

Dissertation

On

ACUTE INTESTINAL OBSTRUCTION

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MADRAS MEDICAL COLLEGE &
GOVERNMENT GENERAL HOSPITAL
CHENNAI – 600 003.**

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CERTIFICATE

This is to certify that “**ACUTE INTESTINAL OBSTRUCTION**” is a bonafide work done by **Dr. KATHIRVEL.C** M.S., post graduate student, Department of General Surgery, Government General Hospital, Chennai - 3 under my guidance and supervision in fulfillment of regulations of The Tamilnadu Dr. M.G.R. Medical University for the award of M.S. Degree Branch II, (General Surgery) during the academic period from March 2003 to February 2006.

Prof. P.S. VENKATESWARAN
M.S., M.CH.,

Professor and Head
Department of General Surgery
Government General Hospital
Chennai 600 003.

Prof. RAJKUMAR WILLIAMS
M.S.,

Chief Surgical Unit V
Department of General Surgery
Government General Hospital
Chennai 600 003.

Prof. KALAVATHI PONNIRAIIVAN M.D.,
The Dean
Government General Hospital
Chennai 600 003.

INTRODUCTION

Acute intestinal obstruction is one of the main surgical emergency problems which a general surgeon has to face every day irrespective of day & night. It is quite an exciting experience to examine, investigate, diagnose, explore and look in to the abdominal cavity where it would reveal the puzzling conditions. A surgeon adds this experience to his knowledge every day from each and every case.

Acute intestinal obstruction can result from a variety of causes. Success in the treatment of acute intestinal obstruction depends largely upon early diagnosis, skillful management and the appreciation of the importance of treating the pathological effects of the obstruction just as much as the cause itself.

The abdomen is said to be a magic box and so long as its lids remains unopened heaven alone knows what lies within it. But every attempt should be made to arrive at a provisional diagnosis with the available investigatory modalities before embarking on surgery. The rapid onset and progress of the clinical features and the spread with which the morbidity set in endangers the patients as well as the relatives. In spite of the recent advances still this condition holds a major share of mortality due to so many practical factors which provided the scope to study and analyse this interesting subject.

Since life to death is an one way traffic, it is not harm to open an acute abdomen in doubt rather than to wait and worry later for our act. The acronym

is better to open and see, rather than wait and see. This dissertation is undertaken because early diagnosis and early interference is of immense value in preventing morbidity and mortality in cases of acute abdominal emergencies.

AIM OF THE STUDY

To Analyse ,60 cases of acute intestinal obstruction and their presentation.

- To identify the causes of acute intestinal obstruction.
- To identify the factors modifying the prognosis of the patient.
- To study about the morbidity and mortality rate in the analysed cases.

HISTORICAL HIGHLIGHTS AND PIONEERS IN TREATMENT

The historical aspects of acute intestinal obstruction is very interesting if it is known by its evolution.

- Praxagoras incised strangulated loop and established artificial anus in 3rd BC.
- Hippocrates and Celsus (460BC) used the pattern of Egyptian treatment which was the administration of purgatives and enema once in a month to clear the bowels as a prophylactic measure . Other methods practiced were.

1. Percutaneous puncture in distended colon.

2. Cupping the abdomen.

- Ambrose Pare (1510 – 1590)
 - A French physician who identified the bowel obstruction first time and reported a patient who died of twisted bowel.
- Marinus Sanctus gave many pounds of metallic mercury to pass through the obstruction by weight - 16th century .
- Franco in 1561 divided the constriction ring by surgical intervention.

- Mery in 1701 excised gangrenous bowel and made colostomy.
- Littre 1713 suggested the possibility of proximal decompression.
- 1776 Pillare successfully made a caecostomy for a case of cancer rectum.
- 1793 Duct performed first successful sigmoid colostomy .
- 1836 Diffexi Back resected the small bowel & anastomosed .He had a major role in the study of suturing the bowel.
- 1839 Amussat found that a colostomy could be made in the left lumbar region for ca rectum.
- 1800 Lancrances sutured the traumatic wounds of the colon .
- 1813 Tracers Lambert- Famous for his lamberts suture.
- 1833 Lissrang and Reynard did the resection and anastomosis for cancer rectum.
- 1846 – 1892 Paul & Black proved that exteriorisation of the Colon is more useful than primary anastomosis.
- 1892 Murphy introduced button method of anastomosis 1908 Paul and Mickulickz advocated exteriorisation procedure.

- Praeger and Keep 1908 introduced the aseptic anastomotic principle.
- Homes Hurler 1908 - Surgical stapling was developed by him in Austria and the modifications performed by Vonpetz and Friedrich of Ulm (1934).
- Ein Horn (1910) & Wangenstein (1913) introduced the Levin's duodenal tubes.
- Abbot & Miller 1934 - Introduced the long intestinal tubes.
- Lantor 1946 & Graffon Smith 1952 – Familiarise the Ryles tube as gastric suction tube.
- Cliebble & Hipsley 1921 - Hydrostatic reduction of intussusception.

REVIEW OF LITERATURE

Classification of Intestinal Obstruction

According to causes

- Dynamic or mechanical
- Adynamic or paralytic ileus

Mechanical Obst - may be

- Extra luminal - Simple
- Intramural - Strangulating
- Intraluminal

Special varieties of strangulation

- Intussusceptions
- Volvulus - Simple
- Compound
- Closed loop obstruction

According to clinical presentation

- Acute
- Chronic
- Acute on chronic obstruction

Pathophysiology

Each day the GIT secretes and reabsorbs 8-12 litres of intestinal, gastric, pancreatic and biliary secretion .

Food & drink - 2000ml

Salivary gland -1000ml

Gastric juice -1500ml

Pancreatic juice - 1500ml

Bile - 500ml

Intestines -2000ml

Most of the fluid and electrolytes load (90%) is absorbed in the small intestine and about 1.5lt reach the caecum. The colon normally absorbs the water & sodium and secretes potassium ,so that stool water is about 200ml.

The normal adult man has mean intestinal gas volume of about 100ml and excretes a variable amount ranging from 300 – 2000ml daily.

The composition which is highly variable contains

$N_2 = 24 - 80\%$ $CH_4 = 0 - 26\%$

$O_2 = 0.1-2.5\%$

$H_2 = 0.6 - 50\%$

$C O_2 = 5-29\%$

With mechanical obstruction intestinal fluid accumulates and the intestine distends. Its volume increases in the square of radius. Thus the volume of a intestine of 2 cm diameter approximates to 300ml. This will increase to 1300ml if the same segment dilates to a diameter of 4 cm.

Major disruption of orderly balance of secretion and absorption begins with intestinal obstruction.

Two phases were recognized

1. In early intestinal obstruction. (<12hrs) water and electrolyte accumulates in the lumen because of decrease in absorption.
2. By 24hrs second phase begins; intraluminal water and electrolyte accumulates rapidly.

In simple mechanical intestinal obstruction the nondistended proximal intestine is spared initially and the net increase in intestinal volume is minimal because intraluminal fluid is dispersed and absorbed. As obstruction persists and the proximal intestine dilates absorption decreases and the secretion increases. This isoosmolar fluid tends to cause an isoosmolar volume contraction which is further aggravated by vomiting producing sequestration of fluid in the third space.

Motility of the intestine

Early in the course of intestinal obstruction small intestinal contractile activity appears to increase in frequency and intensity. This causes a crampy

abdominal pain The intestines contract vigorously to propel the intestinal contents distally past the obstruction with sustained increase in intraluminal pressure, contractile activity gradually decreases and ends. After a period of quiescence (Fatigue) this cycle recur.

Later in the course of obstruction as the proximal intestine dilates progressively, it becomes sluggish. Contractions become sluggish and absent producing absence of bowel sounds (silent abdomen).

Blood Flow

Intestinal blood flow is related inversely to intraluminal pressure. When intraluminal pressure increases capillary blood supply decreases and blood flow interrupted in smaller calibre vessels. These alterations of mucosal Blood flow and overall total blood flow are especially pertinent to closed loop obstruction in which when great intraluminal pressure are attained.

Bacteriology

In the absence of obstruction, jejunum and proximal ileum of the small intestine contains less number & more of gram + facultative organism($<10^4$ /ml).In contrast in the distal ileum, coliform (gram negative)and anaerobes bacteroides species dominate (10^5 - 10^8 /gm). Colonic bacterial count is about 10^9 - 10^{12} /gm.

With the establishment of intestinal obstruction,the microflora of the small intestine changes dramatically not only type of organism but also in absolute numbers of organisms proximal to the obstruction.

Coliforms multiply profusely, reaching concentration of 10^9 - 10^{10} /ml consisting mostly of Escherichia coli, Bacteroides fragilis, Streptococcus fecalis, Klebsiella spp., Proteus spp., Pseudomonas spp. and clostridium spp. The fecal flora proliferates in direct proportion to the duration of the obstruction and to the extent of intestinal distension.

Distal to the obstruction intestine retains less number of bacteriae. In any case with established intestinal obstruction, preoperative antibiotic prophylaxis is indicated and the peritoneal spillage of intestinal content should be avoided during surgery.

Systemic Effects Of Intestinal Obstruction

The effects of intestinal obstruction related to site, extent and duration of the obstruction. The common denominator in the systems respond to intestinal

obstruction ,involves the isotonic contraction, dehydration that accompanies sequestration of extra cellular fluid in the intestinal and peritoneal compartment.

Cardiovascular effects manifest as tachycardia & hypotension secondary to cardiac irritability due to catecholamines and hypovolemia. Respiratory compromise occurs with severe abdominal distension and secondary to aspiration of vomitus. Acute renal failure or renal shut down may occur.

When strangulated obstruction supervenes the fore-mentioned systemic effects are magnified and hemorrhage occur both into the intestinal wall and in to the lumen. Intestinal infection may precipitate metabolic acidosis and sepsis with associated vascular collapse due to transmigration of gram negative bacteriae and systemic inflammatory response syndrome.

Clinical presentation

Cardinal symptoms of intestinal obstruction are nausea & vomiting, colicky abdominal pain, constipation or obstipation and abdominal distension.

Pain

Crampy abdominal pain and distension are absent when the proximal small intestine is able to decompress in a retrograde manner into the stomach, with more distal obstruction pain is episodic, crampy, often diffuse, poorly localized and lasting for 1-3min. Between spasms, pain resolves completely.

With ileal obstruction, the quiescent interval between spasm is 1-3min, in more distal obstruction 10-15 min may separates the episodes of pain. These episodes of colic occur simultaneously with borborygmi and the patient doubles up with pain. This is in contrast to peritonitis in which patient lie still.

The onset of constipation is a late development indeed the patient may continue to have bowel movement and to pass feces as the distal bowel empties.

Vomiting

In early stages of obstruction, the vomiting contains undigested food particles. With time it becomes obvious when the obstruction becomes complete and the intestine is dilated. If the vomiting turns feculent that indicates late & established intestinal obstruction.

With closed loop obstruction or with incarceration of intestine in a hernial orifice, a relentless vomiting may be the initial symptom. This represents an abdominal reflex related to acute unrelieved intestinal obstruction and strangulation. In large bowel obstruction vomiting will be a late feature.

Distension

Distension is minimal or absent in upper small bowel obstruction and mesenteric vascular occlusion. It is delayed in colonic obstruction. Severe distension usually occurs in low small bowel obstruction.

Constipation

In large bowel obstruction constipation will be an earlier feature. Obstipation occurs in the very late stage.

Pyrexia

Fever may signify

1. Onset of strangulation
2. Intestinal perforation
3. Presence of inflammation due to IL – 1, 6, 8 - associated with obstructive disease, abscess due to diverticulitis or localized perforation of an obstructed bowel, colonic cancer and inflammatory bowel disease.
4. Hypothermia has sinister significance and may be due to severe hyponatraemia and end stages of septic shock.

Abdominal tenderness

Localised tenderness in the abdomen or an external hernia indicates strangulation with overt infarction and /or perforation. The development of rigidity with rebound tenderness signifies onset of peritonitis.

Water ; Salt depletion and hematological changes

Water and salt depletion result in increased haematocrit, loss of skin turgor, dry tongue, poor capillary refilling & sunken eyes there is a rise in serum amylase and leucocytosis.

Percussion

Percussion reveals tenderness with resonant note in cases of dilated intestines. Free fluid in the abdomen detected.

Auscultation

Reveals obstructed bowel sounds with abnormal borborygmi and rushes that coincides with abdominal colic. With late and unrelieved obstruction bowel sounds will be absent.

Digital Rectal Examination

Distal growth may be palpable. In cases of proximal obstruction rectum will be empty, dilated and roomy. Sometimes intussusception may be felt per rectally.

Radiological studies

The accuracy of diagnosis of intestinal obstruction by roentgenography is 55-80%.

Gas shadow

When the jejunum, ileum or the colon distended with the gas each of its characteristic appearance allows it to be distinguished radiologically. the diameter of the viscus is no criterion as to whether it is small or large bowel. Obstructed small intestine is revealed by relatively straight segments that generally lie more or less transversely. Obstructed large intestine is diagnosed by its haustration, distended

caecum by rounded gas shadow in the right iliac fossa. Jejunum characterized by its valvulae conniventes that arise from antimesenteric to mesenteric border spaced regularly giving rise to concertina effect.

Ileum – the distal ileum is characterless.

Large intestine

Except caecum, large intestines shows haustral folds. Haustral folds unlike valvulae conniventes are spaced irregularly and are not placed opposite to one another. Caecal diameter > 6cm precludes impending perforation.

Fluid levels

Infants under the age of 2 yrs few fluid levels in small intestine are normal. In adults 2 inconstant fluid levels, are taken as physiological. One is at the duodenal cap and the other is in the terminal ileum. In intestinal obstruction, it takes a little time for the gas to separate from the fluid. So fluid levels appears later than gas shadows. When paralysis of intestines has occurred fluid levels become more conspicuous and more in numbers and by the time fluid levels are more pronounced obstruction is advanced. The number of fluids level proportionate to the degree of obstruction and to its site obstruction, low in the colon does not commonly give rise to fluid levels in small intestine but in cases of long standing obstruction this may occur due to incompetency of ileocaecal valve.

No radiographic criteria reliably and consistently indicates strangulation. Fixation of bowel, loop, thickens of volvulae conniventes, increasing intraluminal fluid, low gastric emptying, reduced bowel activity and intraperitoneal fluid accumulation strongly suggest impending devitalisation and merit close follow up examination, clinical correlation. In the acutely obstructed patient bowel wall gas with or without mesenteric portal vein gas or pneumoperitoneum give clear cut evidence that strangulation has taken place.

Gall stone impaction may be evident in the radiography and gas in the biliary tree indicate that bilio enteric fistula has occurred.

Contrast studies

Antigrade Approach

Contrast given through NG tube inspissation and impaction does not occur because of dilution of the contrast agent in the obstructed intestine. Water soluble contrast studies can be useful in suspected perforation cases.

Retrograde approach

Preferred in patients with a presumed distal small bowel obstruction, for suspected colonic obstruction or in acutely ill patients for whom emergency diagnosis is important .

Diagnostic Colonoscopy

Considered in patients suspected of

1. Non strangulated sigmoid volvulus.
2. Distal colonic strictures and colonic growths.
3. Chronically ill patients with caecal dilation suggestive of pseudo obstruction.
 - a. In each of these situation colonoscopy offer therapeutic as well as diagnostic benefits.

The finding of a corkscrew tapered luminal narrowing at colonoscopy suggest a volvulus and often derotation using endoscopy is possible. Endoscopic procedures also have been developed to stenting of obstructed bowel for palliation or for preparation of bowel for surgery. Also it is useful in colonic stricture cases for dilatation and for taking biopsy in cases of colonic growths.

CT of the Abdomen

CECT is useful in patient with subacute obstructive symptoms suspected of having malignancy and is also useful in patient with early postoperative obstruction of the large bowel in those patients associated with abdominal mass or clinical sings of infarction.

Finding from CT scan includes

1. Bowel wall thickening.

2. Soft issue edema associated with inflammation or infection.
3. Intramural or extra intestinal gas.
4. Abnormal fluid collection.
5. Abnormalities of retroperitoneum.
6. Any growth arising from the bowel.

Management

Supportive Management

Nasogastric suction by Ryles tube, intra venous fluid management with NS or RL. The amount needed varies from patient to patient and is influenced by clinical findings, biochemical and hematological parameters.

1. IV line with wide bore cannula and 50 percent crystalloids and 50 percent colloids are used.
2. Gastric decompression with Ryles tube
3. Urinary catheter to monitor the urine output.
4. Blood sugar, Urea, creatinine, electrolyte, Hb% and grouping should be done.
5. Hourly abdominal girth, pulse rate, temp, respiratory rate & BP monitored.
6. Xray chest, Abdomen erect & supine views.
7. Broad spectrum antibiotics covering gram positive, gram negative aerobes and anaerobes.

Operative management

Type of surgery is according to the cause of the obstruction. Early diagnosis and intervention is a must to prevent future complication and hence timing of surgery is very important feature.

Strangulating obstruction

Vascular supply to a segment of the intestine is compromised.

Pathophysiology

Strangulation may be due to extraneous compression of the mesenteric arcade (by an adhesive band or hernial orifice).

Most often the venous outflow obstruction starts first.

Less commonly local pressure necrosis occurs in cases of an obstructive adhesive band or by a hernial orifice.

Venous obstruction results in vascular engorgement, edema and local venous hypertension which leads arterial compromise that starts tissue hypoxia. Capillary integrity lost which causes intramural haemorrhage and stasis that lead to secondary vascular thrombosis and further anoxia. The increased intraluminal pressure with compromised vascular supply leads to mucosal infarction and necrosis. Trans mural migration of gram negative bacteriae into the systemic circulation leads to complications.

Diagnosis

Patient present in extreme shock, hypothermia, acidosis and diffuse peritonitis.

Phosphate level ↑ in serum, urine and peritoneal fluid.

CRP, LDH , Amylase and Alkaline Phosphatase levels increase.

Intra Operative differentiation between viable and nonviable intestine.

Functional intestinal obstruction (ileus)

Ileus occurs due to failure in enteric nervous system.

Management

The incidence of this condition greatly reduced by routine NG aspiration and withholding timely by mouth after surgery until normal bowel sounds and/or passage of flatus returns.

1. The primary cause must be removed.
2. Normal bowel activity will return if offending factor is relieved.
3. Close attention to maintain the circulatory blood volume and correction of both fluid & electrolyte imbalance.
4. Surgical treatment is necessary if the ileus is secondary to a life threatening disorder.

Specific small intestinal obstruction

I. Hernia

Inguinal and femoral hernias account for 80% of cases though the proportion of femoral and umbilical hernias in which strangulation is greater than that of inguinal hernia. The constricting agent being in umbilical hernia is the fascial defect in the abdominal wall.

Obstructed and strangulated hernias require emergency surgery. The constricting ring is divided, non viable bowel requires resection and anastomosis, hernial defect repaired.

II. Adhesions - congenital and acquired

1. Adynamic areas – variations of anatomy
2. Foreign bodies – Talc, Starch
3. Inflammatory – chronic disease
4. Radiation enteritis

Prevention

1. Washing the peritoneal cavity.
2. Avoidance of excessive packing gauze.
3. Covering of anastomosis and raw peritoneal surface by greater omentum.
4. Leaving raw peritoneal areas unsutured.

Treatment

1. Adhesiolysis (division of bands).
2. Strictureplasty.
3. Resection and anastomosis of the bowel if it is non viable.

Intussusception

Telescoping of a segment of intestine into an adjacent one. Most common in children. In adult it may be due to polyp, Meckels diverticulum or submucosal lipoma.

It may be ileoileal, ileocecal, ileocolic or colocolic.

Treatment

Manual reduction of the bowel, If not viable resection and anastomosis.

Volvulus

It is a twist or rotation of a loop of intestine about its mesenteric attachment. It is therefore a sudden obstruction of closed loop variety if the rotation is complete, ischaemia or total vascular occlusion may occur.

Operation is the untwisting of the loop and the causative band should be divided.

Internal hernia

A portion of the small intestine passes into one of the retroperitoneal fossa or into a congenital defect in the mesentery (Stammer's hernia – hernia through the mesocolic defect).

Treatment - Division of the constriction.

Stricture

Usually due to TB, Crohn's disease and ischemic stricture.

Tuberculosis

- Ulcerative & Hyperplastic
- Cocooning in tuberculous peritonitis.

Ulcerative

Multiple transverse ulcer in the terminal ileum, overlying serosa is thickened, reddened and covered with tubercles. Strictures which are the cause of intestinal obstructions.

Hyperplastic

Occurs usually in the ileocecal region(ileocecal mass) and the regional lymphnodal involvement occurs.

Treatment

1. Strictureplasty for supple strictures.
2. Dense wide strictures and multiple strictures in a short segment of bowel – Resection and anastomosis.
3. Limited ileocecal resection (limited Right Hemicolectomy).

Miscellaneous

Obstruction may be due to worms, bolus of food (tricho bezoar, phyto bezoar), foreign body and gall stone.

Embolism and Thrombosis of SMA

Possible sources are from left atrium in cases of atrial fibrillation, mural MI, atheromas, aneurysms and atrial myxoma.

Primary thrombosis due to athero or arteriosclerosis.

Treatment

- Embolectomy in early cases.
- If bowel viability is in question resection and anastomosis.
- Vascular grafting can also be done.

Sigmoid colon volvulus

Volvulus is defined as twisting of a hollow viscus organ either in longitudinal axis or its mesenteric attachment.

Predisposing factors

1. Narrow attachment of the mesocolon.
2. Long pelvic mesocolon
3. Overloaded colon – providing the twisting force to the lumen of the bowel. The dietary and bowel habits of the person plays a major role in its causation.
4. Adhesions.

Clinical diagnosis

The triad of abdominal pain, distension and constipation are the predominant signs and symptoms.

- The duration of the symptoms are short (1956 Bolt).

- In old age, present as acute large bowel obstruction with.
 - Distended flanks
 - Visible large bowel loops
 - Empty and ballooned rectum on digital rectal examination
 - Frimann-dhal sign on X-ray
- It is difficult to differentiate between viable and gangrenous bowel in volvulus clinically. Understanding this is very important because one of the methods of management of this is conservative approach and obviously this cannot be undertaken if gangrene has already set in.
- Clinical features that suggest the presence of gangrene are severe pain, deterioration in general condition of the patient with tachycardia, hypotension, marked abdominal tenderness and absent bowel sounds.

DIAGNOSIS

A plain Abdomen X-ray erect film is most useful in establishing the diagnosis. The significant radiological features include

1. Inverted 'U' sign
2. Liver overlap sign : Haustral margins overlapping the lower border of liver shadow.
3. Left flank overlap sign : Haustral margins overlap the dilated descending colon.

4. Frimann-Dahl sign : The two limbs of the loop converge inferiorly giving rise to 3 white lines representing the outer walls and the two adjacent inner walls. It is usually on the left side of the pelvis.
5. Huge amount of air accumulates in the loop giving an air fluid ratio > 2:1.

Bursel and Bakes conducted a study and concluded the following are the most significant findings.

1. Apex of the loop ↓ (L) Hemidiaphragm
2. Inferior convergent to left
3. Left flank overlap sign

The least specific signs are

1. Distended sigmoid loop
2. Air fluid ratio > 2 : 1

This is because a similar picture seen in

1. Distended but non twisted sigmoid colon
2. Pseudo volvulus : Distended transverse colon looping down.

Treatment

1. When there are no signs of gangrene or peritonitis, resuscitation followed by endoscopic derotation is the initial management.

Derotation easily accomplished by using a rigid proctoscope but a flexible sigmoidoscope or colonoscope might also be effective.

2. A rectal tube may be inserted to maintain decompression.
3. The risk of recurrence is high with this kind of management.
4. For this reason elective colectomy has to be done after stabilizing the patient with bowel preparation.
5. Clinical signs of gangrene or perforation mandates emergency surgery. Similarly presence of necrotic mucosa, ulcer and dark blood noted on endoscopy suggest strangulation and is an indication for operation.
6. If bowel is non viable 3 options of surgery
 - a. Resection and primary anastomosis
 - b. Paul Mickulicz exteriorisation.
 - c. Hartmann's procedure

Caecal Volvulus

This is more common in female individuals. Clock wise twist commonly occurs. Patients will present with abdominal pain, distension, constipation and vomiting. Plain X-ray abdomen erect will show bird's beak appearance. Barium enema also diagnose the condition.

Treatment

1. If the bowel is viable – Caecopexy after untwisting the volvulus.

2. If the bowel is non viable - Right hemicolectomy.

Compound volvulus

This is otherwise called as ileosigmoid knotting. Commonly this condition present as acute intestinal obstruction. Most of the time bowel is not viable hence resection and anastomosis of the ileum and Hartmann's procedure for sigmoid colon has to be carried out.

Malignant Obstruction

In the west up 90% of patients suffer obstruction secondary to carcinoma. But the converse is not true. Only 15% of large bowel malignancies present with obstruction. The risk of obstruction by a colorectal malignancy varies with the site of malignancy.

In decreasing order of risk.

1. Splenic flexure – up to one half go in for obstruction.
2. The rest of the colon except rectum – 1/5th risk
3. Rectal carcinoma – 1/10th risk.

However, because of the unequal percentage distribution of malignancy in different parts, distal colonic malignancy, being the most common. In clinical practice approximately 1/4th of all malignant large bowel obstruction cases are situated in the left colon i.e. at or distal to the splenic flexure.

DIAGNOSIS

Based on

1. **Clinical fracture**

Abdominal pain, distension, constipation, alternating bowel habits and vomiting, Abdomen pain is present in 90% of cases.

2. **Plain abdominal X-ray**

Gaseous dilatation of the large bowel proximal to the site of obstruction and a distal cut off. Caecal ballooning due to distal colonic growths.

3. **Thin contrast enema**

It demonstrates the site of obstruction.

MANAGEMENT

The aim in the management of patients with malignant obstruction is to relieve the obstruction with low mortality and morbidity to ensure adequate clearance where possible to ensure long term survival, but also provide good palliation in the remainder. It is generally accepted that for obstruction proximal to splenic flexure, resection and primary anastomosis is optimal therapy. An internal bypass is justified in patients with an irresectable tumours and in high risk patients with extensive distant spread of disease.

The controversy arise in the more common more distal lesions. Most surgeons have been reluctant to ignore the traditional wisdom that it is unwise

to anastomose dilated, edematous, unprepared bowel, so the therapy is initially to decompress the bowel. At a second operation obstruction is resected and anastomosis done. Presently it is considered staged surgery may result in poor long term prognosis, that has led on to increased performance of primary resection and anastomosis following intra operative colonic wash outs.

1) Decompression And Delayed Resection

Decompression can be achieved by

- a) Blow hole ostomy or
- b) A full fledged laparotomy and ostomy construction.

Disadvantages

1. The procedure by itself is followed by a mortality risk of around 16% partly because only high risk patients are chosen for this procedures. Mortality is usually secondary to CVS complication.
2. Once created, the decompression may not be adequate in 5%, hence a future specific procedure may still carry a high mortality.
3. The stoma itself suffer problems of retraction, prolapse, necrosis etc.
4. More than half the number of patients initially intended for a future corrective procedure, but never made it either due to poor general condition or disease progression or poor compliance for the next surgical procedure.
5. The overall hospital stay (> 40 days) is economically draining.

Other methods of decompression.

a. Laser lumanisation

b. Transmural stents

II RESECTION AND DELAYED ANASTOMOSIS

The tumour is resected and the proximal bowel is brought to the surface as end colostomy, while the distal stump is closed (Hartmann's procedure).

Disadvantages

1. Morbidity and mortality secondary to intra abdominal sepsis.
2. The stoma may necrose and retraction occurs in up to 20% of patients.
3. Reversal of the procedure is a major task due to adhesions.
4. Disease progression and decreased long term prognosis exist.

III RESECTION AND PRIMARY ANASTOMOSIS

The ideal management of malignant obstruction is to remove the tumor and restore bowel continuity in one sitting. However segmental resection of left sided lesion involves anastomosis in unprepared, dilated and edematous bowel which would give mortality rate of up to 50% from anastomotic leakage.

To minimize these risks peroperative bowel preparation is employed.

1. On-Table lavage

A large foley catheter is introduced into the cecum either through the appendix stump or through the terminal ileum across the ileocecal valve. Warm saline is run in an antegrade fashion and feces removed distally. This method is useful if fecal load is a viscous fluid.

2. Simple decompression of flatus and extrusion of solid feces is better than lavage if the load is solid as the lavage would make the feces fluid and difficult to manage.

Using the above procedures, resection and primary anastomosis is found to have morbidity and mortality rates atleast equal to that of other procedures.

Advantages

1. Stoma and its associated problems avoided.
2. Decreased hospital stay.

Added proximal decompression

Proximal decompression not known to decrease the incidence of dehiscence.

Subtotal colectomy

Advantages

1. The entire unprepared bowel is removed.
2. Ileocolic anastomosis has lesser leak rate than colocolic anastomosis.

3. Obstructing carcinoma has increased risk of synchronous malignancy in proximal bowel multiple colonic polyposis.

Disadvantage

1. Diarrhoea is more common
2. Colonic nutrition function is lost

Indication

1. > 50 year of age
2. Positive family history
3. Obstructing carcinoma
4. Caecal perforation due to back pressure.

MATERIALS AND METHODS

The study was conducted in Madras Medical College Government General Hospital during the period of 2004 August 2005 August. The study included 60 patients taken at random. The cases included those patients who were diagnosed preoperatively as acute intestinal obstruction by clinical, biochemical and radiological parameters. The details of the patient included their name, age, sex, IP, peroperative diagnosis, operative procedure and any complications.

On admission the patients diagnosed as acute intestinal obstruction were resuscitated with nasogastric aspiration, intra venous fluids, Preop, perop and postop antibiotics. Those patients with postoperative infection, appropriate antibiotics changed after pus culture and sensitivity test.

OBSERVATIONS AND RESULTS

Total number of cases analysed as acute intestinal obstruction during the period 2004 August to August 2005 taken at random- 60

Small intestine – 51

Large intestine – 9

ETIOLOGY

1. Obstructed inguinal hernia – 23
2. Adhesive intestinal obstructed – 10
3. Incisional hernia – 4
4. Umbilical hernia – 4
5. Ileocecal TB – 1
6. Femoral hernia – 1
7. Ileal volvulus – 1
8. Intussusception – 1
9. Ca caecum & ascending colon – 1
10. Ca descending colon – 2
11. Ca Transverse colon – nil
12. Ca sigmoid – 2
13. Ca rectum – 1
14. Sigmoid Volvulus – 2
15. Ileal knotting – nil
16. Stricture ileum – 1
17. Meckels diverticulitis – 1
18. Internal hernia - nil
19. Ischaemic enteritis - 2
20. Adynamic ileus - 4

SEX INCIDENCE

Males – 49

Females – 11

Ratio ; 4.5 : 1

AGE INCIDENCE

Age	No.of Cases
12-20	1
21-30	9
31-40	10
41-50	18
51-60	14
61-70	4
71-80	4
> 80	Nil

INCIDENCE OF STRANGULATION

Total no. of cases – 15

* 25%

Causes	No. of cases
1. Strangulated hernia	7
2. Adhesive Intestinal Obstruction	3
3. Incisional hernia obstructed	2
4. Ischaemic enteritis	2
5. Intussusception	1

Mortality – 11.6%

No. of cases – 7

Morbidity – 41.7%

No. of cases – 25

DISCUSSION

- In this study, 60 cases of acute intestinal obstruction taken at random were analysed. Small intestinal involvement is in 51 cases and large intestine in 9 cases.
- Obstructed inguinal hernia was the most common cause of acute intestinal obstruction which is 38.3% of cases.
- Adhesive intestinal obstruction was the second most common cause which is 16.7% of cases (the most common cause of intestinal obstruction in world wide).
- Males are affected more commonly than females.
Ratio is 4.5 : 1
- Commonest age group in which intestinal obstruction occurred is 41 – 50 yrs.
- Mortality was 11.6% and was associated more commonly in older age groups and who presented with gangrenous bowel.

- Morbidity was 41.7%. Wound infection was the main cause of morbidity and was associated with patients who presented with peritonitis, older age groups and strangulation.

SUMMARY AND CONCLUSION

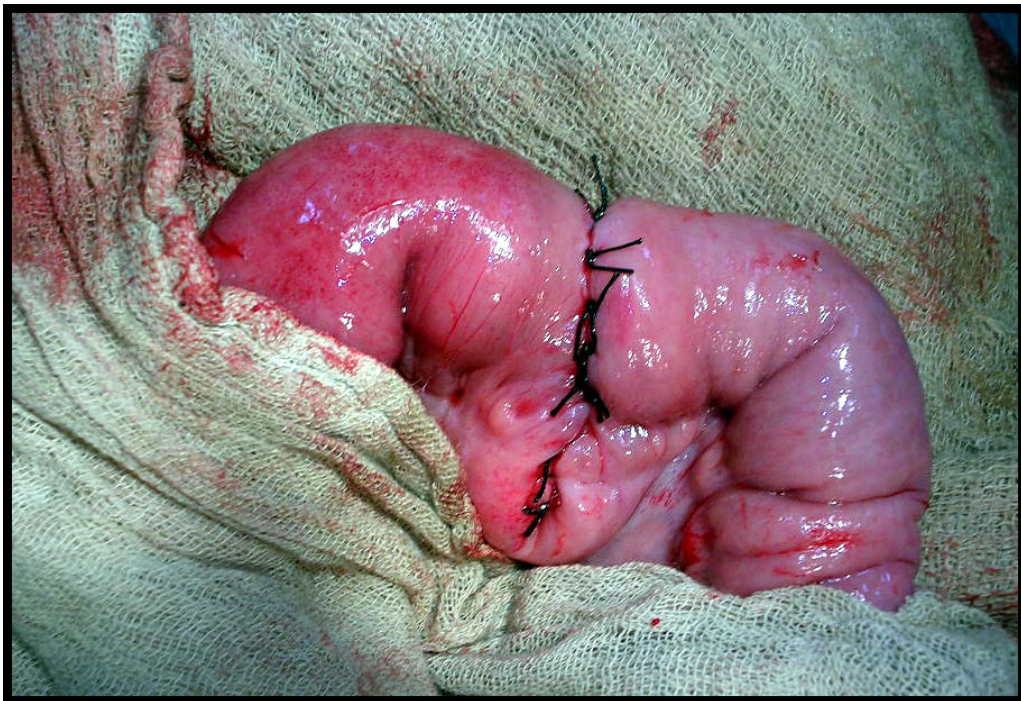
- Incidence of involvement of small intestine in Acute intestinal obstruction is 85% and large intestine 15%.
- Most common cause of small intestinal involvement in Acute Intestinal obstruction is obstructed inguinal hernia followed by Adhesive intestinal obstruction (the most common cause of intestinal obstruction in world wide).
- Males are affected more commonly than females.
- As age increases risk of strangulation increase.
- Strangulation was associated with increased mortality and morbidity.
- Gangrene bowel was diagnosed mostly by clinical features
- By this study, that has been carried out, our conclusion is intestinal obstruction due to hernia were more common in our population than the western population where post operative adhesive obstruction is the commonest cause. May be because of illiteracy, ignorance and delaying of the surgery for the hernia is the reason for, hernia being the commonest cause of intestinal obstruction in our population.



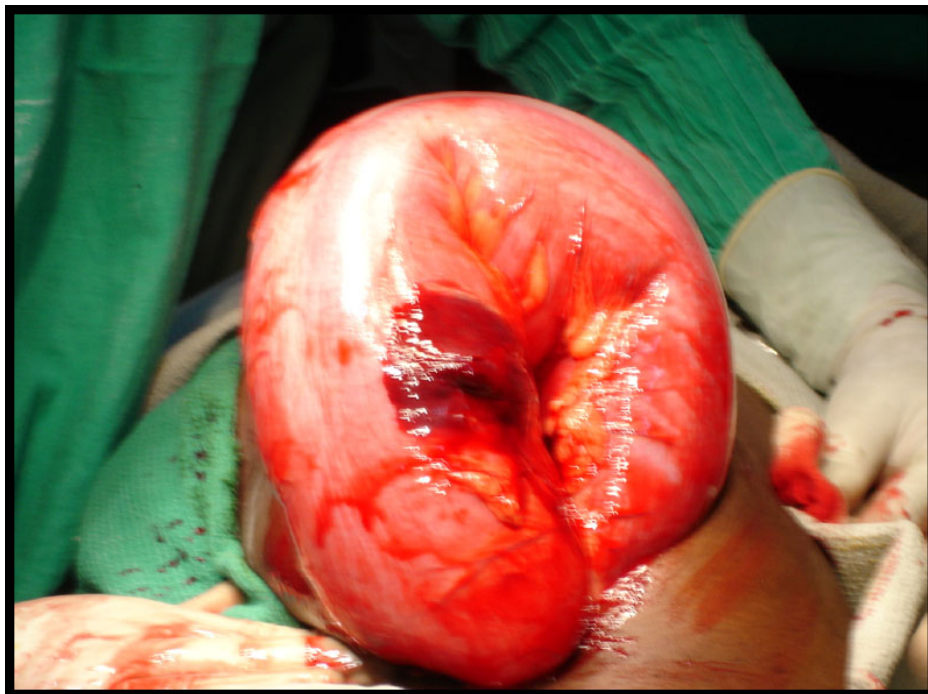
INTUSSUSCEPTED MECKLES DIVERTICULUM



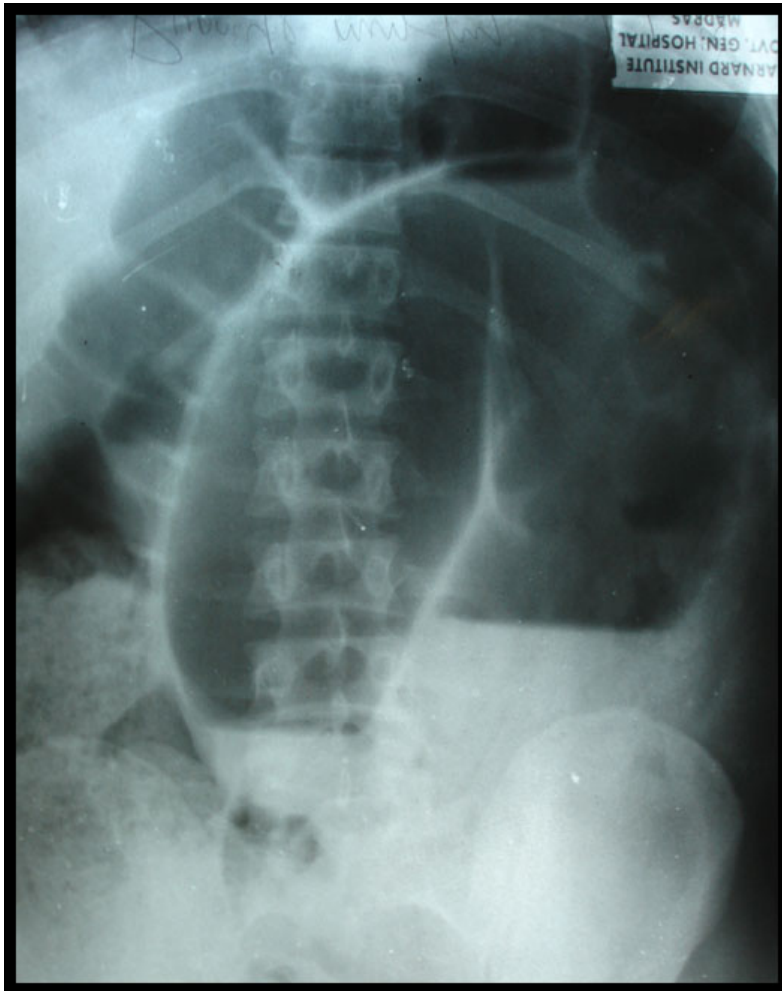
MECKLE'S DIVERTICULITIS



**MECKLE'S DIVERTICULITIS
ILEAL ANASTOMOSIS**



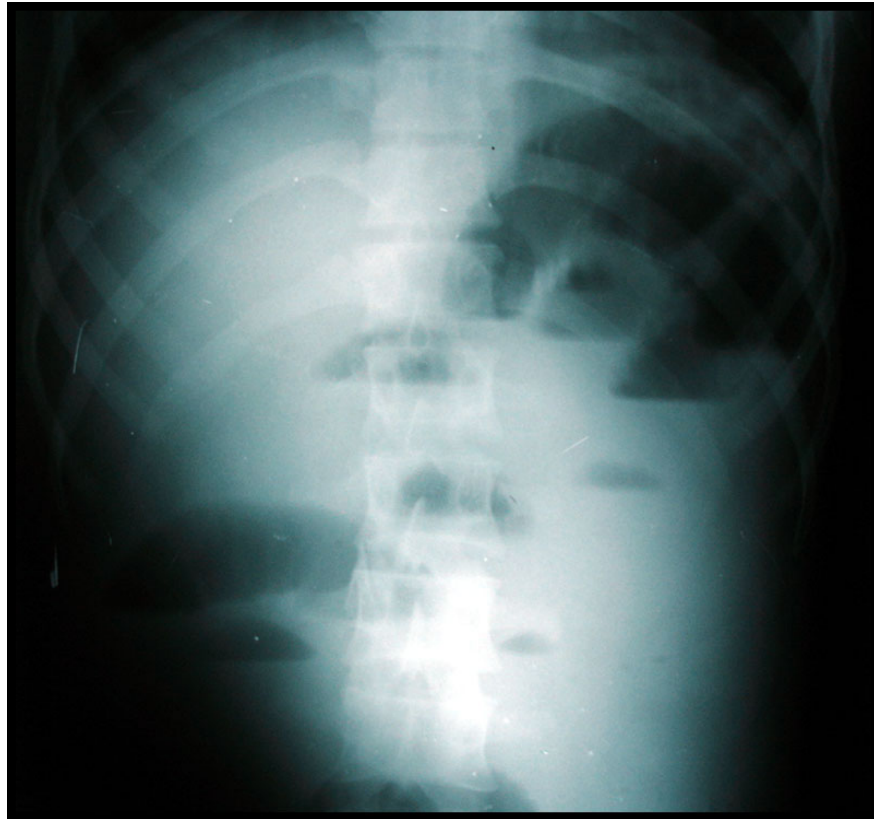
SIGMOID VOLVULUS – LAPAROTOMY



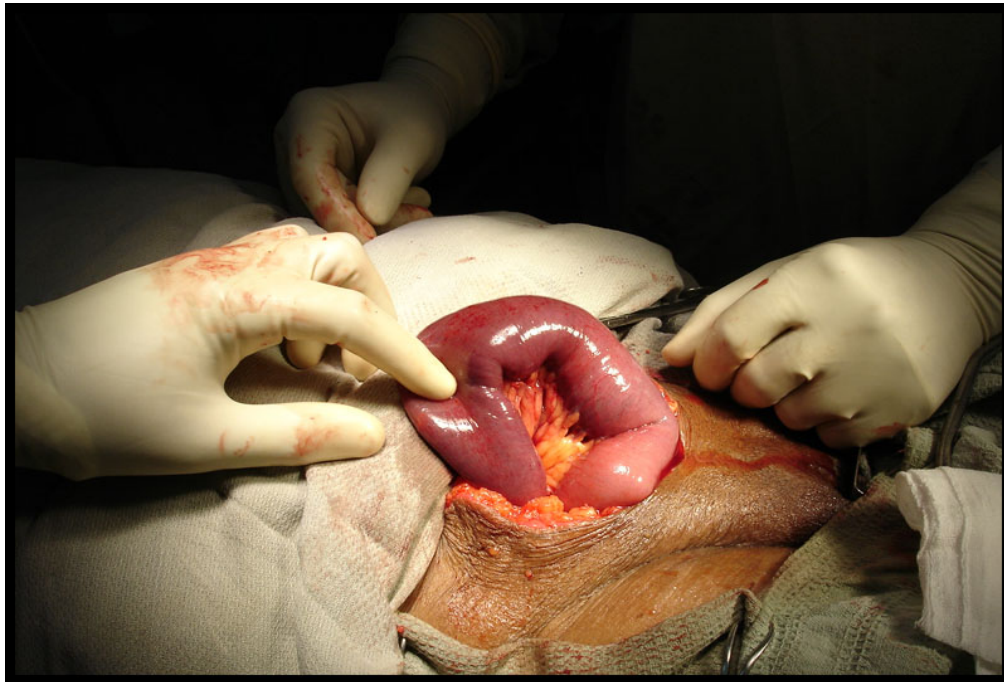
SIGMOID VOLVULUS



OBSTRUCTED RIGHT INGUINAL HERNIA



AXR ERECT - MULTIPLE AIR FLUID LEVELS



**OBSTRUCTED INGUINAL HERNIA -
ILEAL LOOP OBSTRUCTED**

AETIOLOGY

Lesions intrinsic to the intestinal wall	Lesions extrinsic to the intestinal wall
<p>A. Congenital</p> <ol style="list-style-type: none"> a. Malrotation b. Meckel's diverticulum 	<p>A. Adhesions</p> <ol style="list-style-type: none"> 1. Post operative 2. Congenital 3. Post inflammatory
<p>B. Inflammatory</p> <ol style="list-style-type: none"> 1. Infections <ol style="list-style-type: none"> a. Tuberculosis b. Actinomycosis 2. Crohn's disease 	<p>B. Hernia</p> <ol style="list-style-type: none"> 1. Abdominal wall (external) including inguinal, femoral, umbilical, epigastric, lumbar, intestinal, obturator, sciatic and perineal. 2. Intra abdominal (Internal) 3. Post operative <ol style="list-style-type: none"> a. Incisional b. Parastomal c. Internal hernia of mesenteric defect
<p>C. Neo plastic</p> <ol style="list-style-type: none"> 1. Primary neoplasms 2. Metastatic neoplasms 	<p>C. Congenital</p> <ol style="list-style-type: none"> 1. Annular pancreas 2. Volvulus 3. Persistence of yolk sac 4. Perineal encapsulation
<p>D. Miscellaneous</p> <ol style="list-style-type: none"> a. Intussusception b. Endometriosis c. Radiation enteropathy / stricture d. Post ischaemic stricture e. Stricture due to potassium tablets or phenylbutazone etc. 	<p>D. Neoplastic</p> <ol style="list-style-type: none"> 1. Carcinomatosis 2. Extraintestinal neoplasm 3. Soft tissue (retroperitoneal, mesenteric etc.)
<p>Intraluminal causes</p>	<p>E. Inflammatory</p> <ol style="list-style-type: none"> 1. Intra abdominal abscess 2. Starch peritonitis 3. Splenosis
<p>A. Gall stone ileus</p> <p>B. Enterolith</p> <p>C. Bezoar</p> <p>D. Foreign body</p> <p>E. Parasites</p>	<p>F. Miscellaneous</p> <ol style="list-style-type: none"> 1. Intra abdominal abscess 2. Starch peritonitis 3. Splenosis

CAUSES OF ADYNAMIC OBSTRUCTION

1.	Reflux Inhibition of Intestinal Motility due to increased sympathetic discharge with hyperpolarisation of smooth muscle cells which becomes non responsive to both neural and hormonal stimulation. a. Abdominal operations b. Pneumonia c. Crush injuries d. Fracture spine e. Retroperitoneal haemorrhage or exudates f. Hyperextension of spine (plaster jacket) etc.
2.	Metabolic abnormalities a. Hypokalemia b. Ureaemia c. Various ketoacidosis d. Hyponatremia
3.	Intra peritoneal sepsis due to both reflux inhibition of intestinal motility and direct effect of bacterial toxins on the myenteric nerve plexuses.
4.	Mesenteric vascular disease a. Arterial embolism b. Arterial thrombosis c. Venous thrombosis d. Various vasculitis e. Low flow state due to decreased cardiac output and reflex mesenteric vasoconstriction (non occlusive vascular insufficiency)
5.	Drugs Antipsychotics (Tricyclic antidepressants), Hexamethonium bromide etc.

DIFFERENTIATION BETWEEN VIABLE AND NONVIABLE INTESTINE

Intestine	Viable	Non viable
Circulation	Dark colour becomes lighter; mesentery bleeds if pricked.	Dark colour remains ; No bleeding if mesentery is pricked.
Peritoneum	Shiny	Dull and Lusterless
Musculature	Firm pressure rings may or may not disappear	Flabby, thin and friable. pressure rings persist.
Peristalsis	May be present	No peristalsis.

DIAGNOSIS OF LEVEL OF OBSTRUCTION

Level of Obstruction	Onset	Pain	Dehydration	Distention	Radiographic finding
1. High small Bowel	Sudden	Mostly upper abdominal; variable	Extreme	Absent	May show gasless abdomen or distention of duodenum r proximal jejunal loop.
2. Low small bowel	More gradual	Central abdominal severe colicky	Less marked	Moderate; central in position	small loops of small bowel in supine film as lying transversely ; Fluid levels in erect film.
3. Large bowel	Usually insidious	Central or lower abdomen ; Colicky or generalized discomfort due to	Slight	Progressive; Extreme in late stage peripheral except volvulus	Gas seen in the colon mainly proximal to the obstruction which is not evacuated even after

		distension			enema
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APPROXIMATE ELECTROLYTE CONCENTRATIONS OF GIT²⁷

Site	Na⁺	K⁺	Cl⁻	HCO³⁻
Saliva	50	15	30	40
Stomach	55	15	115	10
Bile	145	5.2	90	35
Pancreas	141	4.6	77	90
Jejunum	120	5.0	100	–
Ileum	117	5.0	106	25
Caecum	75	–	–	50

**MECHANICAL – INTESTINAL OBSTRUCTION
CLASSIFICATION
ETIOLOGY OF MECHANICAL – INTESTINAL OBSTRUCTION
IN ADULTS**

LESIONS INTRINSIC TO THE INTESTINAL WALL	LESIONS EXTRINSIC TO THE INTESTINAL WALL CONTINUED.
<p>A. Congenital</p> <ol style="list-style-type: none"> 1. Malrotation 2. Meckel's diverticulum 	<p>2. Intra-abdominal (internal)</p> <ol style="list-style-type: none"> a. Congenital, including paraduodenal, foramen of Winslow, diaphragmatic, mesenteric defect, paracecal, intersigmoid, broad ligament.
<p>B. Inflammatory</p> <ol style="list-style-type: none"> 1. Infections <ol style="list-style-type: none"> a. Tuberculosis b. Diverticulitis 2. Crohn's disease 3. Eosinophilic granuloma 	<p>3. Postoperative</p> <ol style="list-style-type: none"> a. Incisional b. Parastomal c. Wound dehiscence d. Internal hernia of mesenteric defects <p>4. Acquired</p>
<p>C. Neoplastic</p> <ol style="list-style-type: none"> 1. Primary neoplasma <ol style="list-style-type: none"> a. Benign b. Malignant 2. Metastatic neoplasms 	<p>C. Congenital</p> <ol style="list-style-type: none"> 1. Annular pancreas 2. Volvulus 3. Persistence of yolk sac 4. Peritoneal encapsulation
<p>D. Traumatic</p> <ol style="list-style-type: none"> 1. Hematoma 2. Ischemic stricture 	<p>D. Neoplastic</p> <ol style="list-style-type: none"> 1. Carcinomatosis 2. Extraintestinal neoplasm 3. Soft tissue recurrence (eg. Retroperitoneal, mesenteric)
<p>E. Miscellaneous</p> <ol style="list-style-type: none"> 1. Intussusception 2. Endometriosis 3. Radiation stricture 4. Retroperitoneal fibrosis 5. Postischemic stricture 6. Stricture due to potassium tablets or phenylbutazone 7. Intramural hematoma in patients on oral 	<p>E. Inflammatory</p> <ol style="list-style-type: none"> 1. Intra-abdominal abscess 2. Starch peritonitis 3. Splenosis <p>F. Miscellaneous</p> <ol style="list-style-type: none"> 1. Superior mesenteric artery syndrome (Wilky's syndrome). 2. Cocoon related to peritoneovenous shunts

anticoagulants	3. Sclerosing peritonitis due to practolol
LESIONS EXTRINIC TO THE INTESTINAL WALL A. Adhesions Postoperative (commonest) Congenital Bands Postinflammatory B. Hernia Abdominal wall (external), including inguinal, femoral, umbilical, ventral, epigastric, lumbar, interstitial, obturator, sciatic, speigilean and perineal	INTRA LUMINAL OBSTRUCTION A. Gallstone illus B. Enterolith C. Bezoar D. Foreign body E. Balloons of intestinal tubes F. Parasites including Ascaris and tapewarm G. Cholestyramine H. Intraluminal diverticulum

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