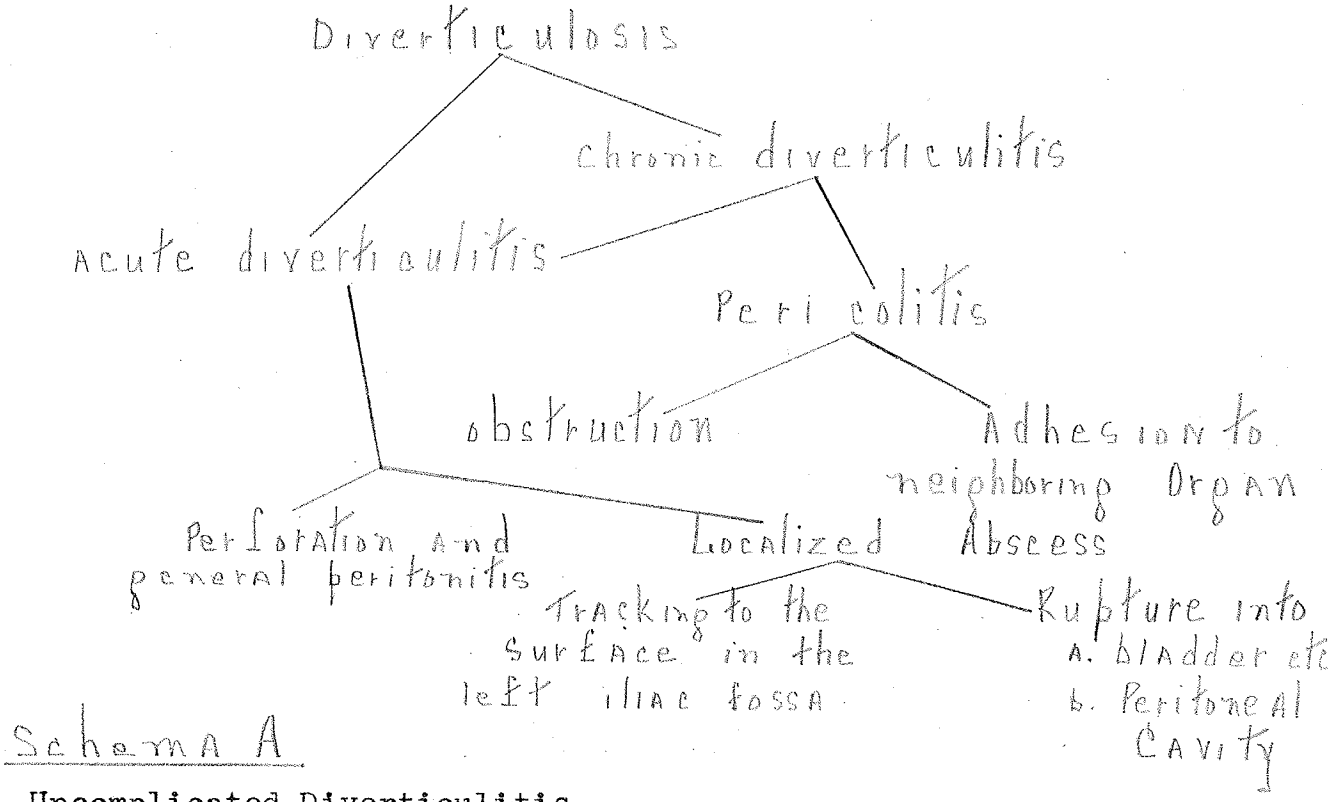
The Clinical Aspect of Diverticulosis

The symptoms of uncomplicated diverticula are those of the disordered action of the bowel which is their exciting cause. Diagnosis is made accurately by x-rays. Pre-diverticular state is to be treated. Constipation, putrefaction, infections and irritations should be eliminated if possible. Constipation is not to be treated blindly by purgatives. Corrections in diet, avoidance of excessive smoking and attention to bowel habit, anti-spasmodics and regulation of whole life are of importance. Colon washouts are only done when symptoms suggest mild infection.

Diverticulitis

Retention of faecal matter leads to inflammatory

changes in the walls of the diverticulum. Once a portion of hard faeces is squeezed into a diverticulum, it cannot readily get out because of the narrow opening and because of the deficiency of muscle in the diverticular wall. Such a faecal concretion may lead to a variety of secondary inflammatory changes as shown below:



Schema A

Uncomplicated Diverticulitis

Included here are chronic diverticulitis with and without mild acute or subacute exacerbations.

Symptoms

1. Pain: it may be a dull ache or a colicky attack, sometimes brought on by defaecation. It is most often felt in left iliac fossa, but also in right iliac fossa, lower abdomen, peri-umbilical, epigastric, pain in back,

and does not always correspond with the situation of the colon. Patient may only complain of rumbling noises, windy spasms, flatulence, soreness and fullness of stomach and felt most during defaecation or when the patient was more than ordinarily constipated.

2. Bowel history: there is constipation but few will have normal bowel movements; others will have constipation and diarrhoea alternating.

3. Blood and mucus in stools.

4. Bladder symptoms: suggest proximity to bladder.

Physical Signs

Obesity is not always present. Colon may be palpable: normal or thickened, tenderness, palpable lump, blood only, blood and mucus, mucus only, occult blood in stools, are other physical signs. In rare cases, a diverticulum can be seen through a sigmoidoscope.

Diagnosis

History, symptoms and physical signs are suggestive but diagnosis is made by x-rays.

Treatment

Diverticulosis and uncomplicated diverticulitis should be treated medically:

(a) Bland diet;

(b) Liquid Petrolatum for constipation;

(c) Colon washouts: using two tubes and six pints of normal saline.

Operative treatment is only indicated when there is onset and persistence of bladder symptoms; onset of symptoms of chronic obstruction and recurrent attacks of inflammation. Operation consists of laparotomy with a view to determine the possibilities of excision and if this is thought possible a preliminary transverse colostomy or caecostomy is done. Operation should be postponed to an interval between the attacks. If the condition is unsuitable for excision, a permanent colostomy is made as near as possible to the inflamed area and left opened for a minimum period of one year.

Diverticulitis with Complications

The complications of diverticulitis are numerous. See Schema A

1. Acute Diverticulitis (Non-Perforating)

Inflammatory reaction may be severe but it varies greatly. There is no way of telling between those in whom resolution is certain and those in whom danger of peritonitis or perforation appears imminent. One must judge by the severity of the general symptoms and the local signs. In diagnosis, pain is rarely central as it is in appendicitis but is usually in the line of the colon. Pain is never so intense as in appendicular colic or in ureteric colic. The patient is never doubled up with pain. Leucocytosis, fever, tenderness, usually left side, are physical signs.

Treatment

Treatment is to bring the inflamed area to the surface of the abdomen and a colostomy performed above it. Later, when acute inflammatory reaction has subsided, resection of that part of the colon with end-to-end anastomosis is done.

2. Acute Diverticulitis with Perforation and Peritonitis

This is rare. Perforation is a culminating point of severe attack of diverticulitis. It is possible for the onset of perforation to be sudden, and may follow some strain. Distention and generalized peritonitis follow and may be rapidly fatal in spite of operation. Other cases will be mild.

Treatment

The treatment is immediate operation through lower left rectus incision. After the abdomen is opened - if perforated diverticulum is lying free, it should be excised, the hole in the bowel closed by purse string and covered over by omentum. If the perforation is too edematous, omentum should be sutured over it. If perforation cannot be found, exteriorize the inflamed loop of bowel. In all cases, do colostomy proximally. Caecostomy is usually not enough. Abdomen is drained, leaving two grams of Sulfanilimide or 100,000 units of Penicillin in the peritoneal cavity. The usual supportive post-operative treatment is heat, blood

transfusion if necessary; Penicillin, ecetera are used if necessary. Prognosis is usually not good.

3. Abscess

This is perhaps the commonest complication. There is attack of acute diverticulitis with fever and not infrequently diarrhoea. Abscess may rupture into general peritoneal cavity, into vagina, bladder, small bowel or rectum. Treatment is to drain the abscess as soon as possible. Colostomy, as a rule, is not necessary. A fistula may result and persist for a long time.

4. Obstruction

This is usually due to stenosing peri-diverticulitis. It is very difficult to distinguish it from annular carcinoma. The obstruction is usually of very slow onset and is rarely complete.

It is treated by caecostomy followed by resection of affected loop of bowel, with end-to-end anastomosis. Chronic obstruction of small bowel can occur with ileum becoming adherent to an area of diverticulitis of the colon.

5. Colovesical Fistula

This is a serious complication. The characteristic symptom of the condition is pneumaturia and passage of faecal material per urethra. In all cases there is a

previous attack of acute diverticulitis, followed sooner or later by pyuria, frequency, pneumaturia and faeces in the urine. There is acute cystitis and ascending infection from the bladder to the kidneys. Cystoscopy will reveal the fistula.

Diagnosis is not hard. X-ray is of help and diagnosis is confirmed by cystoscopy.

Treatment

Colostomy alone is not satisfactory. There is discomfort of colostomy and no guarantee of cure of bladder symptoms. Separation of bladder and large bowel and suturing both, all in one stage, is usually not a permanent cure. Radical closure of both visci following colostomy is a difficult and often unsuccessful operation. Transverse colostomy is done. Distal bowel is washed out regularly. Three or four months later, the fistula is resected and the sigmoid and bladder closed. Omentum is sewn between the sutured openings in the bladder and colon. After twelve months colostomy is closed.

In rare cases of diverticulitis of the caecum, resection of the right colon and ileo-transverse colostomy is the correct procedure.

6. Diverticula of the Colon associated with New Growth

The new growth is usually carcinoma but a polyp has been seen. New growth is associated in five to six

percent of all cases of diverticula of the colon. The association is a coincidence. Diverticulitis is not a predisposing condition to malignancy except in so far that cancer is probably more liable to originate in tissues which are the seat of chronic inflammation than in normal tissues.

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MRS. F.T. - Age 55 years

Admitted to Winnipeg General Hospital on May 12th, 1936.

Her entrance complaints were:

Abdominal pain - 2 attacks.
Vomiting with attacks.
Vaginal discharge for 2 years.

On April 7th, 1936 at 8:30 p.m. had an attack of severe, crampy, lower abdominal pain. She vomited several times. There was no warning of the impending attack. There was no change in bowels or urinary bladder. The attack lasted for three hours and after that remained sore. Was brought to the hospital next morning and heat was applied. She was discharged same day, feeling much better.

The second attack occurred on May 8th, 1936, identical with the first one and subsiding the next day. She was admitted four days' later for investigation. The rest of the history was negative except for constipation.

Physical Examination; revealed no abnormal findings except varicose veins of both legs. Partial stenosis of bowel was suspected. Barium series were done; May 20th, 1936. This was reported as negative except for numerous diverticula of the sigmoid colon.

Barium enema was done with the same findings.

Patient was discharged May 26th, 1936.