PUBLIC HEALTH MINISTRY OF UKRAINE
KHARKIV NATIONAL MEDICAL UNIVERSITY

SURGERY

Part I

EMERGENCY SURGERY OF THE ABDOMINAL CAVITY

Textbook for medical students

ХІРУРГІЯ

Частина І

НЕВІДКЛАДНА ХІРУРГІЯ ЧЕРЕВНОЇ ПОРОЖНИНИ

Навчальний посібник для студентів медичних закладів

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S94 **Surgery. Part 1. Emergency surgery of the abdominal cavity**: textbook for medical students / I.A. Kryvoruchko, V. M. Lisovyi, V.V. Boyko, O. A. Tonkoglas. – Kharkiv : KhNMU, 2017. – 194 p.

The textbook is completed in accordance with the student's study program. It subsequently existing and new data on the etiology, pathogenesis, diagnosis, comprehensive treatment of surgical diseases of the abdominal cavity and prevention of postoperative complications.

For the 4th year students of higher medical institutions which study by English. The textbook will be to useful students of other courses.

UDK 617

Хірургія. Частина 1. **Невідкладна хірургія черевної порожнини**: навч.
S94 посібник для студентів медичних закладів / І. А. Криворучко, В. М. Лісовий, В. В. Бойко, О.А. Тонкоглас. – Харків: ХНМУ, 2017. – 194 с.

Навчальний посібник доповнено відповідно до програми навчання студентів. У ньому послідовно відображаються існуючі та нові дані щодо етіології, патогенезу, діагностики, комплексного лікування хірургічних захворювань органів черевної порожнини та профілактики післяопераційних ускладнень.

Для студентів 4-х курсів вищих медичних закладів, що навчаються англійською. Посібник буде корисним також студентам інших курсів.

УДК 617

CONTENTS

PREFACE

The higher school takes the leading place in system of education. It is connected with economy, science, technology and culture of a society. Therefore its development is the important component of strategy of the general national development.

In XXI century, it is necessary precisely to represent, what should be the higher professional education and the specialists who study in the higher school now and in the future.

We have different opinion about XX century, but all progress of century associated with technical progress.

Due to quick specialization the modern surgery has high achievements in various areas such as – cardiovascular surgery, surgical endocrinology, surgery of the digestive channel, surgery of the liver, biliary tract and pancreas, purulent-septic surgery. The development of mini-invasive surgery recently began and it includes: endoscopic diagnostics and operative interventions, radiography endovascular interventions under the control of angiography, especially actively developing is puncture interventions under the control of ultrasonic and a computer tomography, etc. These and many other directions; have cardinally changed approaches in diagnostics and treatment of many surgical diseases.

Studying of surgical diseases in the Ukrainian medical higher school and Kharkiv National Medical University is carried out at the surgery departments. One of the main tasks of departments is the systematized teaching including anatomical and physiological features of organs, modern representations about etiology, pathogenesis, diagnostics, clinical features and complex treatment, and also preventive maintenance of postoperative complications. These questions, in our opinion, are one of the cores for the preparation of the doctor of the general practice.

In the textbook presents the description of the basic diseases and their treatment is given.

Professor V. M. Lisovyi

Chapter 1

ACUTE APPENDICITIS

Acute appendicitis is the most common acute surgical disease of the abdomen. It affects 67 % of the population. The mortality is about 0,2 % – 0,3 % and depends on complications of acute appendicitis. Appendicitis is defined as an inflammation of the inner lining of the vermiform appendix that spreads to its other parts. Appendectomy for appendicitis is the most common performed emergency operation in the world.

HISTORICAL

In 1886, Reginald H. Fitz, a pathologist, made the decisive connection between right iliac fosse inflammation and appendiceal perforation, and coined the term appendicitis (Fig. 1.1). After a review of 466 cases, Fitz openly challenged Dupuytren's ideas: "As a circumscribed peritonitis is simply one event, although usually the most important, in the history of inflammation of the appendix, it seems preferable to use the term appendicitis to express the primary condition". Fitz further noted the importance of early recognition of the perforating appendix and the urgent demand of treatment, according to surgical principles. In 1889, McBurney described the clinical features of acute appendicitis and the point of maximal abdominal tenderness. The gridiron incision commonly attributed to McBurney was actually devised by McArthur.

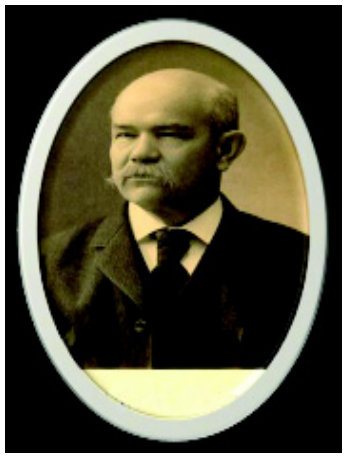


Figure 1.1. *Reginald H. Fitz*

1.1. ANATOMY

Appendix a direct continuation of the cecum. It is located at the confluence of three longitudinal strips (teny) (Fig. 1.2). It has: base, body, and the tip. Its length at an average of 5–10 cm, but may vary from 0.5 to 30 cm or more.

From without inwards the structure of appendix is as follows: 1) serous coat is composed of peritoneal coat, which covers the whole of the appendix except along the narrow line of attachment of the mesoappendix; 2) muscle coat consists of outer longitudinal muscles and inner circular muscles as seen in the case of small intestine; 3) submucosa: the submucous coat of the appendix is very rich in lymphoid tissue. It

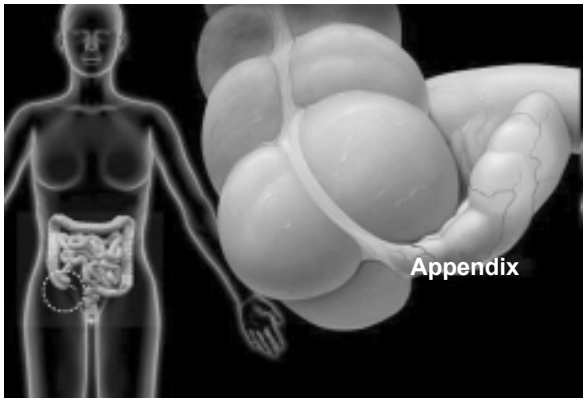


Figure 1.2. *Vermiform appendix*

contains lymphoid follicles, which are known as "abdominal tonsil". The number of submucosal lymphoid follicles are few at birth. This number gradually increases to a peak of approximately 200 follicles between the ages of 12 and 20. After that the number is gradually reduced and reaches to about half at the age of 50 years and almost absence of lymphoid tissue at the age of 60 years; 4) the mucous coat resembles that of large intestine.

The appendicular artery is the main arterial supply to the appendix and the appendicular artery is an end artery. It is a branch of the lower division of ileocolic artery and passes behind the terminal ileum to enter the mesoappendix a short distance from the base of the appendix. The mesoappendix often does not continue up to the tip of the appendix. In this case the artery lies in direct contact with the tip of the appendix. Inflammation of the appendix will cause thrombosis of the artery, which precipitates gangrene of the tip of the appendix and ultimate perforation. Accessory appendicular artery supplies the base of the appendix and this artery should be properly ligated otherwise haemorrhage will continue after appendectomy. This is a branch of the posterior cecal artery (Fig. 1.3).

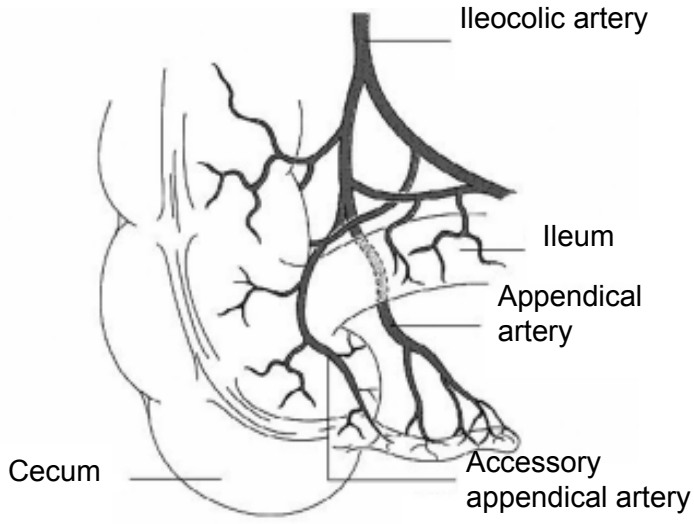


Figure 1.3. *The appendicular artery*

The appendicular vein, which follows the appendicular artery along the free border of the mesoappendix, drains into the ileocolic vein, which is a tributary of the inferior mesenteric vein of the portal venous system. Inflammatory thrombus may cause suppurative pylephlebilis in case of a gangrenous appendicitis.

Lymphatic vessels draining the appendix travel along the mesoappendix to drain into the ileocecal lymph nodes.

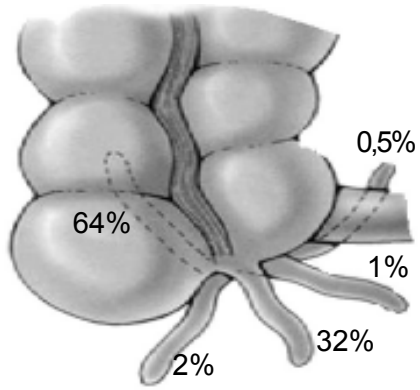


Figure 1.4. *Anatomical positions*

If the cecum does not migrate during development to its normal posi-tion in the right lower quadrant of the abdomen. Various anatomical positions (Fig. 1.4). Appendix / cecum relationship: the relation of the base of the appendix to the cecum is constant, while the tip can be found: 1) in the right iliac fossa; 2) pelvic location; 3) medial location; 4) retrocecal; 5) under the terminal segment of the ileum; 6) lateral; 7) subhepatic; 8) left-hand (with situs viscerum inversus).

The vermiform appendix plays a useful role in the defence mechanism of the body.

1. The lymphoid follicles present in the appendix for maturation of B-lymphocytes.

2. The appendix participates in the secretory immune mechanism in the gut. Appendix forms an integral part of the "gut associated lymphoid tissues" (GALT) and forms globulin for immune mechanism. Yet appendix is not indispensable in this regard and removal of the appendix produces no detectable defect in the functioning of the immunoglobulin system.

1.2. ETIOLOGY AND PATHOGENESIS

To explain the mechanisms of inflammation in the appendix proposed many theories, of which the main ones are **mechanical, infectious and neurovascular.**

Acute appendicitis results from obstruction followed by in infections. Such an increase is related to continuous secretion of fluids and mucus from the mucosa.

Intestinal bacteria within the appendix multiply, leading to the recruitment of white blood cells and the formation of pus. This causes further increase in the intraluminal pressure leading to venous outflow obstruction. This in turn causes ischemia of the appendiceal wall, resulting in loss of epithelial integrity and bacterial invasion. The bacteriology flora, customarily found in acute appendicitis, is a mixed colonic flora with both aerobic and anaerobic organisms. Most frequently seen organisms are E. coli, enterococci, bacteroides (gramnegative rod), nonhaemolytic streptococci, anaerobic streptococci and etc.

Thrombosis of the appendicular artery and veins rapidly leads to perforation and gangrene of the appendix. As this process continues, a periappendicular abscess develops. This may be localized, if walled off by the omentum. Alternatively, a generalized peritonitis may result.

1.3. CLASSIFICATION

IN OUR CLINIC WE USE KOLESOV'S CLASSIFICATION (1972):

1. Appendicular colic.
2. Acute simple appendicitis.
3. Acute destructive appendicitis: a) phlegmon; b) gangrenous; c) perforated.

Complications: appendicular mass; appendicular abscess; peritonitis; pylephlebitis; sepsis.

1.4. PATHOLOGY

FOUR TYPES:

Acute catharal (simple) appendicitis. By acute catarrhal appendicitis is understood that variety of acute inflammation of the appendix in which the pathological alterations are wholly or almost wholly confined to the mucous membrane, the other coats of the organ presenting but little or no deviation from the normal. In this connection the term catarrhal is employed strictly within its pathological significance. Catarrhal appendicitis – hyperemia, edema, lymphatic hyperplasia, leukocytes'

infiltration of the mucosa and submucosa with endoluminal seroleukocytic exudate. If appendiceal canalization is impeded, the exudate expands the organ, thereby constituting a hydrops of the appendix.

Acute phlegmonous (purulent) appendicitis. Phlegmonous appen-dicitis, in keeping with the definition of phlegmon, presents extended compromission of all layers of the organ, with numerous abscesses in the wall and abundant fibrinopurulent exudate on the serosa. Inflammation also involves adjacent structures: the cecum, the last ileal loop and the contiguous parietal peritoneum. As such, it constitutes a fibrinopurulent peritonitis limited to the right iliac fossa. This circumscribed peritonitis repre-sents the anatomic basisof the socalled ileocecal plate.

Acute gangrenous appendicitis. Gangrenous appendicitis – since, as we've said, the appendicular artery is a terminal structure, any thrombotic event will naturally lead to total or partial necrosis of the organ.

Acute perforated appendicitis. Thrombosis may arise in the most serious forms of obstructive appendicitis, and is promoted by the necrotizing action of anaerobic germs or also by the presence of an endoluminal foreign object. The event evolves into the breakdown of the wall, perforation, consequently, peritonitis.

1.5. CLINICAL FEATURES

1. Complains:

Pain in the right iliac region (constant; moderate; without irradiation). The disease begins suddenly, amid well-being, without a prodromal period. The most constant symptom is a pain in the abdomen, which, as a rule, has a permanent character.

Usually anorexia.

Nausea.

Vomiting. Most patients vomit only once or twice. Vomiting is usually not persistent. Vomiting appears after the onset of pain.

2. Objective sings of disease:

Subferile fever (before 38°C). In case of generalised peritonitis following rupture of appendicitis temperature may shoot up to 40°C.

Pulse rate. The pulse rate is usually normal or slightly elevated. High pulse rate should question the diagnosis. Pulse rate increases in proportion with the temperature of the patient. In case of generalized peritonitis following rupture pulse rate may rise up to 100 per minute.

Right lower quadrant pain on palpation (the single most important sign).

Peritoneal signs.

Localized tenderness to percussion.

Other confirmatory peritoneal signs (absence of these signs does not exclude. appendicitis).

Triad Delofua (classical triad of acute appendicitis): 1) spontaneous pain in the right iliac region; 2) muscle tension right iliac region on palpation of the abdomen; 3) hypersensitivity of the skin right iliac region.

3. Pathognomonic signs:

Kocher's sign: epigastric pain or wandering pain throughout the abdomen, which shifts to the right iliac region in 2–4 hours.

Kummel's sign: pain originated in the umbilical region and only after 2–4 hours move to the right iliac region.

Rovsing's sign: palpation of the left lover quadrant by the left hend with simultaneous pushes of descending part of the colon with right hend. Sympton is positive when the patient feels pain in the right ileal region (Fig. 1.5).

Razdolsky's sign: with superficial palpation, it is possible to identify the zone of hyperesthesia in the right iliac region at the McBurney's point (Fig. 1.6).

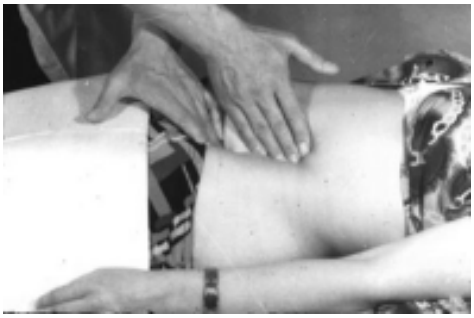


Figure 1.5. *Rovsing's sign*

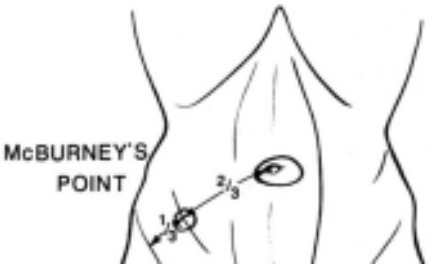


Figure 1.6. *McBurney's point*

Obturator test (Koup's 1st): passive internal rotation of flexed right thigh with the patient in supine position will elicit pain. This positive obturator sign is diagnostic of pelvic appendicitis (Fig. 1.7).

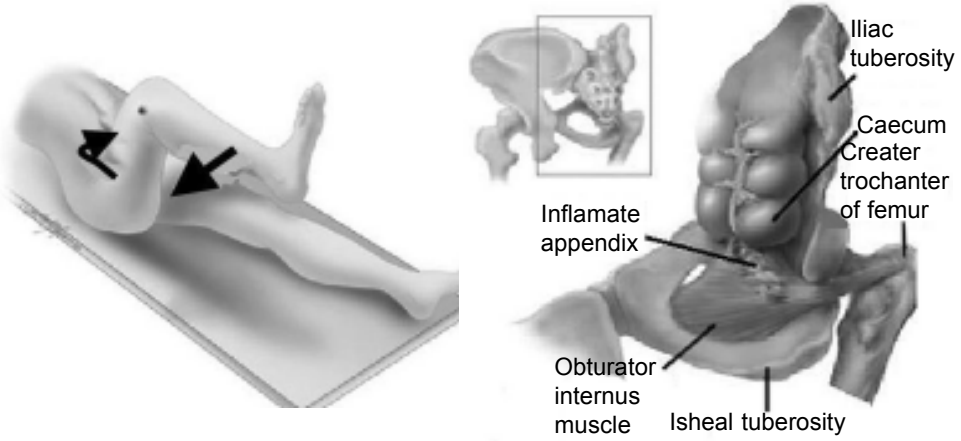


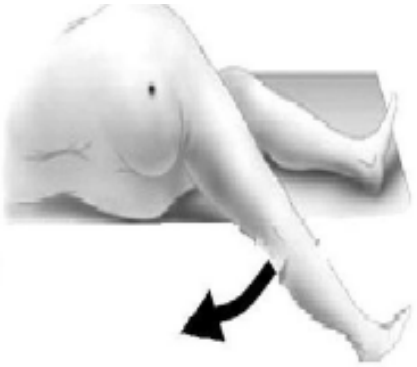
Figure 1.7. *Obturator sign (inflamed appendix is in pelvis, in contact with obturator muscle)*

Psoas (Koup's 2nd) sign (Fig. 1.8): this test is performed by having the patient lie on his left side. The examiner then slowly extends the patient's right thigh, thus stretching the iliopsoas muscle. This will produce pain to make the sign positive. This indicates presence of irritative inflamed appendix in close proximity to the psoas muscle. This is possible in retrocaecal appendicitis.

Obraztsov's sign: worsening of pain in the right iliac region during palpation while the patient raises the right leg bent at the knee.

Sitkovsky's sign: strengthening pain in the right lower quadrant in the position of patient on the left side (Fig. 1.9).

Bartomier-Michelson's sign: increased pain on palpation at the right iliac region as the person being examined lies on his/her left side compared to when he/she lies on his/her back.



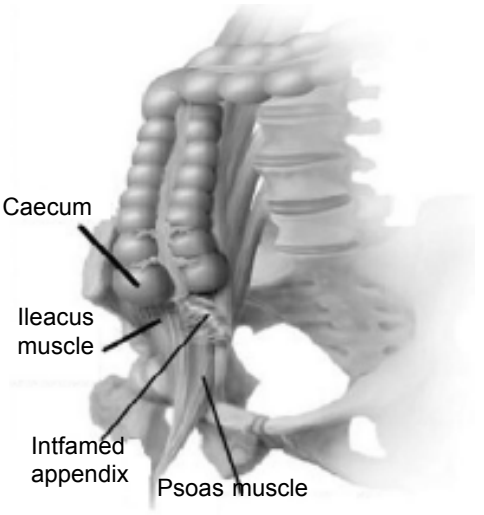


Figure 1.8. *Psoas sign (inflamed appendix is in retroperitoneal location in contact with psoas)*

Yaure-Rozanova's sign: increased pain on palpation with finger in right Petit's triangle (with retrocecal location of inflamed appendix).

Krymov's sign: increased pain when examining the peritoneum with the tip of the finger through the outer opening of the right inguinal ring.

Blumberg's sign: worsening of pain in the right iliac region when one's hand sharply off the abdominal wall after pressing it in the right iliac region. This is the classic method of demonstrating peritoneal inflammation is rebound tenderness.

Voskresensky's sign (sign of "sliding" or sigh of "shirt"): worsening of pain in the right iliac region when passing surgeon's right palm over the anterior abdiminal wall from the right subcostal area down on the patient's shirt stretched with the left surgeon's hand.

Rectal examination. This is important and should be performed in every patient suspected of suffering from appendicitis. Its primary function is to exclude any pelvic lesion particularly in females. But any manifestations in pelvic region can deal with acute appendicitis (in case of pelvic position of appendix vermiformis). In case of pelvic appendicitis there may not be any tenderness on the anterior abdominal wall, so rectal examination is very essential to exclude such appendicitis. When inflamed appendix lies in the pelvis, presence of a mass or tenderness will be present (Fig. 1.10).

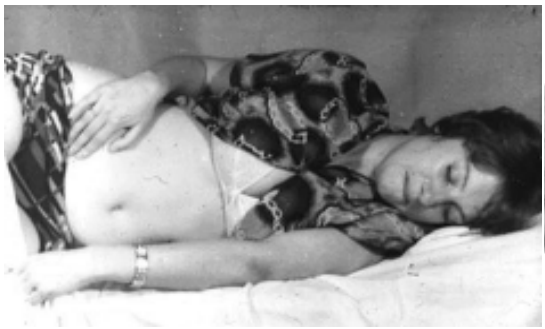


Figure 1.9. *Sitkovsky's sign*

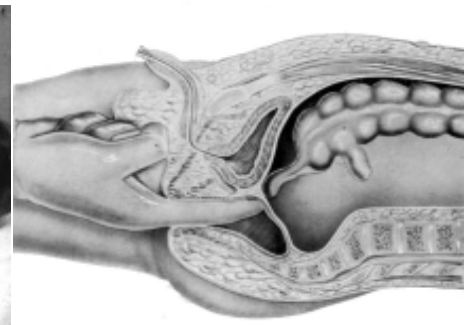


Figure 1.10. *Rectal examination*

1.6. SPECIAL INVESTIGATIONS

1. Blood examination will reveal moderate leucocytosis ranging from about 10 000 to 18 000 per cubic mm. with polymorph nuclear predominance. It must be remembered that in case of normal total and differential WBC count, the diagnosis of appendicitis should be questioned. In case of perforated appendicitis the total white cell count may rise above 18 000.

2. Urine examination. Except for high specific gravity due to dehydration, routine urine examination will usually reveal normal result in case of appendicitis. Only when the inflamed appendix lies near the urethra or bladder would white cells and even red cells be seen in the urine.

3. Imaging studies: include X-rays, US, CT.

X-RAY EXAMINATION. There is no sign of appendicitis in X-ray examination. Plain films may show a fecolith at the appendicular region. A distended loop of small bowel in the right lower quadrant may be seen. Less often a distended cecum or a gasfilled appendix may be detected. Abnormal findings include: fecolith, appendiceal gas, localized paralytic ileus, blurred right psoas, and free air (Fig. 1.11).

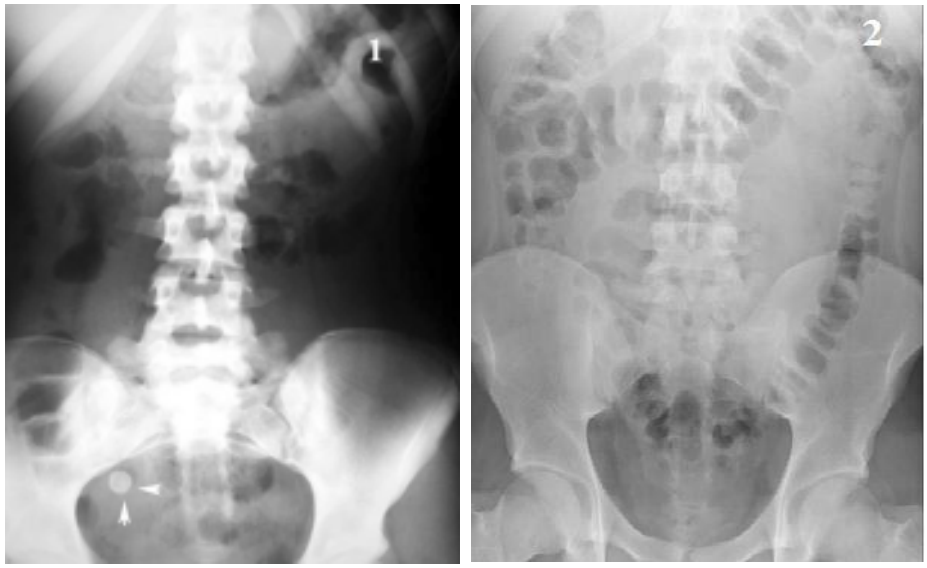


Figure 1.11. *Plain film radiography: 1 – appendicolith (arrows); 2 – paralytic ileus*

ULTRASOUND (US). Basis of this technique is that normal bowel and appendix can be compressed whereas an inflamed appendix cannot be compressed. Limitations of US: retrocecal appendix may not be visualized, perforations may be missed due to return to normal diameter (Fig. 1.12).

COMPUTER TOMOGRAPHY (CT): best choice based on availa-bility and alternative diagnoses. In one study, CT had greater sensitivity, accuracy, predictive value. CT appears to change management decisions and decreases unnecessary

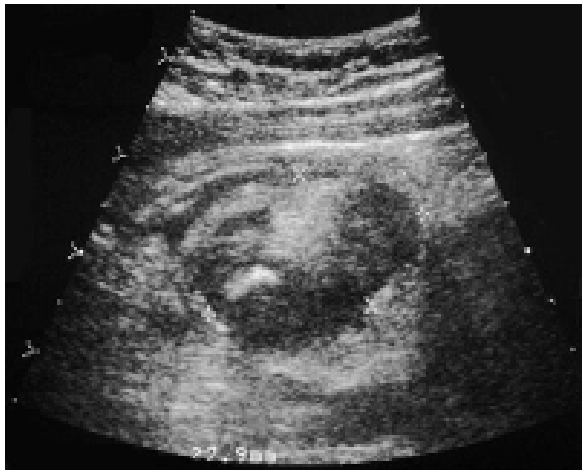


Figure 1.12. *Transverse abdominal US demonstrates a noncompressible mixed echotexture mass in the RLQ consistent with appendiceal abscess/phlegmon*

appendectomies in women, but it is not as useful for changing management in men. CT scan – results: acute appendicitis -thickened dilated appendix; periappendiceal fat stranding; scant free fluid; incidental small left ovarian cyst (Fig.1.13).

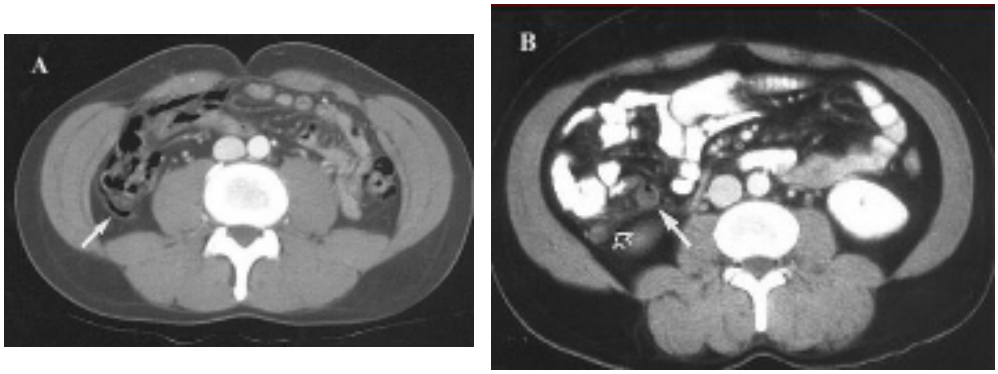


Figure 1.13. *CT scan abdomen & pelvis: A – normal appendix (white arrow); B – acute appendicitis: thickened dilated appendix; periappendiceal fat stranding; scant free fluid*

1.7. SPECIAL POPULATIONS

Infants.
The elderly.
Pregnant.

Acute appendicitis in infants:

- acute starting of the deseases;
- high temperature (38–49°C);
- cramping abdominal pain;
- repeated vomiting and diarrhea;
- pulse rate does not correspond to temperature;
- fast progress of destructive changes in the appendix;
- expressed symptoms of intoxication;
- frequent progress of diffuse peritonitis.

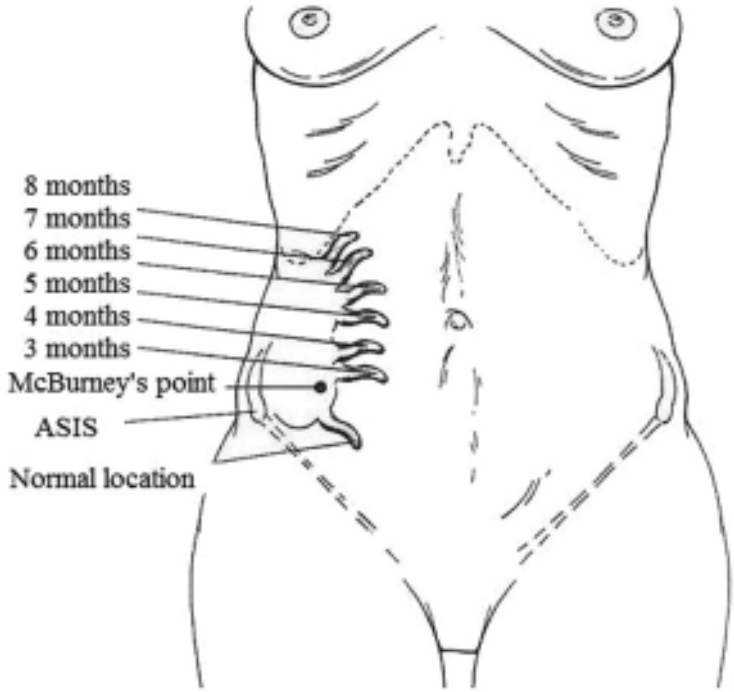


Figure 1.14. *The displacement of the cecum and the appendix of the growing uterus*

Acute appendicitis in children occurs at any age. Such clinical manifestation of acute appendicitis in infants are caused by a reduced resistance of the peritoneum to infection, small sizes of the gland, as well as increased reactivity of the child's body. In this regard, diagnosis in children is difficult and the disease develops faster than in adults, with a large percentage of destructive and perforated forms.

Appendicitis during pregnancy. Acute appendicitis is the most common extra uterine condition requiring an abdominal operation during pregnancy. Acute appendicitis occurs more frequently during the first two trimesters (Fig. 1.14).

During first 6 months of pregnancy symptoms of appendicitis do not differentiate from those in the nonpregnant women. During the third trimester, the problem is more, since mortality is about 20% – 10 times greater than that in the first and second trimesters. The clinical picture is also altered because of upward and lateral displacement of the cecum and appendix as a result of enlargement of uterus. Pain becomes higher and more lateral and diagnosis of pyelonephritis must be excluded by urine examination. Microscopically examination of urine will solve the problem. In addition, appendicitis in this last trimester tends to be more serious as delay in the diagnosis leads to increased incidence of perforation. Due to changes in the localization of the appendix pain in the abdomen can be defined not only in the right iliac fossa, but the right lateral abdominal flank, right upper quadrant, and even in the epigastric region. Muscle tension can be detected not always, especially in the last third of pregnancy due to the pronounced distension of the anterior abdominal wall. Displaced omentum is unable to wrap up the inflamed appendix. Rupture usually follows generalised peritonitis. Appendicitis in this trimester may lead to premature delivery in many of the patients.

Appendicitis in the elderly. Classic symptoms of pain, anorexia and nausea are also present in most old patients but in less pronounced form. Pain in the right lower quadrant is often very mild and causes little initial concern. So diagnosis at early stage becomes a problem. Rigidity of the right lower quadrant is not so pronounced in elderly patients due to lax abdominal wall. Even the case may be wrongly diagnosed as sub acute intestinal obstruction. To worsen the condition enemas may be given. Impaired blood supply and structural weakness of appendix are said to produce early perforation in these patients. But important than this is the delay in diagnosis.

Features course of acute appendicitis in the elderly (60 and over) age groups are as follows:

- erased as a result of the disease organism are activity and related diseases;
- emperature usually normal, lifting it up to 38°C and above there is a small number of patients;
- abdominal pain expressed slightly;
- protective muscle tension is absent or weakly expressed; the rapid development of destructive changes in the appendix (due to vascular sclerosis);
- a slight increase in the number of white blood cells, a moderate shift in leukocyte formula to the left, even with destructive forms.

1.8. CLINICAL FEATURES ACCORDING TO VARIOUS ANATOMICAL POSITIONS OF THE APPENDIX

RETROCECAL APPENDICITIS. Rigidity and tenderness may not be so obvious on the anterior abdomen (Fig. 1.15). This is because of the fact that cecum is in front of the inflamed appendix which may be retroperitoneal and is not in contact with the parietal peritoneum of the anterior abdominal wall. Tenderness may be present near the loin. There may be rigidity of the quadrates lumborum. Inflamed appendix may lie in close relation with the urethra and may cause slight pyorrhoea or haematuria.

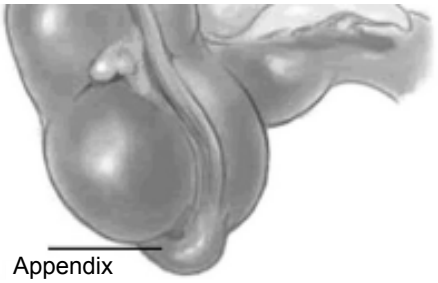


Figure 1.15. *Retrocecal appendix*

PELVIC APPENDICITIS. When the appendix is entirely within the pelvis there may be complete absence of rigidity and tenderness on the right iliac fossa. Rectal examination is helpful to detect such appendicitis. Tenderness will be present on the right side of the rectovesical pouch or pouch of Douglas. In case of such appendicitis patient may complain of tenesmus and diarrhoea. Coupe's obturator test is usually positive in this type of appendicitis. This is due to the fact that inflamed appendix is in contact with the obturator internal muscle. Passive internal rotation of the hip will cause pain in the hypogastric region. Inflamed appendix may lie in contact with the urinary bladder and may cause frequency of micturition and a little bit of pyorrhoea and haematuria.

PREILEAL AND POSTILEAL APPENDICITIS. Continued irritation of the ileum will lead to nausea and vomiting. This symptom becomes very prominent. Tenderness instead of being located on the McBurney's point is elicited more medially near the umbilicus. As inflamed appendix lies near the ileum it may cause slight diarrhea.

SUBHEPATIC APPENDICITIS. When the cecum is higher up than its normal position and the appendix is retrocecal and reaches the subhepatic position, such appendicitis may give rise to difficulty in diagnosis. The case is often diagnosed as acute cholecystitis. In case of such recurrent appendicitis or acute appendicitis the diagnosis is made of peptic ulcer. So one must be careful and should keep in mind such position of the appendix.

1.9. DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS

1. Acute cholecystitis.
2. Perforated peptic ulcer.
3. Cyclical vomiting – mostly seen in children.
4. Acetone is found in the urine and rigidity is absent; intestinal obstruction; carcinoma of the cecum .
5. Meckel's diverticulitis.
6. Acute pancreatitis.
7. Mesenteric vascular occlusion.
8. Twisted right ovarian cyst; retroperitoneal causes.
9. Right kidney colic and right sided acute pyelonephritis.

Pasternatsky's sign: the physician places his left hand on the patient's loin and using his right hand, taps with a moderate force on right hand overlying the kidney region on the loin. If the patient feels pain, the symptom "is positive". This sign is mainly positive in pyelonephritis. It may also be positive in nephrolithiasis, paranephritis, inflammation of pelvis, and also in myositis and radiculitis.

10. Torsion of testis – either descended or undescended testis; haematoma in the retroperitoneal tissue.

11. Pelvic inflammation.

12. Gynaecological disorders: salpingitis; ectopic gestation; ruptured ovarian follicle. It usually occurs halfway between menstrual periods i.e. about 14th to 16th day of the menstrual period. It occurs in young girls. Ovulation results in spilling of sufficient amount of blood and follicular fluid to produce such mild lower abdominal pain. If the right ovary is affected appendicitis may be simulated. Pain and tenderness are rather diffuse. Leucocytosis and fever are absent. There is no history of missed period.

Promptov's sign: with vaginal examination, pain occurs when the uterus is moved upward. Symptom determined in the presence of an inflammatory tumor of the appendages.

Zhendrinsky's sigh: the patient in the position on the back is pressed on the abdomen at the Kymmel's point (2 cm below and to the right of the navel) and ask the patient to sit down without taking his finger away. An increase in pain indicates of acute appendicitis, a decrease in pain indicates of acute adnexitis or salpingitis.

Pathological discharge from the genitals will also testify in favor of acute adnexitis.

13. Right pneumonia and pleurisy.

14. Diabetic abdomen – indicates abdominal pain and vomiting which sometime may precede coma.

1.10. COMPLICATIONS OF ACUTE APPENDICITIS

APPENDICULAR MASS. In majority of cases as soon as the appendix becomes gangrenous, omentum and coils of small intestine cover the inflamed appendix all around. There is no discrete collection of pus inside. This is an attempt of nature to prevent general peritonitis even if rupture of the appendix occurs. Usually such appendicular mass develops on the 3rd day after the commencement of an attack of acute appendicitis. This is a tender mass on the right iliac fossa. This mass usually resolves by conservative treatment. In untreated cases or when the patient does not react to the conservative treatment such appendicular mass may turn into an appendicular abscess and becomes larger in size.

Treatment of appendicular mass. In these cases conservative treatment should be started immediately. Nature has already localised the lesion and it is better not to disturb such localisation. Surgery at this stage is difficult and dangerous as it is difficult to find appendix due to adhesions and ultimately faecal fistula may form. When 48 hours have passed since commencement of the disease, presence of lump may be felt on careful palpation. With a skin pencil the lump is demarcated.

Conservative treatment includes:

1. Intravenous fluid with dextrose saline and Ringer's solution as and when required. Hourly nasogastric aspiration. An intake and output chart.
2. Diet. Nothing should be given by mouth.
3. Antibiotic therapy.

A close watch is kept on the patient while he undergoes the conservative treatment. The followings are the conditions which should stop the conservative treatment and immediate appendectomy should be carried out. This means, nature is failing to control the disease and there is a chance that the appendix may perforate at any moment. The conditions in favour of stopping the conservative treatment are: a) a rising pulse rate; b) vomiting or increase in gastric aspiration; c) increase in the abdominal pain – suggesting impending spreading peritonitis; d) increase in the size of the lump.

Conservative treatment should make the patient better by decreasing the pain, decreasing the amount of gastric aspiration (which indicates the return of peristalsis), temperature is decreased and pulse rate is becoming normal and the size of the lump is reducing considerably and ultimately disappears. About 90% of cases resolve without any problem. The patient is kept under observation for further 4 to 5 days after resolution of the lump. Before the patient is discharged he should take normal diet. He is instructed to have appendectomy done (interval appendectomy) 6 to 8 weeks after his discharge.

APPENDICEAL ABSCESS. A progressive suppurative process in an appendicular mass forms an appendicular abscess walled off by the omentum, inflamed cecum and coils of small intestine. Such abscess may follow rupture of the appendix with the expulsion of small content of the appendix distal to the obstruction. The cecal contents cannot come out due to the occluding faecolith. In such appendicular abscess there may be variable pyrexia and slight increase in the pulse rate.

There is definite increase of the leukocyte count with relative increase of polymorph nuclear cells. The commonest site of the abscess is in the lateral part of the iliac fossa (from retrocecal appendicitis). The second common position is in the pelvis. In untreated cases lethal form of peritonitis is produced by secondary rupture of appendicular abscess.

Treatment of appendicular abscess. Immediate drainage under antibiotic cover is the treatment of choice. The incision for drainage is made just medial to the anterior superior iliac spine at the level of the most prominent portion of the appendicular abscess. If the appendicular abscess is situated more medially the same incision for appendectomy is made. When after opening the peritoneum one sees appendicular abscess, it is better to drain the abscess.

A pelvic abscess may be drained in the female into the vagina and in the male into the rectum.

If the appendix is not removed when the abscess is drained, interval appendectomy should be done 6 to 8 weeks after.

PERITONITIS. Generalized peritonitis is the principal cause of continuing mortality from appendicitis and requires careful and energetic treatment. It is generally agreed that appendectomy must be performed in children whether peritonitis is diffuse or not, since any other course is associated with a higher mortality.

There is continuing controversy about the management of adults. The patient with perforated appendicitis in whom diffuse peritonitis develops, and who has failed to wall off the process so that the perforated appendix is a continuing source of peritoneal contamination, will benefit from appendectomy. On the other hand, a patient whose perforation initially leads to diffuse peritonitis, but in whom the perforated appendix is subsequently walled off as an appendicular abscess, is best managed by operation limited to drainage of the abscess. In both types of patients, general principles governing the management of bacterial peritonitis apply.

The question of if and what to drain in patients with diffuse appendicular peritonitis also has been an issue of controversy for years. While drainage of each localized collection of pus by means of soft rubber drains is certainly indicated, prophylactic placement of multiple drains within the abdominal cavity is an unwarranted practice. Such prophylactic intraabdominal drains are rapidly walled off and do not succeed in draining the peritoneal cavity except for a few hours.

The principles of wound management in these cases are irrigation of the wound with dilute antibiotic solution, subfascial drains only to localized collections of pus, closure of musculoaponeurotic layers, skin and subcutaneous tissues packed open rather than sutured and drained, daily rectal examination to detect development of pelvic abscess. Systemic administration of antibiotics should be continued for as long as clinically indicated.

INTRAABDOMINAL ABSCESSES: pelvic abscesses; interintestinal abscesses; subdiaphragmatic abscess, etc. Transmitted infecting organisms generally reflect the normal intestinal flora and a complex mixture of aerobic and anaerobic bacteria.

The most common strains of aerobic gram-negative bacilli (egg Escherichia coli and Klebsiella) and anaerobic (Bacteroides fragilis in particular) undrained abscess can spread to adjacent structures, damage to the vessels surrounding the passage (due to bleeding or thrombosis), will rupture the peritoneum, or bowel or a skin fistula. subdiaphragmatic abscess may extend to the thoracic cavity, resulting in empyema, lung abscess or pneumonia. abdominal abscess may track down the thigh or perirectal Fossa. splenic abscess is a rare cause of persistent bacterial endocarditis that persists despite appropriate antimicrobial therapy.

Wide range of presenting complaints, the patients may have general malaise and anorexia or weight loss, other people present in critically ill septic shock with

acute abdomen. *Sx:* fever, pain, nausea, vomiting, anorexia. Physical examination: local pain, perhaps a palpable mass, postoperative evaluation of abscess confused by painkillers and pain in the incision, with more than half is within 10 days of the initial operation. *Laboratory*: elevated white blood cell count, the yield of direct aspiration of Gram-positive, anticulture, positive blood cultures ~ 25% depending on the site.

Antibiotic therapy requires parenteral administration of empirical antibiotics. Start treatment before the abscess drainage, and be careful when all signs of systemic sepsis is resolved. As the abscess fluid generally includes both aerobic and anaerobic organisms early empirical therapy directed against both microbes. This can be achieved by antibiotic treatment or combination therapy with broad spectrum, a management representative. Special treatment, then guided by the results of cultures, retrieved from a boil. Patients who are immunosuppressed, yeast fungi species can be an important pathogenic role, and amphotericin B therapy may be indicated.

The drainage of pus is mandatory and is the first line of defense against infections gradually. Percutaneous catheter drainage guided by CT became the standard treatment for most intraabdominal abscesses. It can be difficult to avoid laparotomy, requires anesthesia, eliminates the possibility of wound complications of open surgery, and may reduce the length of hospitalization. It also avoids the possibility of contamination of other areas in the peritoneal cavity. CT guided drainage of the abscess cavity delimited and can provide secure access for percutaneous drainage. When performed by experienced hands, but also avoids the risk of injury to adjacent organs or blood vessels. After surgical drainage, clinical improvement should occur in 48–72 hours. The lack of improvement in this period, the second term of the CT review additional abscesses. surgical drainage should be compulsory, if the remaining liquid can be removed with irrigation catheter manipulation, merger or other investments.

The surgical procedure can also be intended to persist with the content of abscesses, such as hematoma, infection, fungal infection or pancreatic abscesses. Surgical drainage is an option if the skin is dry or fails if the collections are not susceptible to drainage catheter. Surgical approach can be either open or laparoscopic drainage (laparotomy) drainage. transperitoneal approach is safer to open a prudent use of preoperative antibiotics.

Although contamination of otherwise noninfected sites is an important concern, this complication is particularly reduced if the organizations involved are sensitive to selected drugs. transabdominal examination of the abdomen and allows for full cleaning of fibrin. It also allows full bowel movement to identify and remove all synchronized with the abscesses, which occur in more than 23% of patients.

Improved clinical outcomes in three days after treatment indicates drainage. The lack of improvement may indicate inadequate drainage or any other source of infection. If left untreated, inevitably sepsis multiorgan failure. The transabdominal approach open to intraabdominal abscess can be extremely difficult. Even Matt bowel and adhesions, and loss of anatomic integrity can pose serious problems. This is particularly true when the sensitive organs, like a loop of small intestine, observed intermittent wall abscess or cavity. Therefore, whenever possible, CT guided drainage is a useful first step.

PYLEPHLEBITIS. Pylephlebitis can complicate any intraabdominal or pelvic infection that occurs in the region drained by the portal venous system, especially appendicitis. In 1846 Waller documented autopsy findings in a patients with appendicitis, including multipule liver abscesses and pylephlebitis.

Pylephlebitis or infective suppurative thrombosis of the portal vein is a serious condition with significant morbidity and mortality, which can complicate intraabdominal sepsis of any etiology. Although universally fatal in the preantibiotic era, the outcome of this infection has improved somewhat with modern diagnostic and therapeutic modalities.

Diagnoses are based on clinical features, laboratory findings, blood culture and radiogical examinations, or postmortem examination.

Clinical feature includes lever, right upper quadrant pain, diarrhea, jaundice and hepatomegaly. The clinical "feature" that jaundice is rare in pylephlebitis, except in cases complicated by multiple liver abscesses, appears to be valid. Jaundict developed late (no earlier than 5 days after the onset of symptoms) since the relative absences of jaundice early in the illness helps distinguish pylephlebitis from ascending *Escherichia coli*, followed by *Proteus mirabilis, Klebsiella pneumonia, Enlerobacer species, Pseudomonas species* and grampositive cocci (*Staphylococcus aureus, Streptococcus species*). More recently, coloenteric anaerobic bacteria (Clostridia, Bacteroides, Fusobacterium and anaerobic streptococcus species) have been isolated with increasing frequency, as a consequence of both improved culture techniques and selective pressure by the prevalent use of antibiotics.

Modern ragiogical imaging techniques provide supportive diagnostic evidence. With repaid lo diagnostic imaging in pylephlebitis, the current series suggests that ultrasonography is a useful modality for demonstrating portal vein thrombosis. CT scanning also shows promise in this regard, and may be less operatordependent than ultrasonography. In the setting of probable intraabdominal infection, CT scanning may be the most reasonable initial choice for imaging, given its proven ability to detect not only thrombi but pericolonic abscesses as well. A contrastenhanced CT scan confirmed the portal vein thrombose.

Pylephlebitis begins with thrombophlebitis of small veins draining an area of infection. Extension of the thrombophlebitis into larger veins leads to septic thrombophlebitis of the portal vein, which can extend further to involve the mesenteric veins. The superior mesenteric vein was involved in 15 of 44 patients (34 percent) in one case series. Mesenteric vein involvement can lead to bowel ischemia, infarction and death. An associated hypercoagulable state is found in some cases of pylephlebitis. As an example, in one series of 44 cases, 18 were hypercoagulable due to clotting factor deficiencies, malignant conditions, or AIDS.

The list of treatments mentioned in various sources for pylephlebitis includes the following list. Always seek professional medical advice about any treatment or change in treatment plans. Surgical drainage of abscess and dead tissue removal, chloramphenicol, metronidazole, gentamycin, tobramycin, amikacin, clindamycin.

The prognosis of pylephlebitis usually refers to the likely outcome of pylephlebitis. The prognosis of pylephlebitis may include the duration of pylephlebitis, chances of complications of pylephlebitis, probable outcomes, prospects for recovery, recovery period for pylephlebitis, survival rates, death rates, and other outcome possibilities in the overall prognosis of pylephlebitis. Naturally, such forecast issues are by their nature unpredictable.

SEPSIS. Sepsis is a potentially life-threatening complication of an infection. Sepsis occurs when chemicals released into the bloodstream to fight the infection trigger inflammatory responses throughout the body. This inflammation can trigger a

cascade of changes that can damage multiple organ systems, causing them to fail. If sepsis progresses to septic shock, blood pressure drops dramatically, which may lead to death. Anyone can develop sepsis, but it's most common and most dangerous in older adults or those with weakened immune systems. Early treatment of sepsis, usually with antibiotics and large amounts of intravenous fluids, improves chances for survival.

1.11. TREATMENT OF ACUTE APPENDICITIS

Immediate appendectomy is the treatment of choice in acute appendicitis without rupture. Immediate appendectomy should be performed to obviate possibility of rupture of appendix and spreading peritonitis.

PREOPERATIVE PREPARATION. No patient with acute appendicitis should be taken directly to operation theatre on admission. All patients require a preoperative preparation which often requires more than 3 or 4 hours or at least 1 hour before the patient is taken to operation theatre. This is more important in case of patients in whom perforation and peritonitis are suspected.

Nasogastric aspiration is helpful in all patients with appendicitis, particularly in those with peritonitis. Intravenous fluid replacement should be started immediately to establish a good urinary output and to replenish the loss by nasogastric aspiration. High temperature is sometimes a problem in case of children. Temperature should be brought down and it is better that anaesthesia should not be induced in patients whose temperature is over 39°C.

Antibiotics are started immediately. Although prophylactic administration of antibiotics is a matter of controversy, the evidence in various reports in the past decade is clearly in favour of antibiotic administration. Antibiotics are of minor benefit unless the appendix is gangrenous or has perforated. But in cases of gangrenous appendix or perforated appendix antibiotics play a major role in reducing the incidence of wound infection if started preoperatively.

Surgical treatment:
– Open appendectomy.
– Laparoscopic appendectomy.

OPEN APPENDECTOMY. After the patient is anaesthetised, the abdomen is again thoroughly palpated. This will give a clear idea regarding the size of the mass. Position of the cecum is ascertained to choose the right incision close to the appendix.

Incision. When the diagnosis is confirmed McBurney's gridiron incision is made. McBurney's gridiron incision (Fig. 1.17). We make two virtual lines: first – line running from the umbilicus to the anterior superior iliac spine, second one – the line, perpendicular to the first in McBurney's point.

McBurney's point is the name given to the point over the right side of the abdomen that is one third of the distance from the ASIS (anterior superior iliac spine) to the umbilicus. This point roughly corresponds to the most common location of the base of the appendix where it is attached to the cecum. The angle between these two lines must be 90 degrees. Incision is made on the second line this way: one third must be upper than the first line and the two third must be lower than the first line. Length of this incision depends on the thickness of the abdominal wall, the age of the patient,

type of narcosis. Though this is the classical McBurney's incision, yet the surgeon should try to feel the cecum and position the incision accordingly, as sometimes cecum may be abnormally placed (even subhepatic).

The skin, fascia of Camper and fascia of Scarp are divided along the line of incision. The fibres of the external oblique aponeurosis are split along the line of incision and retracted (Fig. 1.18) The muscle fibres of internal oblique are now seen running perpendicular to the line of incision. These fibres and the fibres of the transversus abdominis are separated by inserting the tip of the artery forceps and opening it. The fingers are now introduced and these muscle fibres are retracted to expose the peritoneum. The peritoneum is picked up by two artery forceps and incised to enter the abdominal cavity.

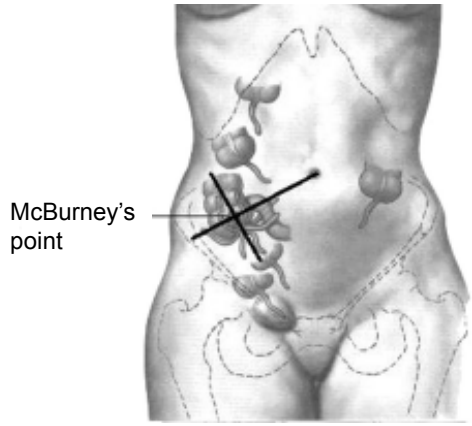


Figure 1.17. *McBurney's gridiron incision.*
Vermiform appendix
(variations in position)

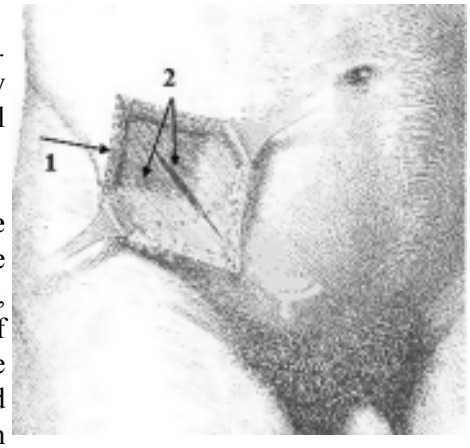


Figure 1.18. *McBurney's incision*

Technique of operation (**antegrade appendectomy** (Fig. 1.19). Isolation of the appendix. After opening the peritoneal cavity, the two fingers are introduced to get hold of the cecum. Coils of ileum, which has no tenie coli, may be taken out. Sometimes sigmoid colon, which has got a mesocolon, is taken out. In a visceroptoic cases the transverse colon, which has got omentum attached to it, is withdrawn.

Cecum is best withdrawn by following the peritoneum on the lateral side of the abdomen and it reaches the cecum which is relatively fixed because the ascending colon has got no peritoneum in its posterior surface. The cecum, which is relatively whitish, which has got tenia coli and no omentum and mesocolon, is taken out of the abdomen with the aid of a pair of Babcock' s tissue forceps. Now the anterior tenia coli are followed downwards to reach the vermiform appendix. Division of the mesoappendix: all other portions of bowel are reinserted into the abdominal cavity except the cecum and the appendix which are surrounded by a wet mop to separate them from the abdominal wound. A pair of tissue forceps is applied to the tip of the mesoappendix. The appendix is lifted up with this tissue forceps. The mesoappendix is pierced at its base with a mosquito artery forceps and the appendicular artery is secured with a ligature through this hole. The mesoappendix is now divided close to the appendix till the cecum is reached. One must be careful about the presence of accessory appendicular artery which should be held with ligature. If the appendix is kinked with firm adhesions, this division of the mesoappendix should be done in segments. Removal of the appendix. The base of the appendix is crushed with a pair of strong artery forceps. By this process only the mucous and the muscular coats are

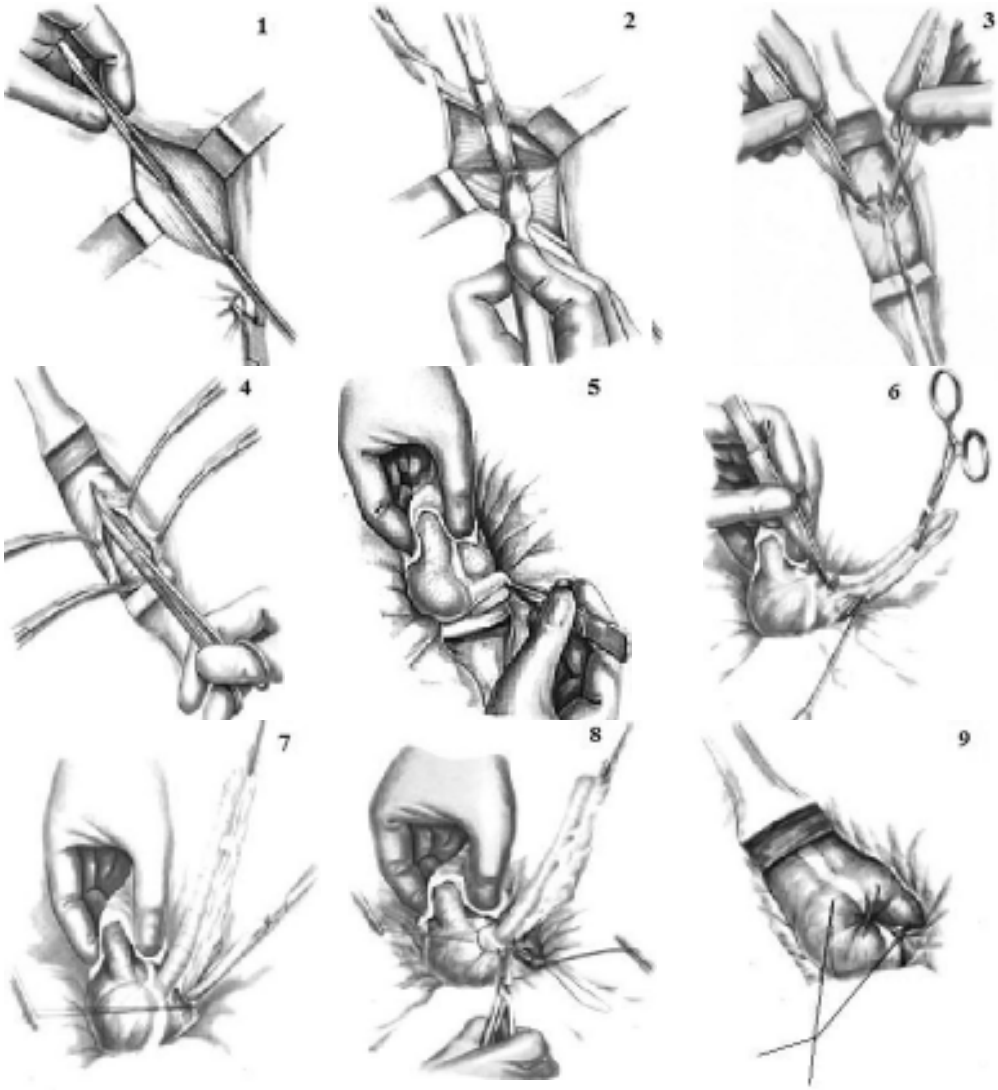


Figure 1.19. *Antegrade appendectomy: 1–4: McBurney's incision; 5: the taenia of the colon are followed to the base of the appendix; 6–7: ligation of the mesoappendix; 8–9: ligation of appendix base and invagination of the stump*

crushed and curled inwards to occlude the lumen but the peritoneal coat remains unaffected. A ligature is tied around the appendix area. A seromuscular pursestring (Lembert suture) or figure of N-suture is inserted in the cecal wall around the base of the appendix. A pair of artery forceps is applied to the appendix 5 mm distal the ligature. The intervening lumen is emptied beforehand by momentary pressure with an artery forceps. A swab is placed beneath the base of the appendix and the appendix is divided close to the forceps. The stump is cauterised with antiseptics and to invaginate while the pursestring suture is tightened.

RETROGRADE APPENDECTOMY (Fig. 1.20).

1. Removal of the appendix and invaginate stump by sutures.
2. Cut off appendix.

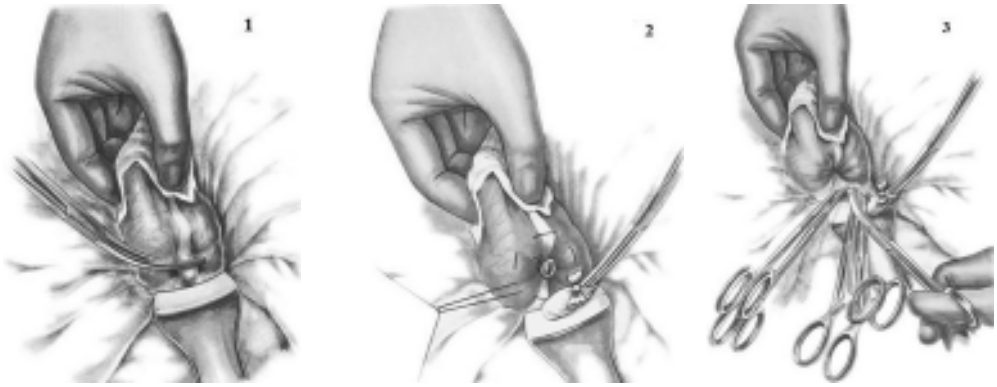


Figure 1.20. *Retrograde appendectomy*

In all cases the appendix, the knife, the swab and other instruments which have come in contact with the contaminated mucosa of the appendix are placed in a bowl and removed from the field of operation. The terminal ileum is drawn out of the wound and inspected for one metre or so to exclude the presence of kinking band of Lane, Crohn' s disease, Meckel's diverticulum etc. In female the right uterine tube and right ovary are also palpated.

Thus completing the operation, abdominal wound is sutured in layers as usual.

LAPAROSCOPIC APPENDECTOMY. A pneumoperitoneum is created in the usual fashion (CO_2). The 3 trocars are inserted. The cecum is retracted (Fig. 1.21–1). The appendix is grasped with a 5 mm clawtype grasper inserted via the suprapubic troacar (Fig. 1.21–2, 3; 1.21–4). The surgeon creating the mesenteric window. Transecting the mesoappendix and appendix and retrieving the appendix. The intraabdominal cavity is irrigated thoroughly with normal saline or antiseptic solution.

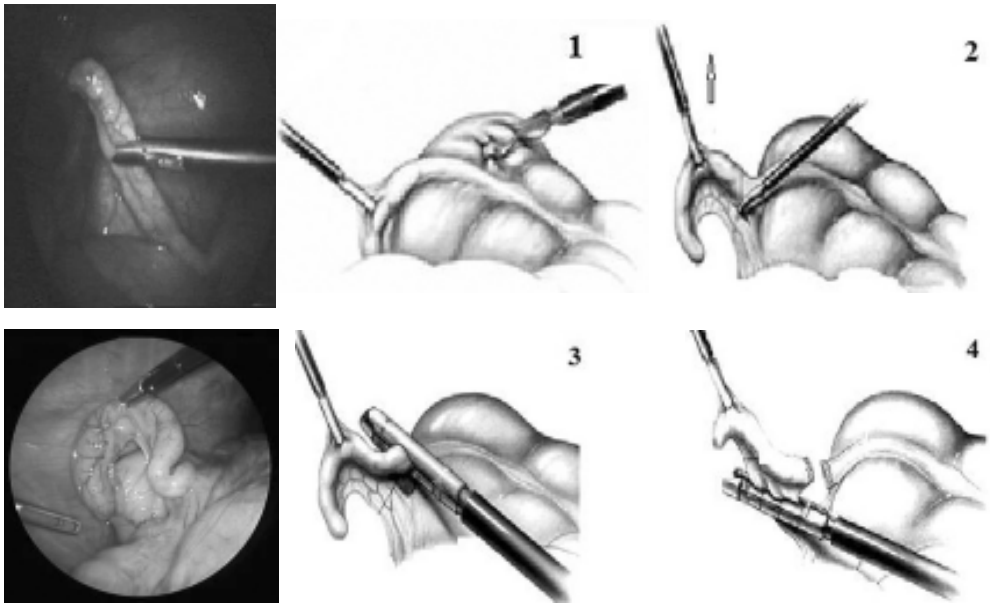


Figure 1.21. *Laparoscopic appendectomy*

1.12. COMPLICATIONS OF APPENDECTOMY

Postoperative complications occur in only 5 per cent of patients if an unperforated appendix is removed intact, but in over 30 per cent of patients with gangrenous or perforated appendicitis. The more frequent complications of appendectomy include: incision infection; peritonitis and abdomen abscess; bleeding; stool fistula; stump infection; adhesive intestinal obstruction.

Infection of the subcutaneous tissues is the most common complication following appendectomy. The organisms recovered from the appendicular fossa in cases of acute appendicitis are most frequently anaerobic *Bacteroides* species, followed by the aerobes *Klebsiella, Enterobacter*, and *Escherichia coli*. Since wound infections in cases of appendicitis are caused by fecal organisms, the classic signs of infection (calor, dolor, rubor, tumor) often are not present. The early signs of a fecal wound infection are undue pain and modest edema around the wound. If such signs are present, the skin and subcutaneous tissue should be opened. A rush of pus should not be expected. Fecal infections induce necrosis of subcutaneous fat, often of considerable extent, but only as damaged fat liquefies does much pus form.

Pelvic, subphrenic, or intraabdominal abscess occurs in up to 20 per cent of patients with gangrenous or perforated appendicitis (Fig. 1.22). Abscesses usually are due to preoperative contamination of the peritoneal cavity by organisms leaking from a gangrenous or perforated appendix. Less often, contamination ensues from intraoperative spillage. Occasionally, an abscess forms around a retained fecolith or other foreign body. The presence of an abscess is manifested by recurrent fever, malaise, and anorexia, usually beginning about 1 week after appendectomy.

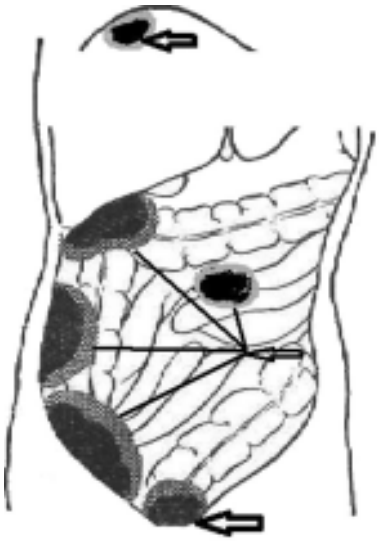


Figure 1.22. *Pelvic, subphrenic and intra-abdominal abscess (arrows)*

A pelvic abscess may cause diarrhea and may be palpated on vaginal or rectal examination. Subphrenic abscess can be diagnosed by the classic signs of effusion in the overlying thorax and immobility of the involved diaphragm. Confirmation of the presence of an intraabdominal abscess may require an exploratory laparotomy. All abscesses must be drained.

Fecal fistula usually is not a dangerous complication of appendectomy. Fistulas may be due to a retained foreign body, such as a sponge, a pursestring suture tied too tightly, a ligature slipping from a tied but noninverted appendicular stump, necrosis from a periappendicular abscess encroaching on the cecum, erosion of the wall of the cecum by a drain, regional enteritis, colon obstruction by an undetected neoplasm, or retention of a mucusproducing tip of the appendix.

Most fecal fistulas close spontaneously; all that is required is to ensure that the tract remains open until drainage ceases. Fecal fistulas will not close spontaneously if the tip of the appendix or a foreign body is present, if the bowel beyond the fistula is obstructed, or if the mucous membrane of the gut is continuous with the skin. In such cases, closure of the fistula requires an operation.

Pylephlebitis, or portal pyemia, is a serious illness characterized by jaundice, chills, and high fever. It is due to septicemia of the portal venous system, leading to development of multiple liver abscesses.

Intestinal obstruction: initially paralytic but occasionally going on to true mechanical obstruction, may occur with slowly resolving peritonitis in complicated appendicitis. Late mechanical obstruction following appendicitis is uncommon. The development of a mechanical bowel obstruction usually requires operative relief.

Sepsis. Sepsis represents the systemic response to an infection, characterized by an exaggerated inflammatory response (SIRS) and widespread tissue injury involving otherwise normal tissue that is unregulated and selfsustaining.

PROGNOSIS

- Mortality rate is 0.06% in unruptured appendix.
- 3% in case of rupture.
- 15% in case of rupture in elderly.

Chapter 2

ACUTE CHOLECYSTITIS

Cholecystitis is an inflammation of the gallbladder that causes severe abdominal pain. In 90–95% of cases, acute cholecystitis is caused by gallstones in the gallbladder. Severe illness and, rarely, tumors of the gallbladder may also cause cholecystitis.

2.1. ANATOMY

The esophagus, stomach, large and small intestine, aided by the liver, gallbladder and pancreas convert the nutritive components of food into energy and break down the non-nutritive components into waste to be excreted.

The gallbladder stores and concentrates bile, and the ducts function as a bile drainage system. The flow of bile through the bile ducts is affected by several factors, including hepatic secretory pressure, the tone in the sphincter of Oddi, the rate of gallbladder fluid absorption, and gallbladder contraction.

Anatomically, the gallbladder is a pear-shaped musculo membranous reservoir lying in the gallbladder fossa on the inferior aspect of the liver. The fundus of the gallbladder lies close to the anterior abdominal wall, near the hepatic flexure of the colon. The surface marking of the gallbladder fundus is in the region of the costal cartilage. At this point, it is covered by peritoneum; its appearance may be obscured, owing to its proximity to the hepatic flexure of the colon. The body of the gallbladder is adjacent to the duodenum, which indents and produces a frequent ultrasonographic artifact that mimics gallstones or a mass in the gallbladder.

The gallbladder is a thin-walled, pear-shaped organ covered by peritoneum and attached to the inferior surfaces of the right quadrate lobes of the liver. Normally, it is 7 to 10 cm. long and 3 to 5 cm. in diameter and has a capacity of 30 to 60 ml. Anatomically, it is divided into a fundus or tip, which protrudes from the anterior edge of the liver, a corpus or body, an infundibulum called Hartmann's pouch, and a narrow neck that leads into the cystic duct (Fig. 2.1). Topographically, the fundus of the gallbladder is located behind the ninth right costal cartilage at the junction of the costal margin with the right border of the rectus abdominis muscle. The cystic duct from the gallbladder is about 2 to 4 cm. long and contains prominent mucosal folds

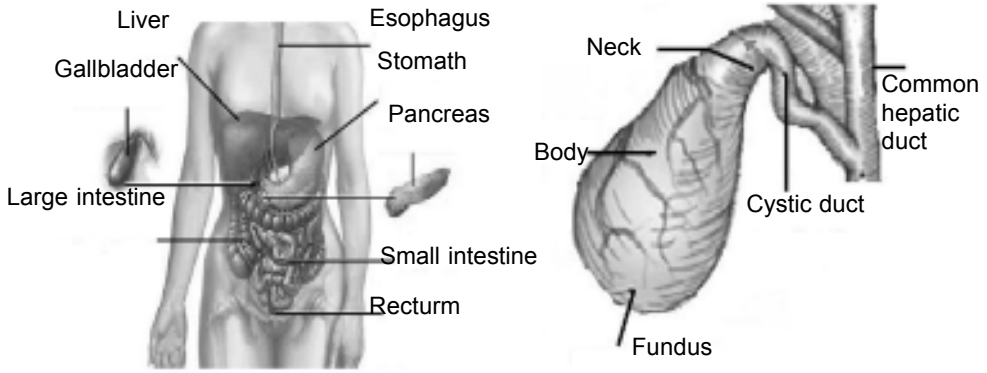


Figure 2.1. *Digestive system. Gallbladder*

called spiral folds or valves of Heister. The cystic duct joins the right lateral aspect of the common hepatic duct to form the common bile duct. The triangle bounded by the common hepatic duct medially, the cystic duct inferiorly and the cystic artery is known as **Calot's triangle**. The fact that cystic artery, right hepatic artery & para-right hepatic duct run within the triangle makes an important area of dissection during cholecytectomy.

An inflamed gallbladder may perforate the colon or duodenum because of the close proximity of the gallbladder to these structures. The mucosa of the gallbladder neck is thrown into folds, giving an echogenic appearance that may also mimic gallstones. A small pouch, known as the Hartmann pouch, projects from the right side of the gallbladder neck. In patients in whom the Hartmann pouch is visible, pathology, particularly dilatation, is often present. The gallbladder fundus is often folded over; in such cases, the gallbladder then assumes a double-barrel appearance.

The physiological function of gallbladder: storage and concentration of hepatic bile; secretion of water and electrolytes; empting bile into the common bile duct.

Biliary tract (Fig. 2.2):

1. Intra-hepatic bile duct.
2. Extra-hepatic bile duct.
3. Gallbladder.
4. Oddi sphincter.

Intra-hepatic bile duct:

1. Bile canaliculi.
2. Segmental bile duct.
3. Lobal bile duct.
4. Hepatic part of left and right hepatic duct.

Extra-hepatic bile duct:

1. Left and right hepatic duct.
2. The common hepatic duct (diameter: 0.4–0.6 cm length: 2–4 cm).
3. Common bile duct (diameter: 0.6–0.8 cm length: 7–9 cm).
4. Gallbladder.
5. Cystic duct.

The extrahepatic bile duct system originates from the liver as the right and left hepatic ducts, each of which is 1 to 2 cm long and drains the respective lobe of the liver. The two ducts join to form the common hepatic duct, a 2 to 4 cm long structure in the porta hepatis. The union of the common hepatic duct with the cystic duct gives rise to the common bile duct, which is 8 to 15 cm long and 5 to 10 mm in outside diameter. The common bile duct descends in the hepatoduodenal ligament to the right of the hepatic artery and anterior to the portal vein, passes behind the first part of the duodenum and through the pancreas, and enters the descending duodenum on its posteromedial aspect about 10 cm distal to the pylorus at the papilla of Vater. The choledochoduodenal junction is an oblique passageway through the duodenal wall occupied by the common bile duct and the main pancreatic duct of Wirsung. These two ducts usually join in a common channel, the ampulla of Vater, which opens into the duodenum at the papilla of Vater; however, the two ducts may join before entering the duodenal wall or may empty into the duodenum through separate openings. The muscle of the choledochoduodenal junction, called the sphincter of Oddi, regulates the flow of bile and consists of several components. The bile secretion is around 0.5–1000 ml per day.

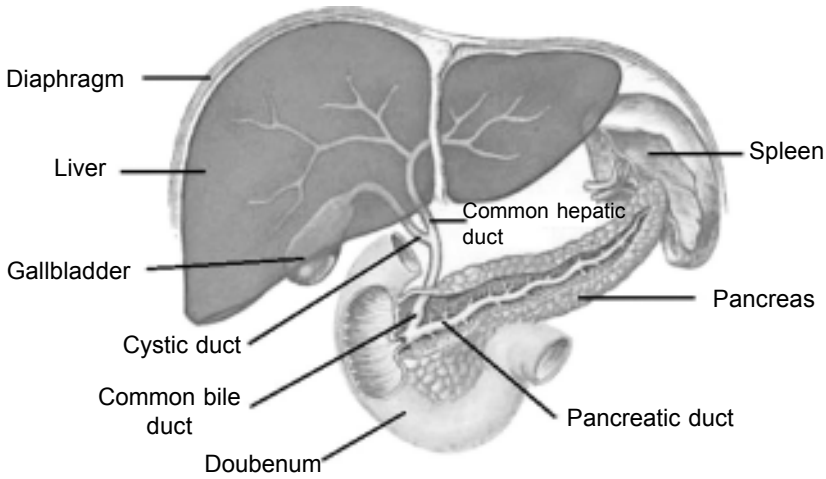


Figure 2.2. *Intra-hepatic and extra-hepatic bile duct*

The arterial blood supply to the common bile duct comes mainly from the retroduodenal artery, a branch of the gastroduodenal artery. The gallbladder is nourished by the cystic artery, which originates from the right hepatic artery, to the right of and behind the common hepatic duct, and divides into anterior and posterior branches. During cholecystectomy, the cystic artery is usually found in the cystic triangle of Calot, a space bounded by the liver, the common hepatic duct, and the cystic duct. The triangle contains the right hepatic artery with its cystic artery branch, a large lymph node, and, in its depths, the right branch of the portal vein. Venous drainage from the extrahepatic biliary system is into the portal vein. Lymphatic vessels from the gallbladder join those from the liver to empty into the thoracic duct. Lymph nodes at the neck of the gallbladder, at the junction of the cystic duct and hepatic ducts, and at the end of the common duct play a prominent role in the lymphatic drainage and are regularly enlarged in cholecystitis. The innervation of the biliary system is similar to that of the liver. Vagal stimulation causes contraction of the gallbladder, whereas sympathetic stimulation produces the reverse actions. The effect of vagal stimulation on the sphincter of Oddi is variable.

2.2. ETIOLOGY AND PATHOGENESIS

Appearance of acute cholecystitis is associated with the action of a few etiological factors.

Acute cholecystitis represents an acute inflammation of the gallbladder, caused in most instances by obstruction of the cystic duct, resulting in acute inflammation of the gallbladder wall; the usual cause of the obstruction is a gallstone. Acute cholecystitis is one of the major complications of cholelithiasis. The inflammatory process begins with a calculous obstruction of the cystic duct or gallbladder neck (Fig. 2.3). The exact mechanism by which gallbladder inflammation is initiated is unknown.

Pathology: cystic duct obstruction → gallbladder → edema →suppurate → gangrene → pericholecystic abscess → perforation.

Infection and stagnation of the bile (bilious hypertension) play leading roles in its development. Microorganisms are identified in 80% of cases early in the course of

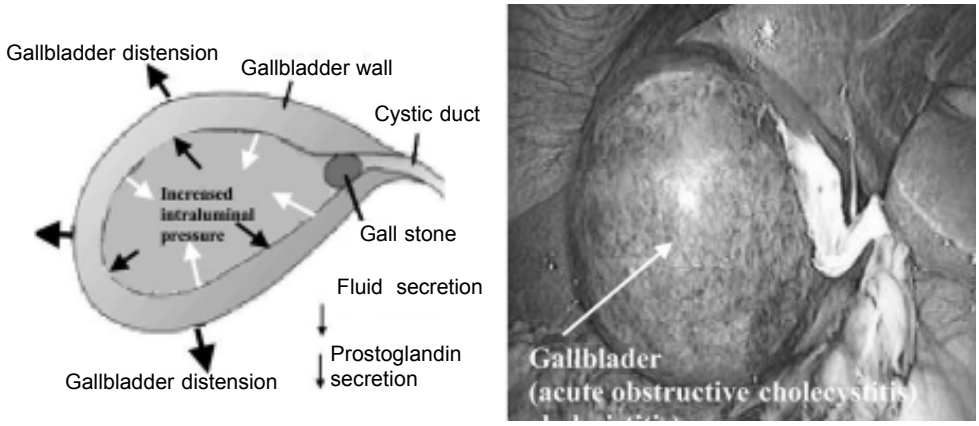


Figure 2.3. *Pathogenesis of acute obstructive cholecystitis*

disease; Escherichia coli is the primary organism found; other organisms include gram-negative aerobic rods, enterococci, and a number of anaerobes. The bacterial invasion is not considered to be a primary event, because in 20% of patients, no bacterial growth occurs in surgical specimens. The general consensus is that bacterial infection is a secondary event, not an initiating one. Supposedly, there are 3 ways that the infection gets into gallbladder, namely – hematogenic, lymphogenic and enterogenetic. In most cases the infection of the gallbladder occurs hematogenically (from the sysemic blood circulation along the system of common hepatic artery or from the gastrointestinal tract on the porta vena.

Spontaneous resolution of acute cholecystitis may occur within 5–7 days after onset of symptoms, because of the reestablishment of cystic duct patency. In the majority of such cases, fibrotic wall thickening of the gallbladder occurs; this is characteristic of chronic cholecystitis. In more than 90% of cholecystectomy specimens, the histologic pattern is one in which acute cholecystitis is superimposed on chronic cholecystitis. If cystic duct patency is not reestablished, inflammatory cell infiltration of the gallbladder wall in association with mural and mucosal hemorrhagic necrosis follows. Gangrenous cholecystitis may be seen in as many as 21% of cases.

Acalculous cholecystitis occurs in a different clinical setting. It occurs more often in males (usually children) and in persons older than 65 years. The pathophysiology of acalculous cholecystitis is not well understood but is probably multifactorial. It is probable that acalculous cholecystitis occurs through the combined effects of the actions of systemic mediators of inflammation; localized or generalized tissue ischemia; and bile stasis.

Often, predisposing factors place persons at risk for bile stasis; such factors include starvation; use of parenteral nutrition; use of narcotic analgesics; and a lack of mobility in postoperative states. Hypovolemia and shock predispose patients to tissue ischemia. Ischemia, such as occurs in association with small-vessel vasculitis, may be a primary cause of acalculous AC; ischemia may also occur as a complication of hepatic chemoembolization. Often, functional cystic duct obstruction is present; such obstruction is related to inflammation and viscous bile. Extrinsic compression may play a role in the development of bile stasis.

In the majority of patients with acalculous acute cholecystitis , secondary infection with gram-negative enteric flora occurs; however, in patients with typhoid

fever, infection with Salmonella organisms has been identified as a primary event. AIDS-related cholecystitis and cholangiopathy may occur secondary to cytomegalovirus (CMV) infection and infections with Cryptosporidium organisms.

In patients who have emphysematous cholecystitis, ischemia of the gallbladder wall is followed by infection with gas-forming organisms that produce gas in the gallbladder lumen, in the gallbladder wall, or both. In 30–50% of patients, preexisting diabetes mellitus is present; the male-to-female ratio is 5:1. Gas may be confined to the gallbladder; however, in 20% of cases, gas is also seen in the rest of biliary tree. Gallstones are not present in 30–50% of cases, and the mortality rate is 15%. There is a predisposition for gangrene formation and perforation, but clinical symptoms are mild; such symptoms can be deceptive. Emphysematous cholecystitis may occur after chemoembolization performed as palliation for hepatocellular carcinoma; after atheromatous embolism during aortography; and after gallbladder hypoperfusion during cardiorespiratory resuscitation.

2.3. CLASSIFICATION

– According to the etiology: 1. Calculous. 2. Acalculous. 3. Parasitic diseases.

– Clinical and morphological forms: 1. Simple. 2. Phlegmonous. 3. Gangrenous. 4. Perforated.

– Complicated and not complicated.

Acute cholecystitis (AC) occurs as a result of inflammation of the gallbladder wall, usually as a result of obstruction of the cystic duct. In 90–95% of cases, AC is initiated by the impaction of a calculus in the neck of the gallbladder or in the cystic duct. Acute acalculous cholecystitis (AAC) represents inflammation of the gallbladder in the absence of gallbladder calculi. AAC occurs more commonly in children and adults who are critically ill or in those who have recently undergone the stress of severe trauma, burns, or major surgery.

2.4. ACUTE CALCULOUS CHOLECYSTITIS

Clinical-morphological forms:

1. Simple (catarrhal).
2. Phlegmonous.
3. Gangrenous.
4. Perforated.

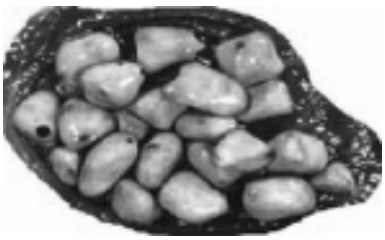


Figure 2.4. *Acute calculous cholecystitis*

Cystic duct obstruction → gallbladder → edema → suppurate → gangrene →

↓ ↓

Cholecystoenteric fistula → intestinal obstruction

Peritonitis

Acute → chronic → atrophy

2.4.1. CLINICAL FEATURES

1. Complains:

Acute calculous cholecystitis (Fig. 2.4) is diagnosed on the basis of symptoms and signs of inflammation in patients with peritonitis localised in the right upper quadrant.

In nearly every patient, abdominal pain of sudden onset is the first symptom of acute cholecystitis. Initially, this may be interpreted by the patient or the physician as another episode of biliary colic and may delay admission to the hospital. Key points of distinction include: the absence of food-related onset and the nature of the pain. At first, the pain is dull localized in the right upper quadrant, and constant. As the inflammatory process continues, parietal peritoneum becomes involved, and the pain becomes more intense and localized in the right subcostal area.

Pain can irradiates to the right shoulder or scapula, lumbar region or right shoulder girdle, sometimes to the heart **(cholecysto-cardial syndrome)**.

Nausea is frequent, as it is in biliary colic, but vomiting is more common. Severe vomiting suggests the presence of a common bile duct stone, acute pancreatitis, or a bowel obstruction. Loss of appetite, malaise are more common in patients with acute cholecystitis.

Suppurative complications of acute cholecystitis are manifested by fever greater than 37–38° C, tachycardia, chills and jaundice.

2. Objective sings of disease:

- Dry and coated tongue.
- Abdominal wall in the right upper quadrant lags behind in the act of breathing.
- During superficial palpation – muscle tension and tenderness in the right upper quadrant.
- During deep palpation – strengthening of local pain, palpation of the gallbladder fundus is possible.
- Patinent can feel pain during percussion in the right upper quadrant but auscultation – no changes.

3. Pathognomonic signs:

- **Mussy-Georgievsky's sign:** tenderness at the point of the phrenic nerve, between the heads of the sterno-cleidomastoid muscle.
- **Kera's sign:** pain during deep palpation in Ker's point.
- **Ortner`s sign:** pain during tapping over the right costal arch by the edge of the hand.
- **Murphy's sign:** sharp pain in the right hypochondrium when the examiner hands press the gallbladder at the height of inspiration.
- **Blumberg's sign:** worsening of pain in the right upper quadrant when one's hand sharply off the abdominal wall after pressing it in the right upper region.

2.4.2. DIAGNOSIS

LABORATORY STUDIES. In the typical patient, there is a mild leukocytosis ranging from 10 000 to 15 000. White blood cell counts greater than 15 000 indicate the development of suppurative complications but are not reliable indicators of complicated cholecystitis.

Hyperbilirubinemia in the range more than 21μmol/l occurs in 20 to 30 per cent of patients, but jaundice is not usually clinically overt. Values exceeding this range suggest the presence of common bile duct stones or septic complications. It should be recognized that hyperbilirubinemia is not pathognomonic of choledocholithiasis and that all patients with acute cholecystitis and jaundice should not be subjected to common bile duct exploration.

Mild elevations of liver enzymes occur in 40 per cent of patients, and slight alkaline phosphatase elevations occur in 15 per cent. Abnormalities depend to a certain extent on the nature, severity, and duration of the disease but are not reliable indicators of complicated cholecystitis. Enzyme abnormalities usually resolve within 24 to 48 hours. In viral hepatitis, enzyme elevations are higher and persist for weeks. Liver enzyme elevation does not aid in the differentiation of alcoholic hepatitis and acute cholecystitis, and liver biopsy is sometimes necessary.

The serum amylase is abnormal in 15 per cent of patients with acute cholecystitis. Hyperamylasemia suggests the coexistence of choledocholithiasis and perhaps pancreatitis. If acute pancreatitis is present, the amylase-creatinine clearance ratio will usually be greater than 5.

RADIOLOGIC STUDIES.

X-ray. The main value of plain x-ray examination of the abdomen in acute cholecystitis is to aid in the exclusion of other diseases. Occasionally, there may be an enlarged gallbladder shadow or evidence of gallstones (15% of gallstones are radiopaque) (Fig. 2.5, 2.6).

Either of these findings is presumptive evidence of gallbladder disease in the patient with acute abdominal symptoms. If air is seen within the gallbladder or biliary tree, emphysematous cholecystitis or a cholecystenteric fistula should be suspected.

Ultrasonography has an accuracy of 96 per cent in detecting gallstones and should be performed routinely in the initial evaluation of a patient with suspected acute cholecystitis. The presence of calculi or an enlarged gallbladder substantiates the clinical diagnosis (Fig. 2.7).

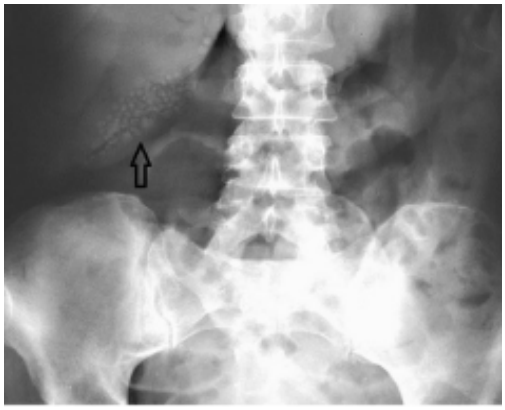


Figure 2.5. *Plain abdominal radiograph of a 68-year-old woman who presented with acute abdominal pain. There are multiple calculi distributed in a pyriform shape in the right upper quadrant; these are suggestive of gallstones (arrow)*

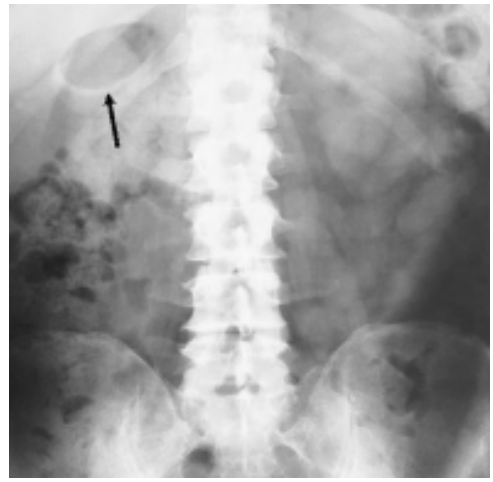


Figure 2.6. *Plain abdominal radiograph in a 49-year-old diabetic woman shows air within the gallbladder lumen due to emphysematous cholecystitis (arrow)*

CT scan. CT scan may demonstrate evidence of acute cholecystitis, including gallbladder wall thickening, pericholecystic fluid and edema (Fig. 2.8).

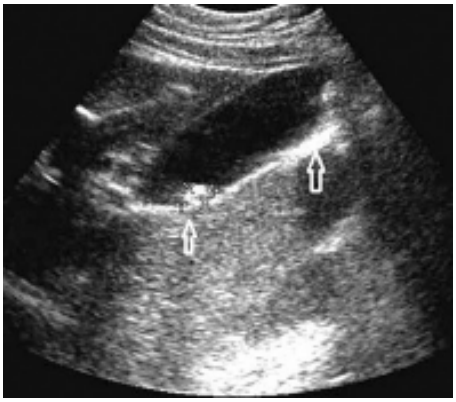


Figure 2.7. *Acute calculous cholecystitis (arrows)*

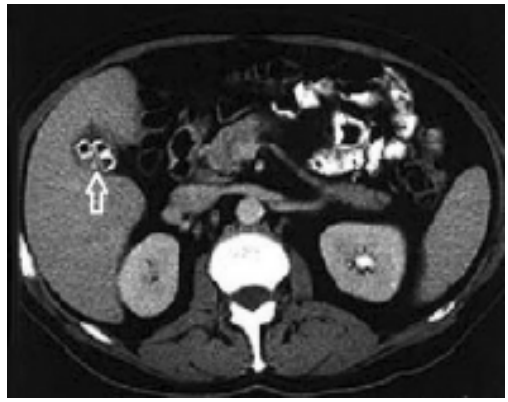


Figure 2.8. *CT scan of the upper abdomen showing multiple gallstones (arrow)*

2.4.3. COMPLICATIONS OF ACUTE CHOLECYSTITIS

- Paravesical infiltrate.
- Paravesical abscess.
- Empyema of gallbladder – Fig. 2.9.

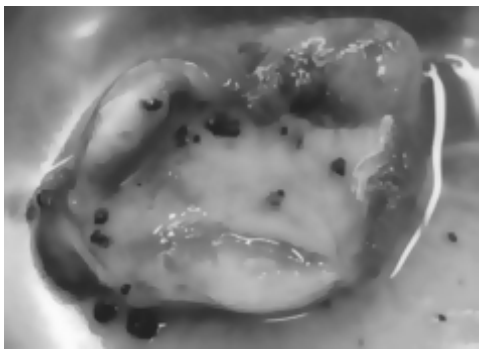


Figure 2.9. *Empyema of gallbladder*

– Perforated gallbladder. Occurs in 10% of acute cholecystitis, usually becomes a contained abscess in the right upper quadrant. Less commonly, perforates into adjacent viscus = cholecystoenteric fistula & the stone can cause gallstone ileus. Often perforates into the abdominal cavity with the progress of local unrestricted or general peritonitis

– Choledocholithiasis; obstructive jaundis; cholangitis; abscesses of the liver; acute pancreatitis; acute syndrome Mirizzi (Fig. 2.10); sepsis and SIRS.

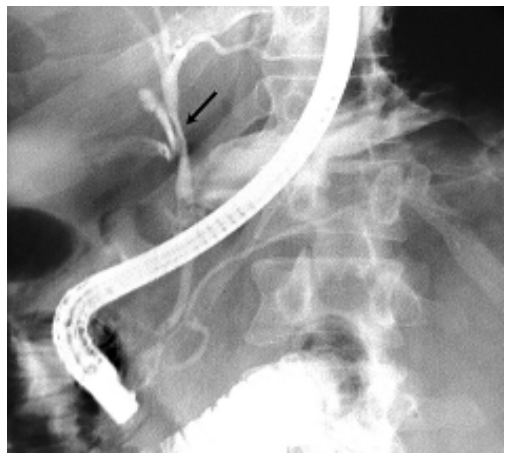


Figure 2.10. *This patient presented with acute cholecystitis, as confirmed at imaging. His pain resolved over a few days, but mildly elevated bilirubin levels persisted. Endoscopic retrograde cholangiopancreatographic study shows smooth narrowing of the bile duct (arrow) at the site of insertion of the cystic duct (Mirizzi syndrome). Note the small calculus in the cystic duct*

Differential diagnosis with: perforated peptic ulcer; acute pancreatitis; retrocaecel appendicitis; right lower lobe pneumonia; hepatic abscess; acute intestinal obstruction; renal colic.

2.4.4. TREATMENT OF ACUTE CHOLECYSTITIS

Conservative treatment:

1. Hospital admission.
2. Bed mode during 7–0 days.
3. Diet № 5a, warm drink; food intake 5–6 time a day by small portions.
4. Intravenous hydration (saline solutions, 5% glucose solution, etc.).
5. Systemic antibiotics (the antibiotic regimen should be appropriate for typical bowel flora – gram-negative and anaerobes).
6. Elimination of pain: M-cholinolitics (atropine sulfate, metacin, plathyphyllin). Spasmolitics (papaverin, no-spa). Analgetics (analgin, baralgin). **Non-narcotic analgetics (!).**
7. Monitoring of the patient: temperature, physical examination, and laboratory values.

Surgical treatment:

1. Attack within 48–72 h of diagnosis.
2. Deterioration in patient's general condition.
3. Complications are present.
4. Perforation.
5. Peritonitis.
6. Acute obstructive suppurative cholangitis.
7. Acute pancreatitis.

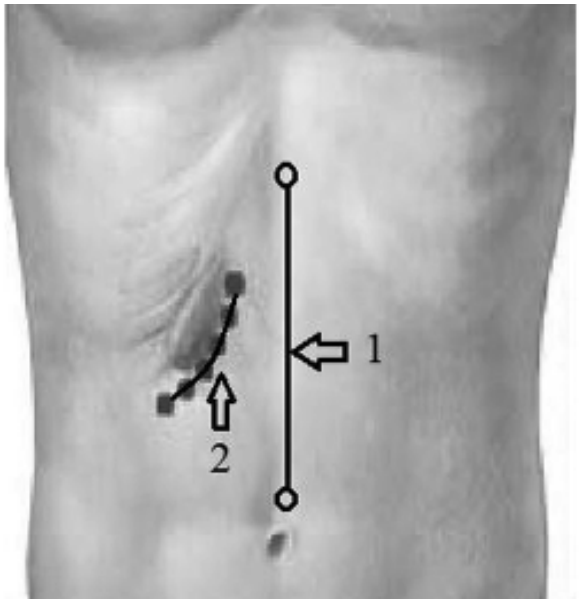


Figure 2.11. *Open procedure incision: 1 – midline laparotomy; 2 – bright subcostal incision*

Early cholecystectomy prevents the development of complications or recurrent attacks during the recommended 6-week "cooling-off" period. In addition, early surgical treatment reduces operative morbidity without increasing mortality. If the diagnosis of acute cholecystitis is firmly established and the patient is an acceptable anesthetic risk, cholecystectomy should be performed within 48 hours of admission. Delayed operation is preferred only for those with coexistent medical problems that make surgical risk prohibitive, such as myocardial infarction, congestive heart failure, or pneumonia.

SURGICAL METHODS: open cholecystectomy; laparoscopic cholecystectomy.

Open cholecystectomy: midline laparotomy; bright subcostal incision; mini right subcostal incision (Fig. 2.11). In this procedure, the gallbladder is removed through an abdominal incision (usually right subcostal) after the cystic duct and artery are ligated. The procedure is performed for acute and chronic cholecystitis. In some patients a drain may be placed close to the gallbladder bed and brought out through a puncture wound if there is a bile leak. The drain type is chosen based on the physician's preference. A small leak should close spontaneously in a few days with the drain preventing accumulation of bile.

The area is now isolated with packs. If the gallbladder is greatly distended, it is aspirated through the fundus by means of a trocar and cannula attached to a suction apparatus. The neck of the gallbladder is grasped. Then the very important dissection to display the junction of the cystic, the common hepatic and the common bile ducts is started. During the course of this dissection the cystic artery is found, and its relation to the common hepatic artery verified. Cholangiography is performed at this stage both to confirm that the anatomy of the biliary tree has been correctly identified and to check for stones in the main ducts. Only when anatomical and radiological tests are both satisfactory should any duct be divided. The cystic duct is ligated. Forceps are applied to the gallbladder side and the cystic duct is divided. From below and upwards, the gallbladder is dissected from its bed, dividing the peritoneum on the gallbladder. Only after the gallbladder has been removed and hemostasis assured, is the abdominal wall closed (Fig. 2.12).

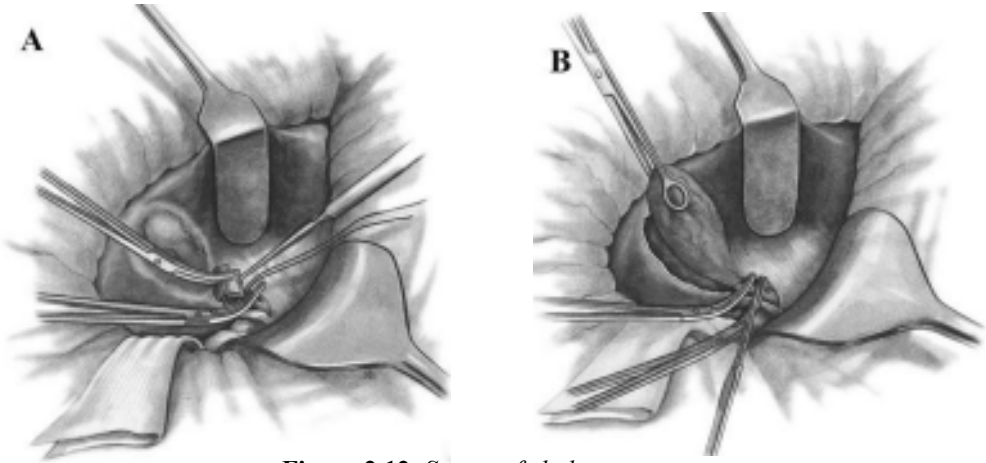


Figure 2.12. *Stages of cholecystectomy*

With severe inflammation in Calot's triangle (bounded above by the liver, medial by the common hepatic duct and below by the cystic duct) it may be wise to open the gallbladder, extract all the stones and bile, and excise as much of the wall of the gallbladder as possible. The cystic duct opening is closed by a catgut suture from 'within' Any mucous membrane remaining on the hepatic side may be diathermied. An alternative is cholecystostomy.

Laparoscopic cholecystectomy (Fig. 2.13). In this procedure four puncture wounds are made in the abdomen, and the gallbladder dissected in the manner described previously. Eventually, the gallbladder is delivered through the umbilical puncture.

This technique is applicable in 85 per cent of patients; its advantage is the rapid speed of recovery.

Contraindication:

- Carcinoma.
- Common bile duct stones and biliary stenosis.
- Severe abdominal infection.
- Operation history.
- Pregnancy.
- Cardiac and respiratory problems.
- Bleeding tendency.

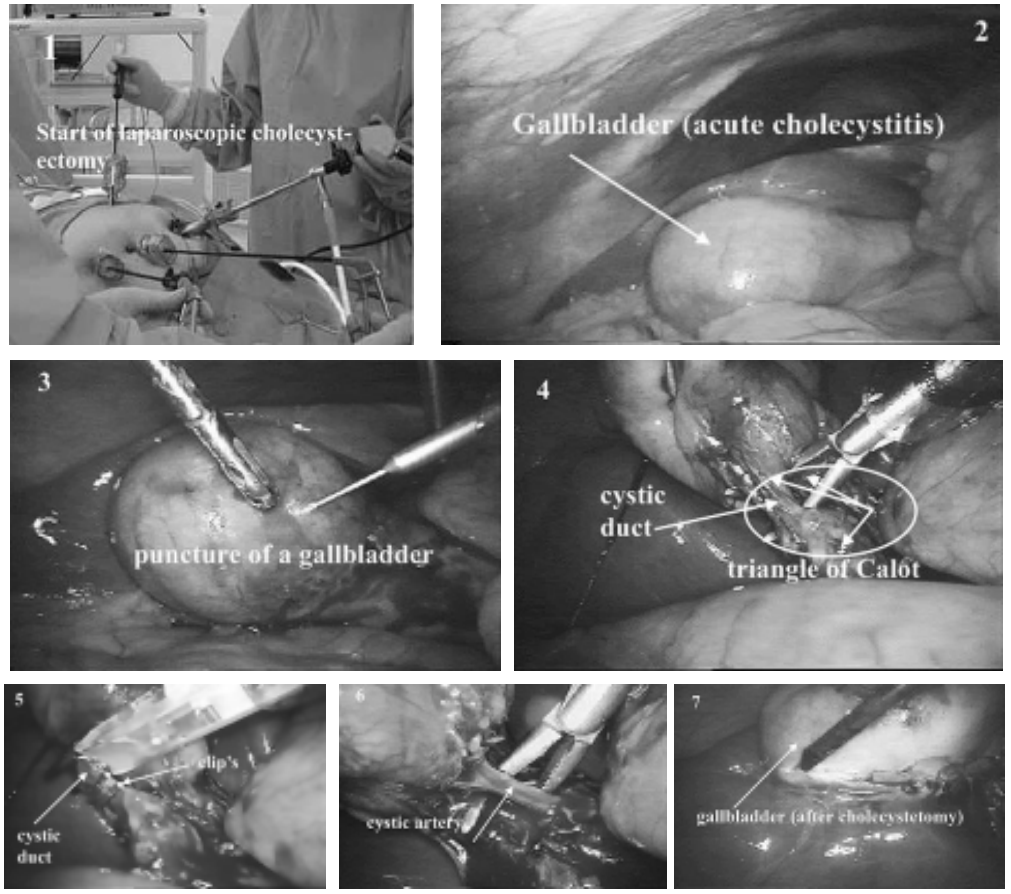


Figure 2.13. *Stages of laparoscopic cholecystectomy*

Conversion to open treatment. Several studies demonstrated that the risk of conversion depends mainly on the degree of inflammation, pathology of gallbladder disease (e.g., thickness of gallbladder wall), age, male sex, and common bile duct diameter. Conversion rate in elective laparoscopic cholecystectomy may be 0% to 15%, but in cases of gangrenous cholecystitis or empyema it may be 50% to 83%. Ultrasound may help to predict the risk of conversion. However, the surgeon has to decide intra-operatively whether to convert to open procedure within a short time. It is necessary to stress that the conversion from laparoscopic cholecystectomy to an open procedure should never be considered as an "adverse outcome" or the surgeon's

failure. The opposite is true – an early decision to convert in face of hostile anatomy, or the failure to progress, reflect solid judgement and understanding that the patient's safety is more important than the surgeon's ego. Clearly, reporting conversion rates without reporting the severity of gallbladder inflammatory disease means nothing.

2.4.5. COMPLICATIONS OF CHOLECYSTECTOMY

- Bile leak ("biloma").
- Bile duct injury (about 5–7 out of 1000 operations. Open and laparoscopic surgeries have essentially equal rate of injuries, but the recent trend is towards fewer injuries with laparoscopy. It may be that the open cases often result because the gallbladder is too difficult or risky to remove with laparoscopy).
- Abscess.
- Wound infection.
- Bleeding (liver surface and cystic artery are most common sites).
- Hernia.
- Organ injury (intestine and liver are at highest risk, especially if the gallbladder has become adherent/scarred to other organs due to inflammation (e.g. transverse colon).
- Deep vein thrombosis/pulmonary embolism (unusual-risk can be decreased through use of sequential compression devices on legs during surgery).
- Fatty acid and fat-soluble vitamin malabsorption.

2.4.6. CHOLECYSTOSTOMY

Percutaneous cholecystostomy has been used in the treatment and diagnosis of acute cholecystitis in patients who are poor risks for any surgical procedure or for general anesthesia. These may include patients with sepsis or severe cardiac, renal, pulmonary, or liver failure. Under local anesthesia, a fine needle is inserted through the abdominal wall and liver edge into the gallbladder under the guidance of ultrasound or computed tomography. Bile is aspirated to ensure adequate placement of the needle, and a catheter is inserted into the gallbladder to decompress the biliary tract.

Open cholecystostomy – is an alternative procedure and is preferred if cholecystectomy is technically hazardous or if the patient is a poor anesthetic risk. Patients too ill for cholecystectomy; are usually older than 65 years, with a compromised cardiovascular system or chronic lung disease. Cholecystostomy is readily performed under local anesthesia. Two stay sutures are inserted on either side of the fundus to steady the organ, the fluid contents of which are aspirated. The fundus is opened and stones are removed from the interior by Desjardins' forceps, aided, always, by a finger milking up a stone or stones from Hartmann's pouch. Minute calculi are often dislodged by strips of dry gauze passed into the interior. A large Foley catheter is placed in the gallbladder and the balloon inflated. The opening in the gallbladder is closed about the tube. The tube is brought through a portion of greater omentum, which is anchored to the gallbladder by the original stay sutures. The catheter is then brought to the surface through a separate stab incision. The abdominal incision is closed, and the catheter connected to a sterile bag (Fig. 2.14).

Figure 2.14. *Stages of open cholecystostomy*

2.5. ACUTE ACALCULOUS CHOLECYSTITIS

Acute acalculous cholecystitis (AAC) may develop without gallstones in critically ill or injured patients, and appears to be increasing in incidence. In addition to injured or postoperative patients, patients with diabetes, malignant tumors, vasculitis, congestive heart failure, and shock or cardiac arrest may develop AAC. Ischemia/reperfusion injury is a central pathogenic feature, but bile stasis, opioid therapy, positive-pressure ventilation, and total parenteral nutrition have all been implicated as co-factors.

The following factors have been associated with acalculous cholecystitis:

- Surgery, particularly abdominal.
- Severe burns.
- Gastroenteritis.
- Severe trauma.
- Total parenteral nutrition.
- Mechanical ventilation.
- Blood transfusion reactions.
- Dehydration.
- Narcotic analgesia.
- Diabetes mellitus.
- Antibiotics, particularly broad spectrum.
- Hepatic arterial embolization (islet cell tumors and hepatocellular carcinoma).
- Postpartum complications.
- Vascular insufficiency and vasculitis, such as systemic lupus erythematosus and Sjцgren syndrome.
- Arteriostenosis/hypertension.
- AIDS, CMV, Cryptosporidium infections.
- Typhoid.

Acute acalculous cholecystitis is especially dangerous during a serious illness or following major surgery. The incidence of AAC appears to be increasing; it is likely that increased awareness and improved imaging studies are identifying more cases. The mortality rate remains about 30% because the diagnosis remains challenging, the affected patients are critically ill, and because the disease itself can progress rapidly due to a high incidence of gangrene (> 50%) and perforation (> 10%).

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2.5.1. PATHOGENESIS

Gallbladder ischemia/reperfusion injury. Gallbladder ischemia/reperfusion injury is a critical factor in the pathogenesis of AAC. Bacterial invasion of ischemic tissue is believed to be a secondary phenomenon; an increasing duration of ischemia increases mucosal phospholipase A_2 and superoxide dismutase activities, and increases mucosal lipid peroxide content. Longer periods of reperfusion produce further increases in mediator activity. The humoral response to gram-negative bacteremia or splanchnic ischemia and mediator release may be of primary importance. Lipopolysaccharide induces a marked host response, including the activation of the coagulation cascades and generation of platelet-activating factor, both of which have been implicated in animal studies of pathogenesis. Numerous observations of clinical low-flow states support this hypothesis, as does the pathologic observation of high rates of gallbladder necrosis and perforation. Gallbladder specimen arteriography reveals marked differences between acute calculous and AAC in humans. Whereas gallstone-related disease is associated with arterial dilatation and extensive venous filling, AAC is associated with multiple arterial occlusions and minimal to-absent venous filling.

Bile Stasis. Volume depletion may lead to concentration and stasis of bile, which can inspissate in the absence of gallbladder emptying. Opioid analgesics induce increased biliary pressure due to spasm of the sphincter of Oddi. Bile stasis may also be induced by positive-pressure mechanical ventilation with positive end-expiratory pressure (PEEP). Bile stasis increases the concentration of lysophosphatidyl choline in bile, which promotes local injury of the gallbladder mucosa by disrupting normal water transport across gallbladder mucosa. Other compounds present in bile, such as beta-glucuronidase, have also been implicated in the pathogenesis of AAC. Long-term therapy with TPN causes bile stasis, and may be associated with an incidence of AAC of up to 30%. Serial gallbladder ultrasound studies in patients on long-term TPN show that the incidence of gallbladder "sludge", only 6% during the first week of TPN, increases to 50% at 4 weeks and 100% at 6 weeks. Unfortunately, periodic stimulation of gallbladder contraction with cholecystokinin does not prevent AAC in critically ill patients, nor does enteral hyperlimentation, which preserves gallbladder motility.

2.5.2. DIAGNOSIS

Most patients with AAC are critically ill, which makes the diagnosis challenging to make. Cholecystitis is but one of many potential causes of systemic inflammatory response syndrome or sepsis that may develop in such patients. Moreover, the differential diagnosis of jaundice in the critically ill patient is complex, and includes intrahepatic cholestasis from sepsis or drug toxicity and "fatty liver" induced by TPN,

in addition to AAC. Rapid and accurate diagnosis is essential, as ischemia can progress rapidly to gangrene and perforation. Acalculous cholecystitis is sufficiently common that the diagnosis should be considered in every critically ill or injured patient with a clinical picture of sepsis and no other obvious source. Physical examination and laboratory studies are too non-specific to be reliable.

2.5.3. CLINICAL FEATURES

Acute cholecystitis is diagnosed on the basis of symptoms and signs of inflammation in patients with peritonitis localised to the right upper quadrant.

Acute inflammation of the gallbladder causes pain, tenderness, and rigidity of the upper right abdomen that may radiate to the midsternal area or right shoulder and is associated with nausea, vomiting, and the usual signs of an acute inflammation. An empyema of the gallbladder develops if the gallbladder becomes filled with purulent fluid.

LABORATORY STUDIES. In the typical patient, there is a mild leukocytosis ranging from 10 000 to 15 000. White blood cell counts greater than 15 000 indicate the development of suppurative complications but are not reliable indicators of complicated cholecystitis.

RADIOLOGIC STUDIES.

X-ray. The main value of plain x-ray examination of the abdomen in acute cholecystitis is to aid in the exclusion of other diseases.

Emphysematous cholecystitis (Fig. 2.15):

1. 4/5 patients are men.
2. 1/3 patients are diabetic.
3. Due to infection or ischemia.
4. Cystic duct may be opened.
5. Gas in lumen/wall/bile ducts.
6. Complications: gangrene and perforation.

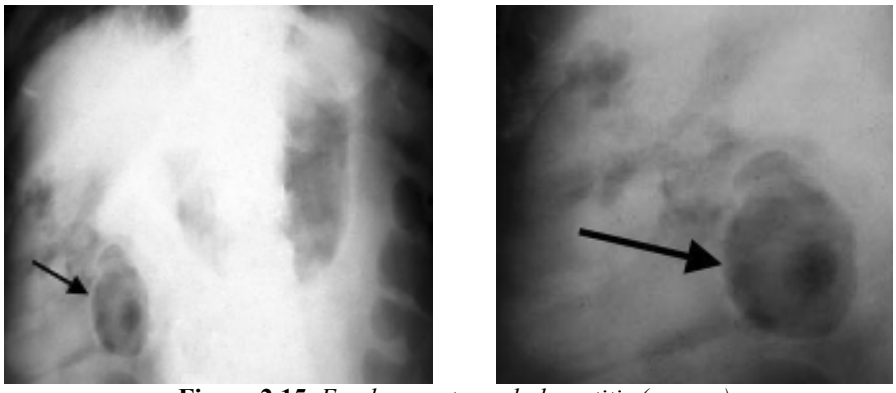

Figure 2.15. *Emphysematous cholecystitis (arrows)*

Ultrasound. Ultrasound of the gallbladder is the most accurate modality to diagnose AAC in the critically ill patient (Fig. 2.16). Thickening of the gallbladder wall is the most reliable criterion. False-positives may occur when conditions including sludge, nonshadowing stones, cholesterolosis, hypoalbuminemia, or ascites mimic a thickened wall. Other helpful ultrasonographic findings for AAC include

pericholecystic fluid, or the presence of intramural gas or a sonolucent intramural layer or "halo" that represents intramural edema.

US: thickened gallbladder wall or edema; pericholecystic fluid; sonographic Murphy's sign.

CT. Computed tomography is as accurate as ultrasound in the diagnosis of AAC (Fig. 2.17). Appropriate criteria for diagnosis of AAC by CT are similar to the criteria described for ultrasound. Only a single retrospective study has compared all three modalities (ultrasonography, hepatobiliary scanning, and CT); ultrasonography and CT were comparably accurate and superior to hepatobiliary imaging. Comparable accuracy, low cost and bedside availability make ultrasonography the diagnostic modality of choice for AAC. Preference may be given to CT if other abdominal pathology is considered more likely.

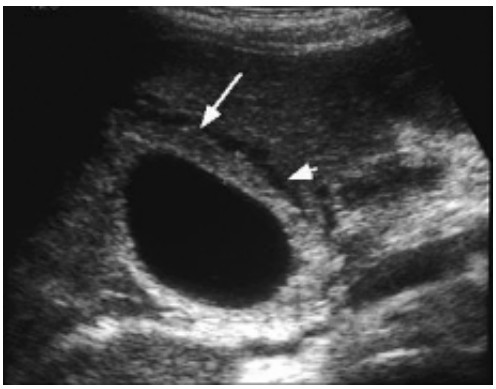

Figure 2.16.
Acute acalculous cholecystitis (arrows)

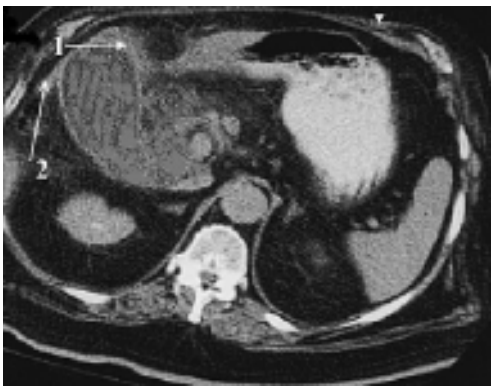

Figure 2.17. *CT scan of acute acalculous cholecystitis: 1 – denotes the gallbladder wall thickening; 2 – denotes the fluid around the gallbladder*

Laparoscopy. Laparoscopy has been reported to be successful for both the diagnosis and therapy of AAC, although reports are limited to small series and there has been no randomized trial. Laparoscopy can be performed under local anesthesia and intravenous sedation at the bedside, and may be advisable to attempt if open surgical drainage is otherwise contemplated. Laparoscopy is possible in patients who have undergone recent abdominal surgery if "gasless" techniques are used. Diagnostic accuracy is high, and both laparoscopic cholecystostomy and cholecystectomy have been performed.

2.5.4. TREATMENT

Conservative treatment:

- Intravenons fluid and electrolyte replacement.
- Nasogastric suction.
- Systemic antibiotics.
- Parenteral analgesia.

Surgical methods: open cholecystectomy or laparoscopic cholecystectomy.

2.5.5. MORTALITY/MORBIDITY

Mortality associated with acute cholecystitis is 5–10%; death mostly occurs in patients older than 60 years.

Acute cholecystitis may be complicated by empyema, gangrenous cholecystitis, gallbladder perforation, pericholecystic abscess, and bilioenteric fistula. Gangrenous cholecystitis is a frequent cause of gallbladder perforation. Suppurative complications are more frequent in the elderly. Most localized perforations may be satisfactorily treated by means of surgery.

Although free intraperitoneal perforation is rare, it is associated with a mortality of 25%. Necrosis of the gallbladder wall occurs in about 60% of cases of acalculous cholecystitis because gangrene and perforation are frequent occurrences. Mortality ranges from 9–66%.

The higher mortality in acute acalculous cholecystitis has been attributed to delayed diagnosis and comorbidities. The morbidity associated with emphysematous cholecystitis is higher, because of GB wall gangrene and perforation.

Recurrent symptoms are common in patients with acute cholecystitis who are treated expectantly; most patients need elective cholecystectomy.

Chapter 3

ACUTE PANCREATITIS

HISTORICAL

Acute Pancreatitis is an inflammatory disease of the pancreas. The pancreas was given its name at the end of I to the beginning of the II centuries our era, when **Rufus** entered into literature; the term "PANCREAS" (from the Greek – "pan" = all; "creas" = meat).

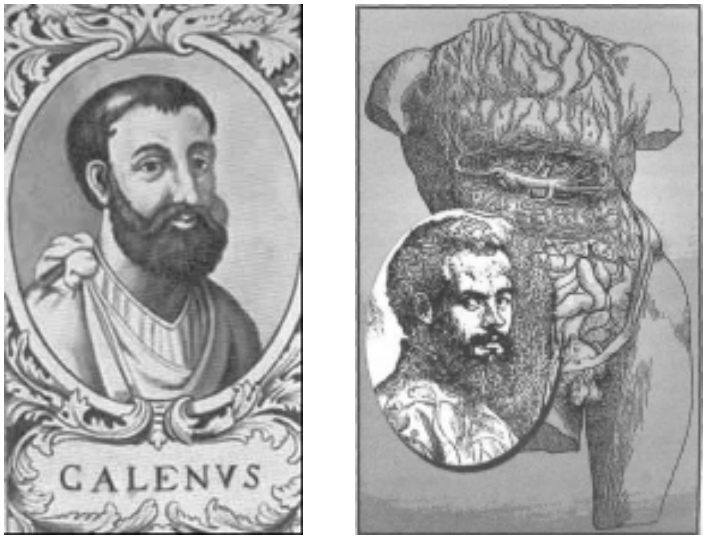

Figure 3.1. *Claudia Galen and Andreas Vesalius*

Andreas Vesalius referred to the pancreas in the fifth book of his opus as a "glandulous organ" and presumed the pancreas to exert a protective effect on the stomach by serving as a cushion upon which it rested. Andreas Vesalius (1514 to 1564) was one of the first anatomists to depict the pancreas (Fig. 3.1)

3.1. ANATOMY

The pancreas develops as two buds (ventral and dorsal) from the primitive duodenum. The accessory pancreatic duct (Santorini) is the duct of the dorsal bud. It persists as the main duct of the body and tail of the pancreas and passes anteriorly through the head of the pancreas to enter the second part of the duodenum at the accessory papilla. The ventral bud duct, which forms the main pancreatic duct in the head of the pancreas, fuses with the dorsal duct in the pancreatic head and then runs posteriorly and inferiorly to lie next to, and eventually join with, the bile duct at the ampulla of Vater.

Pancreatic acinar tissue is organised into lobules. The main duct ramifies into interlobular and intralobular ducts, ductules and finally acini. Acinar tissue comprises about 84 per cent of the pancreas, duct cells and blood vessels 4 per cent, and endocrine cells (the islets of Langerhans) about 2 per cent. The rest is connective tissues, and fat. The islets of Langerhans are distributed throughout the pancreas. Islets consist of differing cell types: 75 per cent are B cells (producing insulin), 20 per cent A cells (glucagon), D cells (somatostatin), and a small number of pancreatic polypeptide-secreting cells. Within

an islet, the B cells form an inner core surrounded by the other cells. Capillaries draining the islet cells drain into the portal vein, forming a pancreatic portal system.

The pancreas is a retroperitoneal, organ. Composed of head, body, and tail. Superior mesenteric vein and artery lie behind neck of pancreas. The average weight of the pancreas is 85 g, and the usual length is 12–15 cm. The normal anteroposterior thickness of the head is less than 2.5 cm; the neck, 1.5 cm; the body, 2 cm; and the tail, 2.5 cm. Duct of Wirsung is major duct, 3–4 mm diameter, joins common bile duct at papilla of Vater. Minor duct is Santorini; 5–10% has Santorini as major drainage of pancreas, with vestigial Wirsung. The head of the pancreas lies over the aorta and under the stomach and transverse colon. The common bile duct courses posteriorly to the head of the pancreas and partially within it (Fig. 3.2).

Arterial supply to head is pancreaticoduodenal arcades from gastroduodenal artery. Splenic, inferior pancreatic arteries supply body (Fig. 3.3). Venous drainage closely parallels arterial supply (Fig. 3.4).

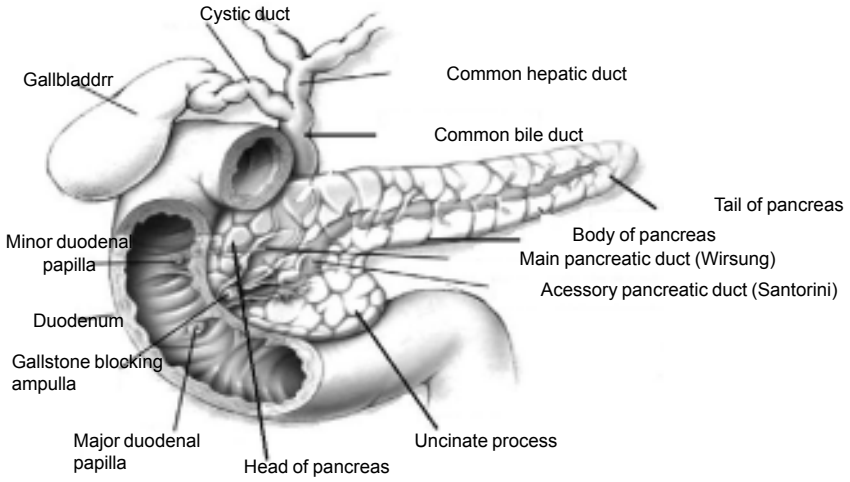

Figure 3.2. *Anatomy of pancreas*

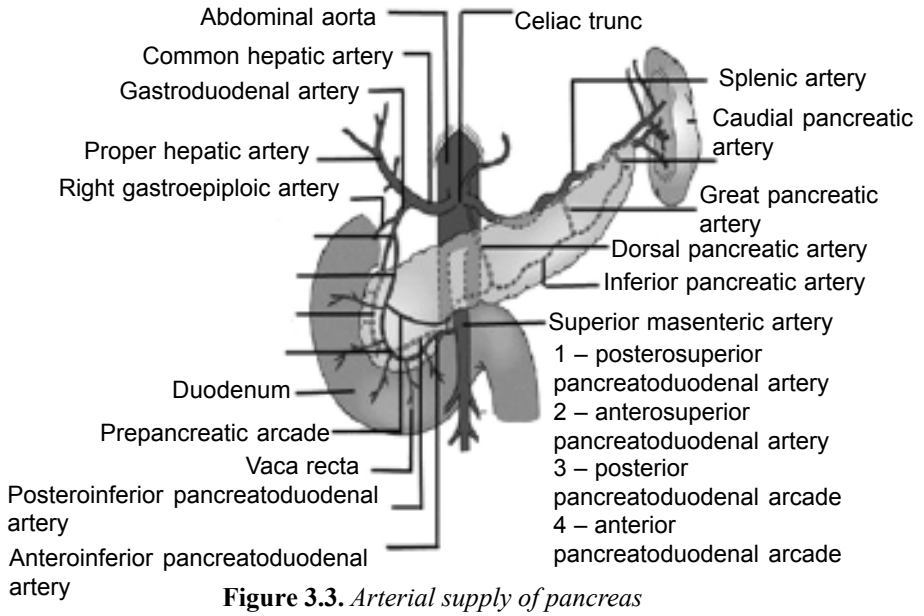

Figure 3.3. *Arterial supply of pancreas*

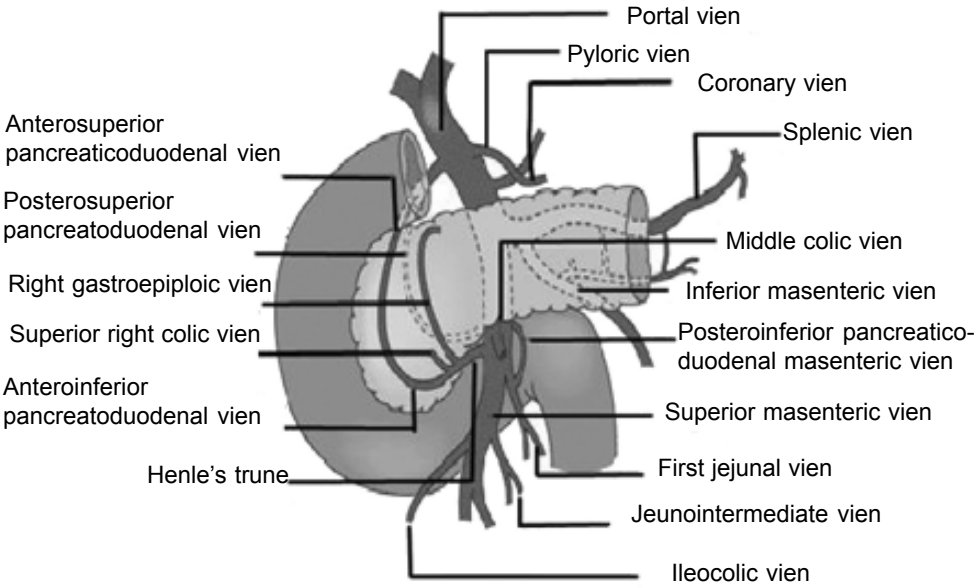

Figure 3.4. *Venous drainage of pancreas*

Lymphatic drainage is diffuse. Rich periacinar network that drain into 5 nodal groups: superior nodes; anterior nodes; inferior nodes; posterior PD nodes; splenic nodes.

Innervation of Pancreas:

- Sympathetic fibers from the splanchnic nerves.
- Parasympathetic fibers from the vagus.
- Both give rise to intrapancreatic periacinar plexuses.
- Parasympathetic fibers stimulate both exocrine and endocrine secretion.
- Sympathetic fibers have a predominantly inhibitory effect.

3.2. PHYSIOLOGY

The pancreas is made up of two tissues: 1) exocrine tissue and 2) endocrine tissue (Fig. 3.5).

Exocrine tissue. In response to a meal, the pancreas secretes digestive enzymes in an alkaline (pH 8.4), bicarbonate-rich fluid. Acinar cells synthesize and secrete digestive enzymes while the duct cells secrete bicarbonate. The daily secretion is about 1 litre, containing 5–8 g of protein in the form of enzymes.

This secretion is under both neural and hormonal control. Stimulation of parasympathetic nerves in the vagus results in secretion of bicarbonate and enzymes, whereas stimulation of splanchnic sympathetic nerves inhibits secretion. The rate of secretion and the bicarbonate content of the pancreatic juice are increased by the hormone secretin, which is released from the duodenal mucosa by luminal acid stimulation. Pancreatic enzyme secretion is potently stimulated by pancreozymin, which is released from the duodenal mucosa by luminal fat and peptides. The pancreas is made of four major cell types:

- Alpha (A) cells secrete glucagon.
- Beta (B) cells secrete insulin.

- Delta (D) cells secrete somatostatin.
- F cells secrete pancreatic polypeptide.

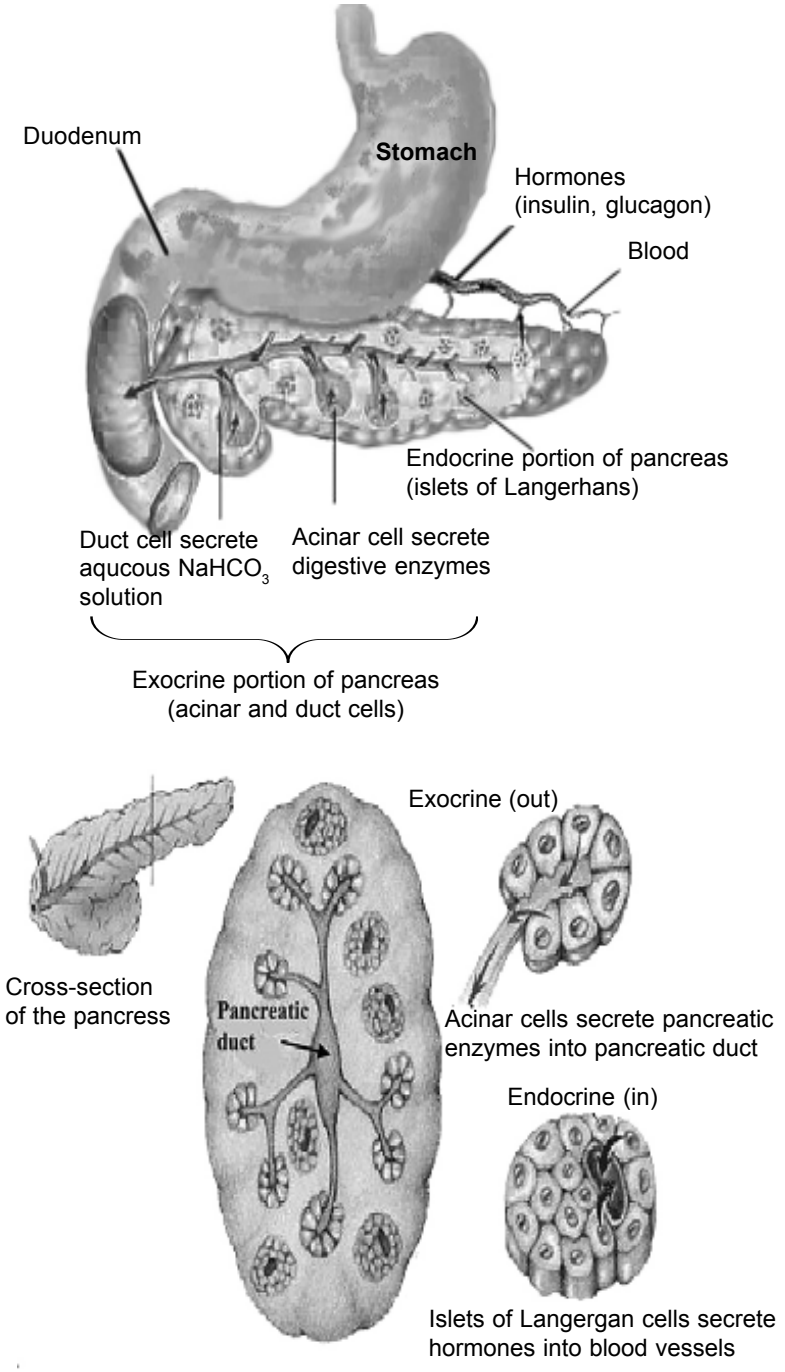

Figure 3.5. *Physiology of pancreas*

Pancreatic secretion can also be stimulated by hormones produced within the pancreas and small bowel, vasoactive intestinal peptide (VIP) and gastrin; and inhibited by the pancreatic hormones somatostatin, pancreatic polypeptide and glucagon.

Pancreatic secretion consists of a cephalic phase, initiated by the thought of food, a gastric phase produced by food in the stomach, and an intestinal phase mediated by secretin and pancreozymin release from the duodenum and jejunum. About 20 digestive enzymes are produced: **proteolytic enzymes** – trypsin; **lipolytic enzymes** – lipase; **starch-splitting enzymes** – amylase and **nucleic acid splitting enzymes** – ribonuclease. Activation by **enterokinaze.**

Endocrine tissue. The endocrine tissue, which consists of the islets of Langerhans, secretes hormones into the bloodstream. The hormones secreted by endocrine tissue in the pancreas are insulin and glucagon (which regulate the level of glucose in the blood), and somatostatin (which prevents the release of other two hormones).

3.3. ETIOLOGY AND PATHOGENESIS

The etiology and pathogenesis of acute pancreatitis have been intensively investigated. The etiology of acute pancreatitis is a complex subject because many different factors have been implicated in the causation of this disease, and sometimes there are no identifiable causes. Two factors, alcoholism and biliary tract stone disease, account for 80 to 90% of the cases (Fig. 3.6). The remaining 10 to 20 % is accounted for either by idiopathic disease or by a variety of miscellaneous causes including trauma, surgery, drugs, heredity, infection and toxins.

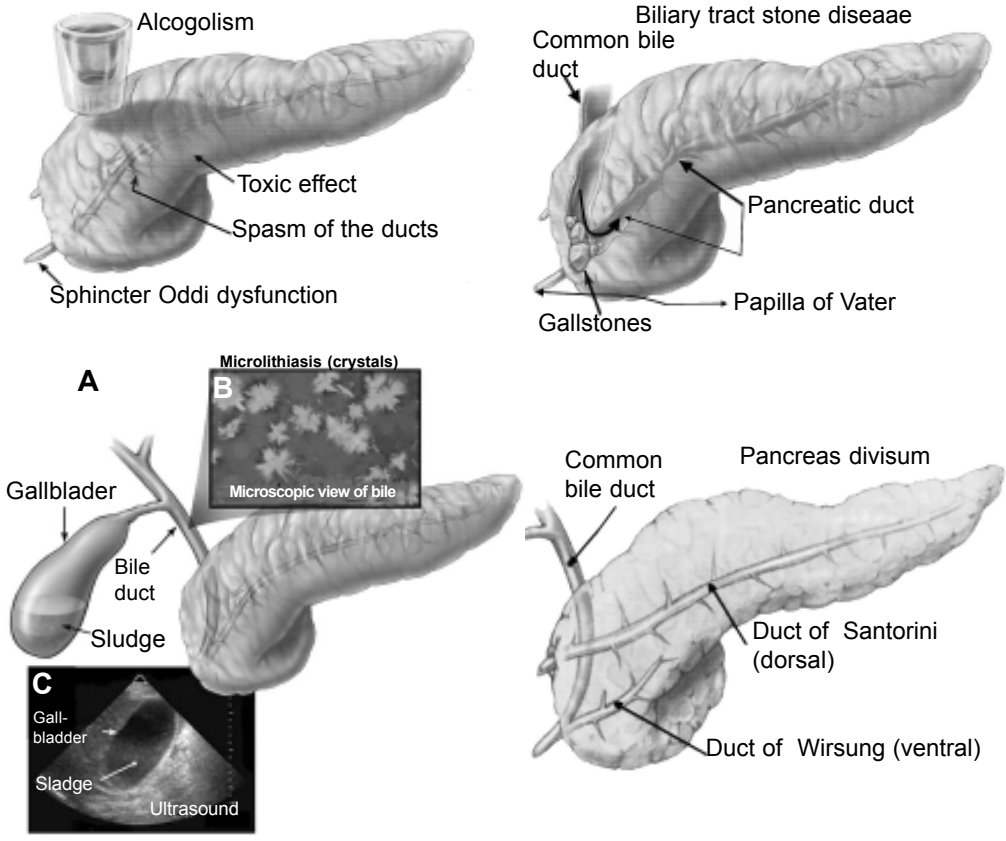

Figure 3.6. *Etiology – alcoholism, biliary tract stone disease and pancreas divisum*

Gallstone-induced pancreatitis is caused by duct obstruction by gallstone migration. Obstruction is localized in the bile duct and pancreatic duct, or both. Duct obstruction promotes pancreatitis by increasing duct pressure and subsequent unregulated activation of digestive enzymes. Biliary sludge refers to a viscous bile suspension that contains cholesterol crystals and calcium bilirubinate granules embedded in strands of gallbladder mucus. Sludge is associated with bile stasis, long-lasting fast, distal bile duct obstruction, and total parenteral feeding (Fig. 3.6). Most patients with biliary sludge are asymptomatic. Biliary sludge is commonly seen in patients with recurrent acute pancreatitis of unknown origin, and cholecystectomy might prevent the recurrence of pancreatic disease.

Alcohol abuse is the second most frequent cause of acute pancreatitis, but the correlation between alcohol and pancreatitis is not completely understood. In experimental models, Gorelick showed that ethanol directly sensitizes acinar cells to cholecystokinin stimulation. As the development of pancreatitis might be affected by both genetic and environmental factors, failure to inhibit trypsin activity or to wash active trypsin into pancreatic ducts might promote alcoholic pancreatitis. In fact, the exact mechanism underlying alcoholic acute pancteatitis has not been extensively elucidated.

Pancreas divisum, a common congenital anatomical variant of the pancreatic duct in about 7% of autopsy series, results from the absence of fusion between the dorsal and ventral ductal systems (Fig. 3.6).

The possible consequence of pancreas divisum is a stenosed or inadequately patent minor papilla, preventing normal drainage of pancreatic secretions and leading to increased intraductal pressure. However, whether pancreas divisum is related to pancreatitis is highly controversial. Whether dysfunction of sphincter of Oddi can trigger acute pancreatitis by increasing intrapancreatic duct pressure is also controversial.

Intraduct papillary mucinous tumor might be another cause of acute pancreatitis. Tumor or mucus produced by it obstructs the main pancreatic duct and its side branch, or both. Logically, the consequence is increased pancreatic duct pressure caused by pancreatic hyperstimulation and pancreatic duct obstruction. Thus, these tumors might trigger acute pancreatitis through the same mechanisms underlying acute biliary pancreatitis.

Endoscopic retrograde cholangiopancreatography (ERCP) is a potential cause of acute pancreatitis. Asymptomatic hyperamylasaemia occurs in 35–70% of patients after the procedure. ERCP has a higher risk of inducing acute pancreatitis when it is performed to treat Oddi sphincter dysfunction than to remove gallstones in the bile duct. Other risk factors for post-ERCP pancreatitis include young age, female sex, number of attempts to cannulate papilla, and poor emptying of pancreatic duct after opacification. Prevention of post-ERCP pancreatitis in high-risk patients might be achieved by placing a temporary pancreatic stent.

Hypercalcaemia is another rare and inconsistent cause of acute pancreatitis. Because the incidence of pancreatitis is low in patients with chronic hypercalcaemia, additional factors are probably needed to induce pancreatitis. Drugs rarely induce acute pancreatitis. Cases of drug-induced pancreatitis have been reported. Many infectious agents are associated with acute pancreatitis, but no microorganism has ever been identified within the pancreas. However, it was reported that acute pancreatitis is associated with viral or bacterial infections, and infestation with parasites.

SUMMARY – CAUSES OF ACUTE PANCREATITIS:

1. Ethanol abuse.
2. Biliary diseases: gallstones; choledocholithiasis; biliary sludge; microlithiasis; mechanical/structural injury: sphincter of Oddi dysfunction; pancreas divisum; trauma; postendoscopic retrograde cholangiopancreatography; pancreatic malignancy.
3. Medications: azathiprine/6-mercaptopurine; dideoxyinosine; pentamidine; sulfonamides; thiazide diuretics; ACEI.
4. Metabolic: hypertriglyceridemia; hypercalcemia.
5. Infectious: viral; bacterial; parasitic.
6. Vascular: vasculitis.
7. Genetic predisposition.
8. Idiopathic.

Under physiologic conditions, the pancreas synthesizes a large amount of protein. A majority of these proteins consist of digestive enzymes. Because the exocrine pancreas produces several enzymes that are potentially injurious to itself, it prevents autodigestion by intracellularly assembling the inactive precursors of these enzymes, called proenzymes or zymogens, which are then transported and secreted outside of the gland (Fig. 3.7). Their activation occurs safely in the duodenum, where the brush-border enzyme enteropeptidase (or enterokinase) activates the trypsinogen, and the resulting trypsin then activates the other zymogens in a cascade reaction. To further protect the pancreas from these potentially harmful digestive enzymes, they are segregated from the cytoplasmic space within acinar cells by being enclosed within membrane-bound organelles, referred to as zymogen granules. Another layer of protection is provided by the synthesis of trypsin inhibitors, which are transported and stored along with the digestive enzyme zymogens. These are available to inhibit small amounts of prematurely activated trypsinogen within pancreatic acinar cells. It is generally theorized that acute pancreatitis occurs when this process goes awry and the gland is injured by the erroneously activated enzymes that it produces. There are three reasons for this theory: (a) the pancreas is digestible by the activated enzymes of the duodenum; (b) activated digestive enzymes are found within the pancreas during pancreatitis, and (c) the histology of pancreatitis is suggestive of a coagulative necrosis.

When the pancreas becomes damaged or the ducts become blocked, the trypsin inhibitor accumulates and activates the pancreatic secretions that escape into the surrounding tissue, resulting in inflammation, thereby causing acute pancreatitis.

Release of kallikrein and chymotrypsin results in increased capillary membrane permeability, leading to leakage of fluid into the interstitium and development of edema and relative hypovolemia. Elastase is the most harmful in terms of direct cell damage, it causes dissolution of the elastic fibers of blood vessels and cuts, leading to hemorrhage. Phospholipase A_2 in the presence of bile destroys phosholipids of cell membranes causing severe pancreatic and adipose tissue necrosis. Lipase flows into damaged tissue and is absorbed into systemic circulation, resulting in fat necrosis of the pancreas and surrounding tissues.

It has become apparent that there are two phases of acute pancreatitis: an early phase (within the first or second week of onset) and a second phase occurring after the first or second week of onset of the disease. During the first phase, the severity is

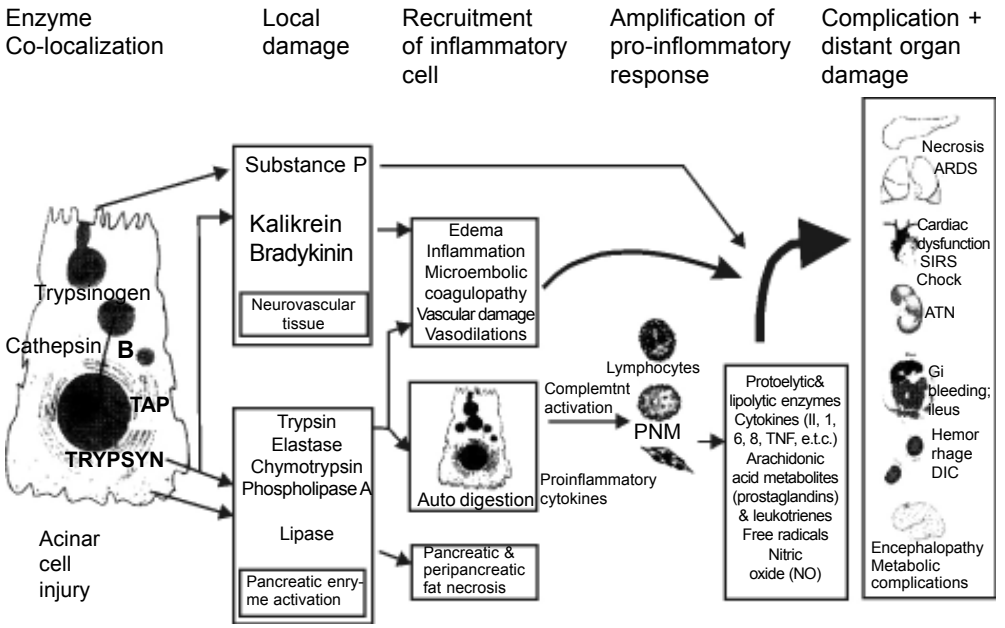

Figure 3.7. *Pathophysiology of acute pancreatitis*

defined by organ failure that persists for>2 days (persistent organ failure), or by death. Organ failure is secondary to the host's systemic inflammatory response elicited by the tissue injury/cytokine response and not necessarily related to the extent of necrosis. Local or systemic infection is usually not yet present or involved in the systemic response (Fig. 3.7). During the second phase, the severity is defined by persistent organ failure, by complications of the pancreatitis that develop in the pancreatic parenchyma and peripancreatic tissues, or by death.

During the first phase, the pancreatic/peripancreatic conditions evolve dynamically; this process evolves from the initial state of inflammation and variable degrees of pancreatic and peripancreatic ischemia and/or edema either to resolution or to irreversible necrosis and liquefaction, and/or further development of collections containing fluid and/or solid material in and around the pancreas. The extent of the pancreatic and peripancreatic changes is usually but not always directly proportional to the severity of organ failure. Over the first week or so, organ failure related to the systemic inflammatory response either resolves or becomes more severe.

In the second phase, the disease either resolves (edematous pancreatitis without necrosis) or tends to stabilize (but not normalize) or progress and enter into a more protracted course lasting weeks to months related to the necrotizing process-necrotizing pancreatitis. Also, during this second phase, changes in the pancreatic/peripancreatic morphology occur much more slowly. Mortality in the second phase is usually related to persistent organ failure and/or to complications of necrotizing pancreatitis. Severity of the overall disease process in this second phase is defined not only by persistent organ failure, but also by the development of complications of necrotizing pancreatitis requiring active intervention (operative, endoscopic, laparoscopic, and/or percutaneous), or requiring other supportive measures (such as the need for ventilator support, renal dialysis, or nasojejunal feedings), leading to prolonged hospitalization, or by death.

These two phases have a distinct pathophysiology. Because the need for treatment in the first phase is determined more by the presence or absence of organ failure and less by morphologic findings in and around the pancreas, one should apply "clinical" parameters for its classification of severity and its treatment. In contrast, in the second phase of the disease, the need for treatment is determined by the presence of symptoms and/or complications of necrotizing pancreatitis. The type of treatment is determined mainly by the morphologic abnormalities of the pancreatic/peripancreatic region as seen on the most readily available imaging test (contrast enhanced computed tomography – CECT) and by the presence/absence of local complications which may manifest systemically, such as infection of necrotic tissues giving rise to bacteremia and sepsis.

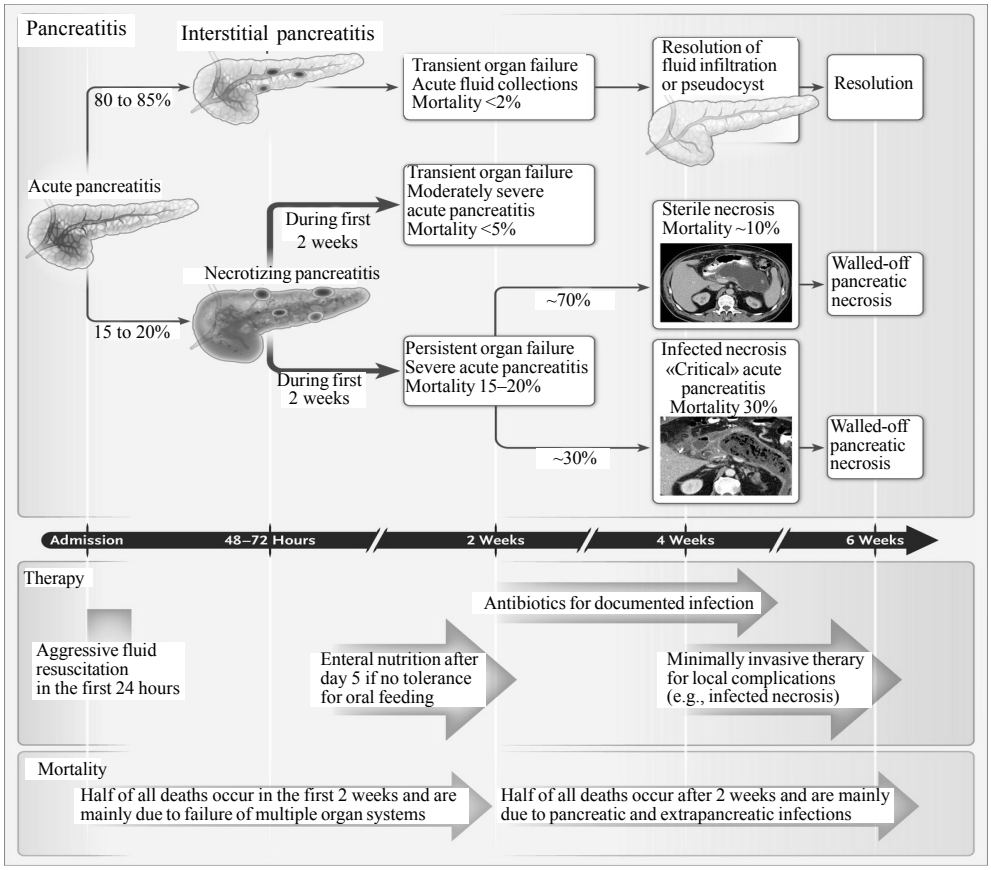

Figure 3.8. *The forms of acute pancreatitis (Chris E. Forsmark et al. The New England Journal of Medicine. November 30, 2016. P. 1972-1981)*

3.4. CLASSIFICATION AND DEFINITIONS

Mostly the Atlanta classification (1992) is used (H.G. Beger's et al., 1991): divided into mild and severe acute pancreatitis and the forms of disease:

1. Interstitial edematous pancreatitis.
2. Pancreonecrosis:
   a) non-infected and b) infected.

3. Abscess of pancreas.
4. Pseudocyst of pancreas:
   a) non-infected and b) infected.

In 2012, experts reconsidered the classification of Atlanta and offered the following definitions **3rd revision based** on worldwide review/suggestions (Table 3.1).

Table 3.1 – Acute pancreatitis – comparison of classification schemes

Atlanta Classification (1992)	**New Classification (2012)**
ACUTE PANCREATITIS :	
Interstitial pancreatitis Sterile necrosis Infected necrosis	Interstitial edematous pancreatitis (IEP) Necrotizing pancreatitis: *Pancreatic necrosis with peripancreatic necrosis:* ➢ Sterile and Infected necrosis. *Pancreatic necrosis alone:* ➢ Sterile and Infected necrosis. *Peripancreatic necrosis alone:* ➢ Sterile and Infected necrosis.
(< 4 weeks after onset of pancreatitis) Acute fluid collection.	**(< 4 weeks after onset of pancreatitis)** *Acute peripancreatic fluid collection (APFC):* ➢ Sterile and Infected. *Acute post-necrotic collection (APNC)* : *Pancreatic necrosis with peripancreatic necrosis:* ➢ Sterile and Infected. *Pancreatic necrosis alone:* ➢ Sterile and Infected. *Peripancreatic necrosis alone:* ➢ Sterile and Infected
(> 4 weeks after onset of pancreatitis) Pancreatic pseudocyst. Pancreatic abscess.	**(> 4 weeks after onset of pancreatitis)** *Pancreatic pseudocyst:* ➢ Sterile and Infected *Walled-off necrosis (WON)* *Pancreatic necrosis with peripancreatic necrosis:* ➢ Sterile and Infected. *Pancreatic necrosis alone:* ➢ Sterile and Infected. *Peripancreatic necrosis alone:* ➢ Sterile and Infected.

INTERSTITIAL EDEMATOUS PANCREATITIS (IEP). Contrast enhanced computed tomography (CECT) in patients with IEP demonstrates diffuse or localized enlargement of the pancreas and normal, homogeneous enhancement of the pancreatic parenchyma (Fig. 3.9).

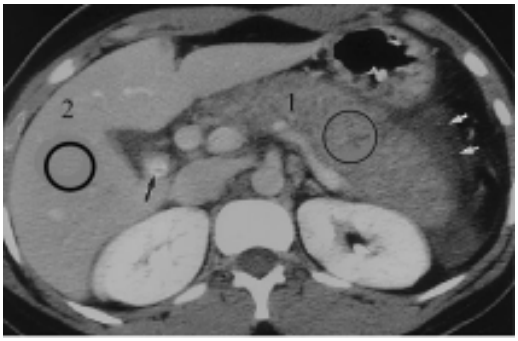

Figure 3.9. *Gallstone-induced pancreatitis. Transverse CT scan obtained with intravenous and oral contrast material reveals a large, edematous, homogeneously attenuating (73-HU) pancreas (1) and peripancreatic inflammatory changes (white arrows). Although the attenuation values are low, there is no pancreatic necrosis. Calcified gallstones are seen in gallbladder (black arrow). 2 – liver (140 HU)*

Similarly, the retroperitoneal and peripancreatic tissues usually appear normal or show mild inflammatory changes in the peripancreatic soft tissues characterized by haziness or mild stranding densities and varying amounts of non-enhancing areas of low attenuation (peripancreatic fluid, see below, pancreatic and peripancreatic collections); the presence of non-enhancing areas of variable density (solid components) in these fluid collections is indicative of peripancreatic necrosis, excludes the diagnosis of IEP, and the process should be termed acute necrotizing pancreatitis with peripancreatic necrosis alone (see below). On occasion, an early contrast enhanced computed tomography (done within the first several days of onset of pancreatitis) exhibits diffuse heterogeneity in pancreatic parenchymal enhancement which cannot be characterized definitively as IEP or patchy necrosis; with these findings, the presence or absence of pancreatic necrosis may have to be classified initially as indeterminate. A contrast enhanced computed tomography done 5–7 days later should allow definitive classification.

NECROSIS. Acute necrotizing pancreatitis has three forms: pancreatic parenchymal and associated peripancreatic necrosis (most common), pancreatic parenchymal necrosis alone (rare), and peripancreatic necrosis alone (about 20% of patients). Thus, it is important to determine the absence of necrosis (interstitial edematous pancreatitis) or the presence of necrosis (necrotizing pancreatitis), the site(s) of necrosis (pancreatic parenchyma and peripancreatic tissue, pancreatic parenchyma alone, or peripancreatic tissue alone), and the absence of infection (sterile necrosis) or the presence of infection (infected necrosis). Necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues. The presence of necrosis in either the pancreatic parenchyma or the peripancreatic tissues defines the process as necrotizing pancreatitis and differentiates necrotizing pancreatitis from IEP. Necrotizing pancreatitis involves one of three types, either pancreatic parenchymal necrosis with peripancreatic necrosis, pancreatic parenchymal necrosis alone, or peripancreatic necrosis alone (without any discernable pancreatic parenchymal necrosis).

PANCREATIC NECROSIS. About 80% of patients with necrotizing pancreatitis have a variable extent of pancreatic parenchymal necrosis on contrast enhanced computed tomography evident by the lack of parenchymal enhancement with intravenous contrast. Most commonly, pancreatic necrosis is associated with a variable extent of peripancreatic necrosis, but on occasion, pancreatic necrosis alone will be seen. The contrast enhanced computed tomography may demonstrate only minimal gland enlargement or diffuse or localized enlargement of the pancreas with one or more areas of non-enhancing pancreatic parenchyma. The extent of pancreatic parenchymal necrosis is quantified in three categories: < 30%, 30–50%, and > 50% of the total pancreatic parenchyma (Fig. 3.10).

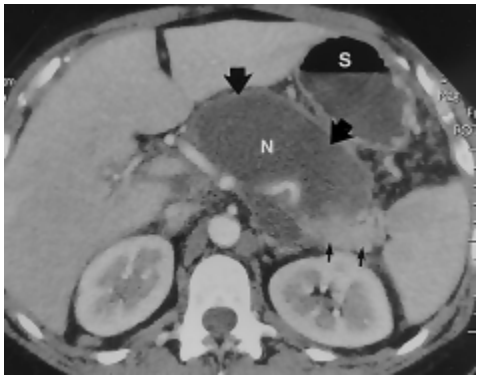

Figure 3.10.
Severe acute pancreatitis
Transverse CT scans obtained with intravenous and oral contrast material reveal an encapsulated fluid collection associated with liquefied necrosis (large straight arrows) in the body of the pancreas. The head, part of the body, and the tail of the pancreas are still enhancing (small straight arrows).
N = liquefied gland necrosis, S – stomach

The presence of pancreatic parenchymal non-enhancement differentiates pancreatic necrosis from IEP and from peripancreatic necrosis alone. The appearance of a limited area of pancreatic parenchymal necrosis estimated to be < 30% of the gland may, on follow-up imaging, prove to be due to fluid within the pancreas rather than necrosis. Therefore, estimates of pancreatic necrosis of < 30% on the initial contrast enhanced computed tomography are less reliable to establish a diagnosis of pancreatic necrosis. A follow-up contrast enhanced computed tomography 4–6 days later depending on the clinical situation would be required to distinguish IEP from pancreatic necrosis when the estimate for pancreatic necrosis is <30% on the initial contrast enhanced computed tomography; an MRI may be preferred if facilities are available.

PERIPANCREATIC NECROSIS ALONE. The presence or absence of necrosis in the peripancreatic tissues is difficult to confirm by contrast enhanced computed tomography early in the course of the disease. contrast enhanced computed tomography suggests the presence of peripancreatic necrosis when there are non-homogeneous non-enhancing areas of variable density containing solid components in one or more areas, especially in the regions of the lesser sac and retroperitoneum. The necrotic area(s) may be exclusively extrapancreatic (peripancreatic necrosis alone) with no recognizable areas of pancreatic parenchymal necrosis on contrast enhanced computed tomography; this entity is recognized in up to 20% of the patients who require operative or interventional management of necrotizing pancreatitis. This distinction proves important clinically, because patients without recognizable pancreatic gland necrosis have a better prognosis and outcome than patients with pancreatic parenchymal necrosis, but more morbidity than interstitial edematous pancreatitis. The Atlanta Classification had no way to subclassify this unique group of patients. If concern is great enough, MRI or ultrasonography may aid in the recognition of solid components within the peripancreatic "fluid" collection.

Infection. Sterile necrosis and infected necrosis are distinguished according to the absence or presence of infection in the non-enhancing pancreatic and/or peripancreatic area(s). Distinction between sterile and infected necrosis is very important clinically, because the presence of infection confers a different natural history, prognosis, and approach to treatment. Patients with sterile necrosis usually do not require intervention unless they remain persistently unwell with ongoing pain, anorexia, early satiety, vomiting, fever, and/or inability to resume oral intake by 4 or more weeks after onset of acute pancreatitis. Infection can be diagnosed definitively only by percutaneous, image guided, fine-needle aspiration (FNA) with a positive Gram

stain and culture. The presence of infection can be presumed when there is extraluminal gas in the non-enhancing area(s) on contrast enhanced computed tomography, usually a pathognomonic sign, which reflects the presence of a gas-forming organism without or with perforation (a rare event) of an adjacent hollow viscus. FNA has a false-negative rate of about 10%. Therefore, a negative FNA should be repeated after an appropriate interval, such as 5–7 days, if a clinical suspicion of infection persists. It must be recognized that proof of infection preoperatively in the absence of extraluminal gas requires image-guided, fine needle aspiration; not all patients with necrotizing pancreatitis, however, require FNA; indeed, FNA should be reserved for the persistently ill patient in whom infection is suspected based on the clinical findings or imaging-based findings.

PANCREATIC AND PERIPANCREATIC COLLECTIONS. Acute pancreatitis can be associated with pancreatic and peripancreatic collections (Fig. 3.11).

IEP can be associated with acute peripancreatic collections and pancreatic pseudocysts (Fig. 3.12). In contrast, the three forms of necrotizing pancreatitis can be associated with pancreatic and peripancreatic collections, including acute peripancreatic fluid collections, pancreatic pseudocysts, acute post-necrotic collections, and walled-off necrosis.

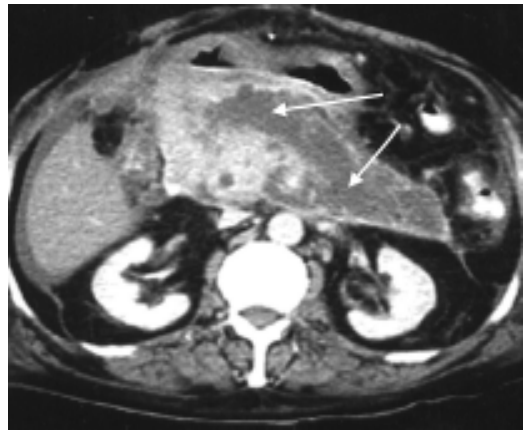

Figure 3.11. *Severe acute pancreatitis. Fluid collection replacing pancreatic body and tail (white arrows)*

ACUTE PERIPANCREATIC FLUID COLLECTION (APFC) (1st 4 weeks after onset of IEP): a) sterile and b) infected.

These fluid collections arise within a few days of onset of IEP and may persist for several weeks. They have no solid components and result from parenchymal and/or peripancreatic inflammation. APFCs exist predominantly adjacent to the pancreas, have no definable wall, and are confined by the normal peripancreatic fascial planes, primarily the anterior pararenal fascia. These APFCs should be differentiated from ascites. In contrast, apparent "fluid" collections that replace pancreatic parenchyma should be considered to represent necrosis. APFCs arise presumably from rupture of a small, peripheral, pancreatic ductal side branch, or they result from local edema related to the pancreatic inflammation and have no connection with the ductal system.

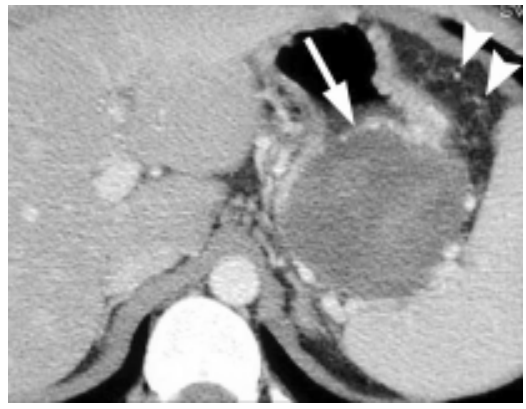

Figure 3.12. *Pancreatic pseudocyst. Image demonstrates a pseudocyst (arrow) in the tail of the pancreas surrounded by a thick enhancing wall. The lesion appears heterogeneous with central areas of higher attenuation, which is suggestive of fresh hemorrhage. Note infiltration (arrow heads) of the peripancreatic fat*

These APFCs do not necessarily reflect pancreatic parenchymal tissue necrosis or even a minor or major ductal disruption.

Most APFCs remain sterile and are reabsorbed spontaneously within the first several weeks after onset of acute pancreatitis. Intervention at this setting for these collections is usually not necessary and, in fact, may be detrimental, because any mechanical intervention by operation or drain insertion may convert a sterile fluid collection to an infected one.

PANCREATIC PSEUDOCYST: a) non-infected and b) infected (suppurative).

Pseudocysts on contrast enhanced computed tomography become defined usually > 3–4 weeks after onset of pancreatitis as a well circumscribed, usually round or oval, homogeneous peripancreatic fluid collection surrounded by a well-defined wall with little to no associated tissue necrosis within the fluid collection; an MRI or ultrasonography may be required to confirm the absence of necrosis within the fluid collection. Pseudocysts develop from an APFC that persists for > 4 weeks after onset of pancreatitis. Prior to 4 weeks, a definite wall may not have formed, and these collections are categorized as an APFC. On rare occasions, an APFC may develop a clearly evident wall (capsule) prior to 4 weeks after onset of acute pancreatitis and be better termed a pseudocyst. Analysis of the pseudocyst fluid usually shows increased amylase and lipase levels indicative of an ongoing communication with the pancreatic ductal system; however, the ductal disruption that led to extravasation of amylase/lipase-rich fluid and pseudocyst formation may eventually seal off spontaneously, explaining the well-known phenomenon of spontaneous regression of pancreatic pseudocysts. The absence or presence of a recognizable ductal communication or a dilated main pancreatic duct at the time of diagnosis may be important clinically, because these findings may dictate different management algorithms; however, the presence or absence of ductal communication cannot be determined reliably by contrast enhanced computed tomography, and it is not necessary to identify the presence or absence of a communication by ERCP for classification in this new, imaging-based classification. MRI or EUS may allow this communication to be determined; the presence or absence of ductal communication may be important in determining therapy, but is not required in this classification.

Determination of presence or absence of infection in a pancreatic pseudocyst is also potentially important. An infected pancreatic pseudocyst contains purulent liquid without an associated solid component (necrosis). This definition differentiates pseudocyst from infected APNC and infected walled-off necrosis (see below). As with all peripancreatic collections, image-guided FNA with Gram stain and culture for bacteria and fungal organisms or the presence of extraluminal gas are necessary to confirm the pre-interventional diagnosis of infection. A diagnosis of infection may change the management, but a FNA is not required for all peripancreatic fluid collections.

ACUTE POST-NECROTIC COLLECTION (APNC):

Site: a) pancreatic parenchyma and peripancreatic tissues; b) pancreatic parenchyma alone; c) peripancreatic tissues alone.

Infection status: a) sterile; b) infected.

Persistent collections in patients with acute necrotizing pancreatitis are termed APNCs to distinguish them from APFCs and pseudocysts. An APNC contains both fluid and necrotic contents to varying degrees as well as areas of loculations; therefore,

this classification has avoided use of the term "fluid collections" and has used the term "collections" to emphasize this concept and to differentiate these collections from an APFC and a pseudocyst. In APNCs, a continuum exists from the initial solid necrosis to liquefaction necrosis depending on duration since onset of the disease. It should be understood that not all pancreatic and peripancreatic collections can be categorized readily into an APFC or an APNC, especially within the first week after onset of acute pancreatitis. Both APFCs and APNCs may appear as homogeneous, non-enhancing areas of low density; however; after the first week or two, APNCs should become evident on contrast enhanced computed tomography, MRI, transabdominal ultrasonography, or EUS.

As pancreatic parenchymal or peripancreatic necrosis matures, liquefaction develops as the necrotic tissue breaks down, usually beginning 2–6 weeks after the onset of pancreatitis. This entity of an APNC has imaging-based morphologic features on contrast enhanced computed tomography (or MRI, EUS, or transabdominal ultrasonography) of both necrosis and fluid within the same circumscribed area. An APNC is not a pancreatic pseudocyst, because it arises from the necrosis of necrotizing pancreatitis and contains necrotic tissue. It is often, but not invariably, associated with disruption of the main pancreatic ductal segment within the zone of parenchymal necrosis. Thus, an APNC may or may not have a connection with the pancreatic ductal system.

WALLED-OFF NECROSIS:

Site: a) pancreatic parenchyma and peripancreatic tissue; b) pancreatic parenchyma alone; c) peripancreatic tissues alone.

Infection status: a) sterile; b) infected.

As the APNC matures, the interface between the necrosis and the adjacent viable tissue becomes established by a thickened wall without an epithelial lining; this process is similar in principle to the development of a pseudocyst. This entity, termed walled-off necrosis (WON), referred to previously in the literature often as organized pancreatic necrosis, necroma, or pancreatic sequestration, represents the late stage of an APNC. WON occurs at the end stages of the necrosis continuum and represents a distinct entity both clinically and therapeutically; this entity was not recognized as such in the Atlanta Conference. A WON may be infected or sterile. The diagnosis of infected APNC can be suspected on contrast enhanced computed tomography by the presence of extraluminal gas, but definitive preoperative diagnosis of infection requires image-guided FNA with Gram stain and culture for bacteria and fungal organisms. Patients with sterile WON may remain ill despite the absence of infection (the so-called "persistently unwell patient"). Rarely, a WON may be mistaken for a pseudocyst on contrast enhanced computed tomography; therefore, MRI, transabdominal ultrasonography, or EUS may be a valuable complimentary test to document the presence of solid debris within the collection. This differentiation is important, because management, especially via a minimally invasive route, is different for WON versus an APFC and a pancreatic pseudocyst and requires removal of the solid debris.

Just as with APNCs, WON more commonly involves the pancreatic parenchyma with areas of peripancreatic necrosis as well, on rare occasion the pancreatic parenchyma alone, or the peripancreatic tissues alone and not containing any pancreatic parenchymal necrosis.

Determination of the presence of a ductal communication is of potential importance, because it may affect management; however, the presence or absence of a ductal communication will likely not be evident on contrast enhanced computed tomography, and it is not necessary to identify the presence or absence of pancreatic ductal communication in this new imaging-based classification. Therefore, ERCP is not necessary or necessarily indicated in the treatment of APNC. MRI or EUS may allow the presence of ductal communication to be established, but neither test is always warranted.

CLINICAL CLASSIFICATION (1ST WEEK):

1. Definition of Acute Pancreatitis. The clinical definition of acute pancreatitis, whether in the presence or absence of underlying chronic pancreatitis, requires two of the following three features: 1) abdominal pain suggestive strongly of acute pancreatitis (acute onset of a persistent, constant epigastric pain often radiating to the back), 2) serum amylase and/or lipase activity at least 3 times greater than the upper limit of normal (although the amylase or lipase activities are usually much greater than three times increased), and 3) characteristic findings of acute pancreatitis on transabdominal ultrasonography or on contrast enhanced computed tomography . Contrast enhanced computed tomography is considered currently to be the best, most universally available imaging modality. Characteristic findings on magnetic resonance imaging (MRI) can replace contrast enhanced computed tomography in centers that have expertise and experience with MRI. If abdominal pain is suggestive strongly of acute pancreatitis, but the serum amylase and/or lipase activity is less than 3 times the upper limit of normal, characteristic findings of acute pancreatitis on contrast enhanced computed tomography, ultrasonography, or MRI are required to confirm the diagnosis of acute pancreatitis. If a diagnosis of acute pancreatitis is established based on abdominal pain and an increased serum amylase or lipase activity, but there are no systemic signs of severe disease (see below), a contrast enhanced computed tomography may not be necessary for patient care.

2. Definition of Onset of Acute Pancreatitis. The onset of acute pancreatitis is defined as the time of onset of abdominal pain (not the time of admission to the hospital). The interval between onset of abdominal pain and admission to the hospital should be noted precisely. This interval refers specifically to admission to the first hospital (not the time that the patient is transferred from the first hospital to a tertiary care hospital).

3. Definition of Severity of Acute Pancreatitis. The definition of severe acute pancreatitis is also based on the two phases of the disease – **the early phase and the late phase.** This classification recognizes two degrees of severity: non-severe acute pancreatitis and severe acute pancreatitis.

THE EARLY PHASE. The definition of the severity of acute pancreatitis during the first 1–2 weeks is based on clinical rather than morphologic parameters. Initially at presentation and during the first 1–2 weeks, patients should be classified temporarily as having severe acute pancreatitis based on persistence of a systemic inflammatory response syndrome (SIRS) for more than 48 hours or based on development of organ failure. SIRS can be defined by presence of 2 or more of the following criteria: pulse > 90 beats/min, rectal temperature < 36° C or >38° C, white blood count < 4000 or > 12 000 per mm^3, and respirations > 20/min or PCO_2 < 32 mmHg. Ultimately, the

definition of severe acute pancreatitis in the first phase of the disease is persistent organ failure for more than 48 hours and/or death. In this early phase of acute pancreatitis, non-severe acute pancreatitis is defined as either the absence of organ failure or the presence of organ failure that does not exceed 48 hours in duration.

DEFINITION OF ORGAN FAILURE. Three organ systems should be assessed to define organ failure: respiratory, cardiovascular, and renal. Organ failure is best and most easily and universally defined in accordance with the Marshall scoring system (Table 3.2) as a score >2 for at least one of these three organ systems: respiratory (pO_2/FIO_2); renal (serum creatinine in μmol/l or mg/dl); and cardiovascular (systolic blood pressure in mm Hg).

Table 3.2 – Marshall scoring system

Organ system	**Score**				
	0	1	2	3	4
Respiratory (PaO_2/FIO_2)	>400	301–400	201–300	101–200	≤101
Renal* (serum creatinine, μmol/l) (serum creatinine, mg/dl)	≤134 <1.4	134–169 1.4-1.8	170–310 1.9-3.6	311–439 3.6-4.9	>439 >4.9
Cardiovascular (systolic blood pressure, mmHg)	>90	<90 Fluid responsive	<90 Not fluid responsive	<90, pH<7.3	<90, pH<7.2

*Considerations should be taken for patients with pre-existent chronic renal failure (baseline serum creatinine ≥134 μmol/l or ≥1.4 mg/dl.

The Marshall scoring system was chosen for its simplicity, universal applicability across multiple international centers, and its ability to stratify disease severity easily. Although not part of this classification, other scoring systems, such as the modified Marshall score (which includes the Glasgow coma score and platelet count) and the SOFA scoring system for patients managed in a critical care unit, which includes inotropic and respiratory support, can be determined at presentation and daily thereafter, so that a comparison can be made with the Marshall scoring system. Persistent multi-system organ failure is defined as two or more organs failing over the same 3-day period. Sequential organ failure should be noted in order to determine its overall impact on morbidity and mortality. For patients with hypotension, it is recommended that central venous pressure or pulmonary capillary wedge pressure be monitored to determine which patients are fluid-responsive. Determination of blood gases is recommended when arterial oxygen saturation is < 95% (on room air) and in selected situations when oxygen saturation is > 95% (such as persistent hypotension, persistent tachypnea with respiratory rate >16/minute, or severe peritoneal irritation as manifested by abdominal rigidity).

THE SECOND PHASE OF THE DISEASE. During the second phase of the disease beginning 1–2 weeks after onset of pancreatitis, severity is defined by persistent organ failure, by the development of complications prolonging hospitalization either requiring active intervention (operative, endoscopic, laparoscopic, or percutaneous) or other supportive measures (such as need for respiratory ventilation, renal dialysis, or nasojejunal feeding), or by death. Severity can be subclassified and stratified for the purpose of clinical studies by a) persistent organ failure, b) need for active intervention to treat pancreatic and peripancreatic collections, c) need for supportive measures, and d) death.

3.5. DIAGNOSIS

1. Complains:

- Abdominal pain: sudden, intense, and continuous mid epigastric or in the left upper quadrant.
- May radiate to back.
- Abdominal pain may be lessened in fetal or orthopneic positions.
- Weight loss, nausea and vomiting.
- Aundice.

2. Objective sings of disease:

Common clinical signs depend on the period of the disease in which the patient is examined by the doctor.

The early phase:

- Tachycardia, decreased blood pressure.
- May be shock.
- Palpable abdominal tenderness.
- Decreased bowel sounds due to paralytic ileus.
- Ascites.
- May be able to palpate pancreas.
- Decreased bowel sounds due to paralytic ileus.
- Left pleural effusion, atelectasis, pneumonia.

The second phase:

With aseptic course:

- Normothermia;
- The fluid isn't infected.

With infected course:

- Severe intoxication;
- Purulent resorptive fever;
- Infected fluid.

3. Pathognomonic signs:

- Gray-Turner's sign: discoloration of the skin due to dissection of peripancreatic hemorrhage may be visible in the flanks (Fig. 3.13, A).

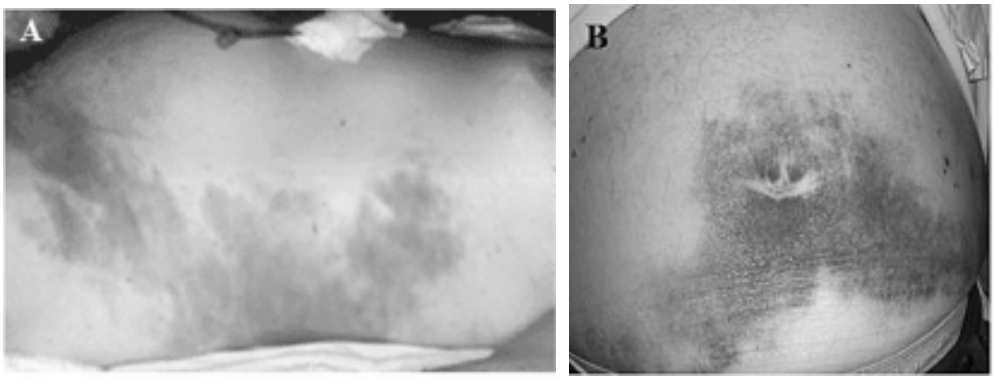

Figure 3.13. *Gray-Turner's sign (A) and Cullen's sign (B)*

- Cullen's sign: bluish discolouration around the umbilicus(Fig. 3.13, B).
- **Mondor's sign:** violet sports on the body and face.
- **Holsted's sign:** cyanosis of skin of abdominal wall.
- **Grunvald's sign:** petechial skin rash in the navel region.
- **Korte's sign:** regional tension of anterior abdominal wall in epigastria region, along the projection of pancreas.
- **Mayo-Robson's sign:** palpation pain in the left costal-vertebral angle.
- **Gobye's sign:** abdominal distension in upper region.
- **Voskresensky's sign:** absence of pulsation of abdominal aorta in epigastria region (sign of parapancreatical infiltration).

The main tasks of special investigations are:

- Differential diagnosis with other abdominal and extraabdominal diseases.
- Detection of the form (Interstitial pancreatitis or pancreonecrosis).
- Detection of the previous system disorders for immediate correction.

3.6. LABORATORY STUDIES

- Amylase and lipase. Serum amylase and lipase levels are typically elevated in persons with acute pancreatitis. Amylase or lipase levels at least 3 times above the reference range are generally considered diagnostic of acute pancreatitis.

The level of serum amylase or lipase does not indicate whether the disease is mild, moderate, or severe, and monitoring levels serially during the course of hospitalization does not offer insight into prognosis.

- Liver-associated enzymes. Determine alkaline phosphatase, total bilirubin, aspartate aminotransferase and alanine aminotransferase levels to search for evidence of gallstone pancreatitis.
- Calcium, cholesterol and triglycerides. Determine these levels to search for etiology of pancreatitis (hypercalcemia or hyperlipidemia) or complications of pancreatitis (hypocalcemia resulting from saponification of fats in the retroperitoneum). However, be wary of the fact that baseline serum triglyceride levels can be falsely lowered during an episode of acute pancreatitis.
- Serum electrolytes, creatinine, and glucose. Measure these to look for electrolyte imbalances, renal insufficiency, and pancreatic endocrine dysfunction.

● CBC. Haemoconcentration at admission (an admission hematocrit value greater than 47%) has been proposed as a sensitive measure of more severe disease. Leukocytosis may represent inflammation or infection.

● C-reactive protein. A C-reactive protein (CRP) value can be obtained 24–48 hours after presentation to provide some indication of prognosis. Higher levels have been shown to correlate with a propensity toward organ failure. A CRP value in double figures (i.e., >10 mg/dL) strongly indicates severe pancreatitis. CRP is an acute-phase reactant that is not specific for pancreatitis.

● Arterial blood gases. Measure ABGs if a patient is dyspneic. Whether tachypnea is due to acute respiratory distress syndrome or diaphragmatic irritation must be determined.

● Trypsin and its precursor trypsinogen-2 in both the urine and the peritoneal fluid have been evaluated as possible markers for acute pancreatitis but are not widely used.

3.7. RADIOGRAPHIC EVALUATION

Abdomen X-ray. X-ray examinations of the chest and abdomen may be useful in establishing the diagnosis of pancreatitis. The identification of a single dilated atonic loop of small bowel ("sentinel loop") or Gobie's symptom (gas in atonic transversum colon) provides contributory evidence for the diagnosis (Fig. 3.14).

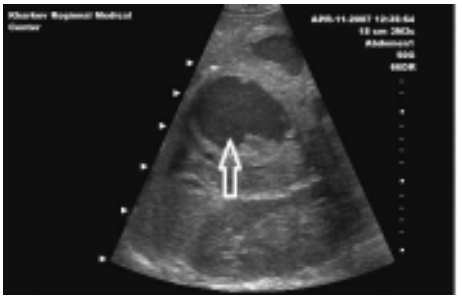

Figure 3.14. *Abdomen X-ray Gobie's sign*

Ultrasound (US) may show enlarged pancreas with stranding, abscess, fluid collections, necrosis or pseudocyst (Fig. 3.15). US may also demonstrate the presence of gallbladder pathology, such as cholecystitis, cholelithiasis, or a dilated common bile duct. US has a major limitation in that it cannot be performed when excessive bowel gas is present as occurs with an ileus.

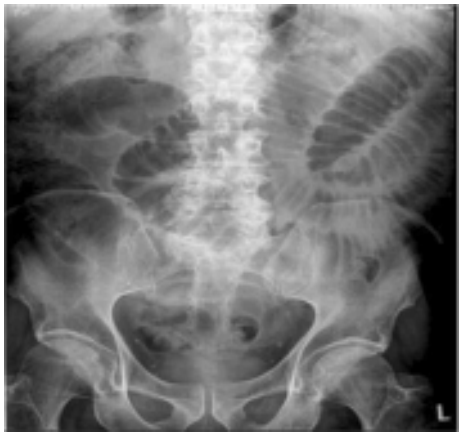

Figure 3.15.
Abdomen X-ray Gobie's sign

Endoscopic Retrograde Cholangiopancreatography (Fig. 3.16) **and Angiography.**

In addition to the diagnosis of IEP versus all three forms of acute necrotizing pancreatitis, the radiologist should address the morphologic findings of: a) Absence or presence of pancreatic parenchymal necrosis (perfusion defects) and, if present, the site(s) and extent (<30%, 30–50%, and >50%); b) characteristics of pancreatic and peripancreatic collections: location-either intrapancreatic or extrapancreatic, homogeneity and density of the collection (i.e. presence of a solid component), presence/absence of a well-demarcated wall, and presence of extraluminal gas, such as bubbles or air-fluid levels; c) other related extrapancreatic findings such as cholecystolithiasis, choledocholithiasis, gallstones,

dilation of the biliary tree, venous thrombosis/obstruction of the portal, splenic, and/or mesenteric vein(s) (+/- perisplenic, perigastric varices), arterial (pseudo)aneurysm, pleural effusion(s), ascites, and inflammatory-like involvement of peripancreatic organs-stomach, duodenum, small bowel, colon, spleen, and kidney, and liver; d) other unrelated intraperitoneal or intrathoracic abnormalities.

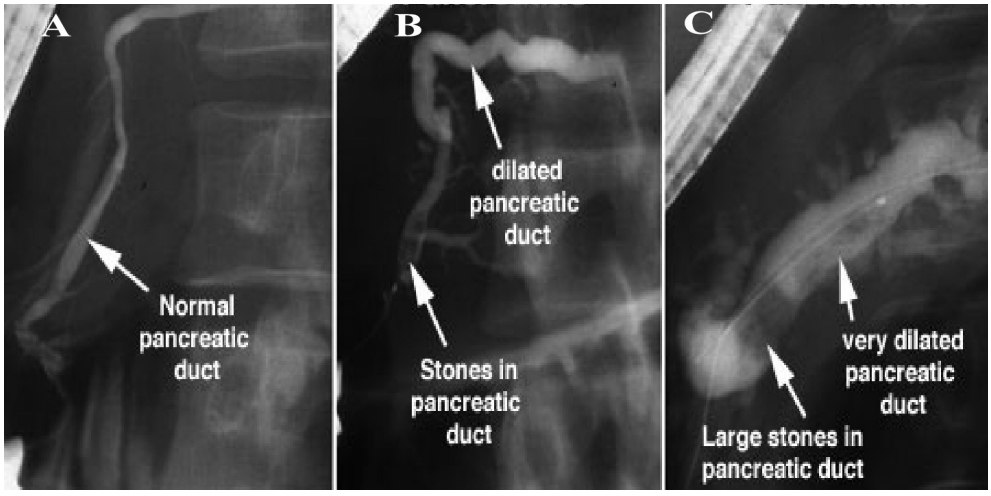

Figure 3.16. *Gall stone pancreatitis by ERCP:*
A - normal pancreas; B - moderate pancreatitis; C - severe pancreatitis

Together, the radiologist and clinician can thus classify the type of pancreatitis and its complications in the patient and plan appropriate management. A multidisciplinary approach in the care of these patients should lead to better overall outcomes.

Computed Tomography (CT) scan can be very useful in predicting severity of disease. The contrast enhanced CT, imaging-based morphologic classification is a clinical tool and as such requires close collaboration between radiologist and clinician. The radiologist describes the morphology and the clinician incorporates the radiologic findings into the clinical setting-severity of patient illness, timing since onset of disease, and associated co morbidities. Dynamic computed tomographic and magnetic resonance imaging have also been used to "stage" pancreatic inflammatory changes for prognosis. Initial studies focused on pancreatic swelling and extra-pancreatic inflammatory findings. The first CT grading system from New York applied five grades: A, normal; B, pancreatic swelling; C, peripancreatic fat abnormalities; D, single fluid collection; and E, multiple collections or gas. The relationship to general morbidity was poor, even though CT grades correlated well with Ranson scores. Non-ionic contrast agents and more rapid dynamic scanning in the late 1980s led to acceptance of CT for the diagnosis of pancreatic necrosis, but few studies have investigated its prognostic role. The importance of pancreatic necrosis in prognosis was acknowledged by its incorporation within the revised New York grading system. Applied in 88 attacks (25% had necrosis), a high CT severity index was associated with 92% morbidity and 17% mortality, and a low index with 2% morbidity and no mortality. Although both CT and magnetic resonance imaging provide morphological assessments of severity and prognosis, they represent an expensive, and not necessarily more accurate, alternative to existing prognosticators. Significant necrosis may occur without organ-system failure, leading to the aphorism "treat the man, not the scan", and the converse

is also true. A preoccupation with prognosis may detract from the most important function of imaging techniques, which remains primarily diagnostic, and to provide precise localisation, differentiation and management of pancreatic collections.

Magnetic Resonance Cholangiopancreatography (MRCP) newest "fad": decreased nephrotoxicity from gadolinium; better visualization of fluid collections: MRCP allows visualization of bile ducts for stones.

Endoscopic US (even newer but used less): does not allow stone extraction or stent insertion.

Fine Needle Aspiration. Fine needle aspiration (FNA) is useful in the early detection of infected pancreatic necrosis. It is the early detection of this condition that has a major impact on the further management and outcome in acute pancreatitis. Since clinical and laboratory findings can be often similar in patients with either sterile or infected necrosis), this important differentiation is best made by a fine needle aspiration. FNA can be performed either under CT or under ultrasound guidance. Ultrasonographically guided FNA is a fast and reliable technique for the diagnosis of infected pancreatic necrosis. Since complication rates are very low, the procedure can be repeated at short intervals to improve the diagnostic accuracy. This technique has been recommended for all patients with necrotizing pancreatitis in whom a systemic inflammatory response syndrome persists beyond the first week after onset of symptoms or when there is deterioration in the clinical situation. The technique is safe and accurate and a positive result is an indication for surgical intervention without undue delay.

In contrast, it has also been reported that percutaneous aspiration and drainage can often predispose to secondary infection of an originally sterile necrosis.

3.8. COMPLICATIONS

LOCAL COMPLICATIONS OF ACUTE PANCREATITIS

< 4 weeks after onset of pancreatitis:

- Acute peripancreatic fluid collection (sterile and Infected).
- Acute post-necrotic collection (sterile and infected).
- Pancreatic necrosis with peripancreatic necrosis (sterile and infected).
- Pancreatic necrosis alone (sterile and infected).
- Peripancreatic necrosis alone (sterile and infected).
- Pancreatic ascites.

> 4 weeks after onset of pancreatitis:

- Pancreatic pseudocyst (sterile and infected).
- Walled-off necrosis.
- Pancreatic necrosis with peripancreatic necrosis (sterile and infected).
- Pancreatic necrosis alone (sterile and infected).
- Peripancreatic necrosis alone (sterile and infected).

Involvement of adjacent organs, with hemorrhage, thrombosis, bowel infarction, obstructive jaundice, fistula formation, or mechanical obstruction.

COMMON COMPLICATIONS OF ACUTE PANCREATITIS:

- Pulmonary: acute respiratory distress syndrome (ARDS); atelactasis; pleural effusions.

- Cardiovascular: cardiogenic shock.
- Neurologic: pancreatic encephalopathy.
- Metabolic: metabolic acidosis; hypocalcemia; altered glucose metabolism.
- Hematologic: disseminated intravascular coagulopathy (DIC); GI bleeding (peptic ulcer; erosive gastritis; portal vein or splenic vein thrombosis with varices).
- Renal: prerenal failure.

DIFFERENTIAL DIAGNOSIS

- Acute edematous pancreatitis and acute necrotizing pancreatitis.
- Other diseases: acute appendicitis; ileus; perforated gastroduodenal ulcer; biliary disease.

3.9. TREATMENT

PRINCIPLES OF TREATMENT OF ACUTE PANCREATITIS:

1. Resuscitations intravascular therapy.
2. Analgesia.
3. Put pancreas to "rest".
4. Nothing by mouth, nasogastric tube only for ileus or vomiting.
5. Treat complications (pulmonary, shock, renal, metabolic).
6. Remove obstructing gallstone in severe gallstone pancreatitis endoscopically
7. Antibiotics for severe disease (after two weeks).
8. Percutaneous aspiration of pancreas to document infection in patient who fails to respond.

CONSERVATIVE TREATMENT OF STERILE PANCREATIC NECROSIS

Much of the initial therapy of pancreatitis is physiologically based. One of the major principles of medical treatment has been the proposition that pancreatic volume and enzyme secretion must be restricted.

The intravascular volume deficit may exceed 30% due to peripancreatic fluid sequestration and vomiting. Volume restoration must be rapid and efficient to maintain regular monitored urine output of more than 40 ml/hr.

Several approaches to the reduction of pancreatic secretion have been employed. Continuous nasogastric aspiration and H_2-blockers have routinely been used to decrease the hydrogen ion stimulation of secretin. In addition, gastric distention by swallowed air is prevented, and a gastrin-induced pancreatic enzyme response is thereby controlled. While these considerations represent sound application of physiologic principles to therapy, a recent prospective study could find no difference in the outcome of patients with moderately severe alcoholic pancreatitis whether or not nasogastric suction was used. Other methods to reduce acid secretion and other pancreatic stimulation include the use of cimetidine, anticholinergics, and glucagon.

Perhaps the most challenging aspect of managing acute pancreatitis is the regulation of intravenous fluid therapy. The amount of fluid required is dictated by the degree of severity of any particular episode. As much as one third of the circulating blood volume may be sequestered in the "third space" as a result of the pancreatic inflammatory process. Careful monitoring of fluid balance by central venous or pulmonary artery pressure along with hourly measurement of urination.

With severe pancreatitis, administration of both crystalloid and colloid solutions may be superior to the use of crystalloids alone. Every effort should be made to ensure that hypoperfusion of the pancreatic microvasculature and subsequent ischemic necrosis; do not occur on the basis of a volume deficit. With early, vigorous, and well-monitored fluid replacement, shock resulting from pancreatitis should decrease in frequency.

About one third of the fatalities due to pancreatitis are associated with respiratory insufficiency. When rapid and shallow respirations are observed, blood gas analysis is indicated, including determination of pO_2, pCO_2, and pH. The finding of abnormal gas exchange should be regarded as an ominous sign indicating the need for respiratory assistance. Ranson and his associates found that 38 per cent of a group of patients with acute pancreatitis exhibited severe arterial oxygen desaturation, as evidenced by an average PaO_2 of 66 mm. Hg. The hypoxemia was improved by the administration of albumin and diuretics. The association between severe pancreatitis and pulmonary insufficiency appears to be more than casual, although the mechanism involved is not yet known.

The severe pain of acute pancreatitis prevents the patient from resting, and results in ongoing cholinergic discharge, which stimulates gastric and pancreatic secretion. Therefore, pain management is of great importance. Administration of buprenorphine, pentazocine, procaine hydrochloride, and meperidine are all of value in controlling abdominal pain. Morphine is to be avoided, due to its potential to cause sphincter of Oddi spasm. Antibiotic therapy has not proved to be of value in the absence of signs or documented sources of infection.

Cautious resumption of oral feeding consisting of small and slowly increasing meals is permissible after the abdominal pain and tenderness have subsided, serum amylase has returned to normal, and the patient experiences hunger. This usually occurs within a week of the onset of an attack of mild acute pancreatitis. A low-fat, low-protein diet is advocated as the initial form of nutrition following an attack of acute pancreatitis.

As indicated previously, hypokalemia or hypocalcemia or both may occur and precipitate disturbances in cardiac function. Repletion with intravenous potassium salts must be accomplished by electrocardiographic monitoring; potassium salts must be administered cautiously when any degree of oliguria is present. The intravenous injection of calcium salts is reserved for the therapy of incipient tetany, as signaled by the appearance of a positive Chvostek or Trousseau sign, and for the development of previously mentioned electrocardiographic conduction changes.

Pancreatitis is also an autodigestive process, and various protease-inhibiting drugs, including aprotinin, gabexate mesylate, camostate, and phospholipase A_2 inhibitors, as well as fresh frozen plasma, have been tested to prevent proteolysis, but with little success. Theoretically, inhibition of proteolytic activity might affect the course of pancreatitis. The release of trypsin from the affected gland is capable of activating pancreatic kallikrein, another proteolytic enzyme, which splits the vasoactive decapeptide kallidin from a globulin. This peptide is a potent vasodilatory and hypotensive agent. An antitryptic antikallikrein polypeptide (Trasylol) has been extracted from bovine parotid glands. This agent has been shown to inhibit trypsin, chymotrypsin, kallikrein, and plasmin. Despite potent in vitro antiproteolytic activity, in a recent large prospective double-blind study, Trasylol was not found to be any

more effective in modifying the course of moderately severe pancreatitis than a placebo, regardless of the dosage given.

Somatostatin is now applied to the treatment of acute pancreatitis, it is used to decrease pancreatic secretion; it also decreases intestinal motility and reduces endocrine/exocrine pancreatic secretion.

Between 15–20% of all acute pancreatitis patients develop severe necrotizing pancreatitis. In this subgroup, stratification according to infection status is crucial. Infection of pancreatic necrosis is currently the most important risk factor contributing to death in severe necrotizing pancreatitis and surgical necrosectomy along with debridement is the most widely accepted modality for management of infected pancreatic necrosis. In contrast, the management of sterile necrosis even when associated with organ failure, is controversial and has evolved considerably in recent years. Although necrosis confirms the presence of severe disease and the extent of the sterile necrotic tissue is proportional to the rate of organ failure, sterile necrosis is primarily managed conservatively with antibiotics, nutritional support and active intensive care measures.

Although the development of infected pancreatic necrosis confers a significant risk of death, welldesigned trialsand meta-analyses have shown no benefit of prophylactic antibiotics. Prophylaxis with antibiotic therapy is not recommended for any type of acute pancreatitis unlessinfection is suspected or has been confirmed (Fig. 3.8). Nonetheless, many patients continue to receive prophylactic antibiotics despite guidelines to the contrary. Antibiotics are usually administered to patients with severe acute pancreatitis. Antibiotics with high penetration into pancreatic tissue include the fluoroquinolones, imipenem/cilastatin, and metronidazole. The mortality rate of patients with infected pancreatic necrosis treated conservatively is 60 to 100%. The use of antibiotics in patients with necrosis without infection is debated. Overall mortality does not seem to change significantly, but there is a lengthening of time to develop infection with the use of antibiotics. Antibiotic use, however, has been suggested to increase the risk of infection with resistant organisms.

Most systemic complications occur during the first week of illness are treated by standard medical measures. Close patient monitoring is the key to their timely recognition. Circulatory shock arises by a combination of volume depletion and hyperdynamic circulatory state with decreased peripheral vascular resistance. The management includes transfer to an intensive-care unit, volume replacement, and vasopressor substances. The occurrence of shock is frequently followed by pancreatic necrosis. Acute renal failure may be caused by circulatory shock and a selective increase in renal vascular resistance. The treatment is that of acute tubular necrosis arising in any setting. The leading cause of respiratory insufficiency during acute pancreatitis is the adult respiratory distress syndrome, although respiratory depression caused by opiate medications, pleural effusions, intravascular volume overload, and shallow respirations due to abdominal "splinting" may contribute. Sepsis is most commonly caused by infection of the bile ducts, of areas of pancreatic necrosis, or of peripancreatic fluid collections.

Summary specific pharmacologic therapy of acute pancreatitis:

- Antiproteolytic agent: Aprotinin; Gabexate mesilate (FOY).
- Anti-inflammatory agent: Lexipafant.
- Inhibitor of exocrine secretion: Glucagon; Atropine; Somatostatin; Octreotide.

INDICATIONS FOR SURGERY IN STERILE PANCREATIC NECROSIS

Before considering the indications for intervention, whether endoscopic or surgical, in sterile pancreatic necrosis, it must be pointed out that the mortality rate in medically managed patients with extensive sterile pancreatic necrosis is approximately 10% and surgical intervention has not reduced this mortality rate. Bearing this in mind, the principal indication for surgery should now be infection of an originally sterile pancreatic necrosis documented by fine needle aspiration. Other indications are sterile necrosis with persistent multiple organ failure and situations where sterile necrosis involves more than 50% of the pancreas. The latter two indications however, need to be individualized to the specific clinical situation. An additional indication for surgery that deserves consideration is symptomatic (severe pain, gastric outlet obstruction etc) sterile pancreatic necrosis where the presence of infection is not the sole determinant of intervention.

Timing of surgical intervention

In the last decade, the timing of surgical intervention for all forms of necrotizing pancreatitis has changed remarkably. Currently, it is accepted that surgical intervention should be as late as possible. The primary reason for this is that demarcation between viable and non-viable pancreatic tissue is better defined and this enables a more complete surgical necrosectomy and debridement that is also easier to perform. For all practical purposes, the timing of surgical intervention is around three weeks after the initial attack of acute necrotizing pancreatitis.

Surgical Techniques

The surgical principles and techniques for sterile pancreatic necrosis are essentially the same as those for infected necrosis. The aim for surgical intervention is local and adequate removal of the necrosis. The commonly adopted techniques have been necrosectomy with closed continuous lavage, repeated necrosec-tomies with planned relaparotomies and necrosectomy with "open packing". These surgical strategies have enabled major centers to reduce mortality rates below 15%. Other options such as pancreatic resections are now considered obsolete owing to their high rate of post-operative complications and the problems associated with exocrine and endocrine deficiency. Peritoneal dialysis has also been shown to be ineffective because it has no effect on the inflammatory processes in the retroperitoneum.

Non-surgical interventions

Though interventional radiology techniques such as percutaneous placement of catheters under CT guidance have been introduced, quite often operative drainage of undrained necrosis is still necessary. The formation of external fistulae is also a problem with percutaneous methods. Endoscopic treatment for pancreatic necrosis has been attempted with some success. There is a belief that endoscopic interventions are less traumatic, reduce the incidence of external fistulae and possibly reduce hospital stay. On the other hand, there is a definite possibility of introducing infection and quite often complete and adequate drainage of necrosis, especially peripheral necrosis, is difficult to achieve. Thus sufficient data in favour of these treatment options is still lacking and surgery appears to be the current gold standard whenever intervention is contemplated in sterile pancreatic necrosis.

Conclusions. The outcome of acute necrotizing pancreatitis depends on the extent of necrosis, organ failure, and the development of infection in a previously

sterile necrosis. While documented infection of pancreatic necrosis is a definite indication for surgery, current opinion supports conservative management of sterile necrosis. However, large, controlled, randomized trials are necessary to assess the role of different treatment approaches in the management of sterile pancreatic necrosis. As things stand today, conservative management of sterile necrosis revolves around judicious use of intravenous antibiotics and nutritional support (enteral or parenteral) along with quality intensive care treatment. Surgical interventions in sterile necrosis, if at all considered necessary, should be carefully individualized to the specific situation.

MANAGEMENT OF INFECTED PANCREATIC NECROSIS

Pancreatic necrosis occurs in 10–20% of patients presenting with acute pancreatitis. Attitudes to the surgical approach to this have changed greatly in the last decade, and the role of surgical drainage is gradually evolving. Of those patients that die following an attack of severe acute pancreatitis, over half will succumb to overwhelming early organ dysfunction within the first week. It is generally accepted that attempted pancreatic resection has little role in these patients, and the only randomised trial was discontinued due to unacceptable mortality in the operative group. Rarely, early surgical exploration may be required where the diagnosis is in doubt, or where bowel ischaemia or haemorrhage is suspected. For the majority however, despite the potential for continued deterioration and even death, the only appropriate treatment in the first week is maximal supportive care within an intensive care environment.

The role of surgical intervention in acute pancreatitis therefore lies in the management of local complications that develop during the evolution of the illness. Significant necrosis of either the pancreatic tissue or the surrounding adipose tissue does not in itself require treatment, as occasionally extensive necrosis may resolve without intervention, although the extent of necrosis is related to the risk of developing complications. The most frequent cause of death in patients with acute pancreatitis is multiple organ failure, and recognition of the link between organ dysfunction and the outcome from surgical intervention has revolutionised the approach to the management of these patients in the last few years.

Previously held dogma that development of infection demands urgent radical intervention9 has been questioned. The overall mortality for radical debridement is between 20 and 30%, however various factors influence this, and mortality rates vary from less than 10% to over 60% in selected series from renowned units. Undoubtedly the results are better following intervention for sterile rather than infected necrosis, minimal rather than extensive necrosis, and drainage/ debridement of a late pancreatic abscess containing necrosis rather than a true infected necrosis requiring debridement within the first 3–4 weeks of the illness. The effect of differing referral patterns and patient co-morbidity must also be taken into account. Series have tended to report results of a single surgical approach applied to a moderately small and diverse cohort of acute pancreatitic patients. The results of these series have therefore often been more influenced by the selection criteria and clinical characteristics of the patients rather than the surgical technique employed. Surgeon preference has often been determined by either good, or bad, past experience, and no consensus has been achieved regarding a curren "gold standard".

Indications for intervention

The widespread use of contrast enhanced computed tomography (CT) has allowed the early identification of necrosis, which develops within the first 3–4 days of the illness. Retro-peritoneal necrosis secondary to acute pancreatitis is not in itself and indication for surgery, as Bradley and his colleagues have shown that even extensive necrosis can be adequately treated conservatively, at least initially. Secondary infection of pancreatic necrosis is most common in the second and third week following disease onset, however broad-spectrum prophylactic antibiotics may delay this further. There is undoubtedly a relationship between the extent of necrosis and the subsequent development of infection. Once infection has occurred conservative management usually leads to escalating sepsis and death. Bacterial contamination of the necrotic material therefore traditionally mandated urgent and radical debridement for control of sepsis.

Infection is usually heralded by an increase in the SIRS response or a secondary deterioration in organ failure scores. Bacteriological confirmation may be obtained by CT or ultrasound-guided FNA of the pancreatic or peri-pancreatic area. Aspiration and culture of ascitic fluid leads to false negative aspirates and should be discouraged. Identification of gas within the retroperitoneum (Fig. 3.17) indicates infection without the need for FNA culture. The confirmation of infection within the necrotic peri-pancreatic tissue should usually still be considered an indication for intervention, however the method chosen will be influenced by the background condition of the patient. Aspiration may be repeated on a number of occasions should the culture be negative. Whilst occasionally sterile necrosis requires intervention, the lower mortality of late surgical drainage suggests that intervention in sterile necrosis should be delayed as long as possible.

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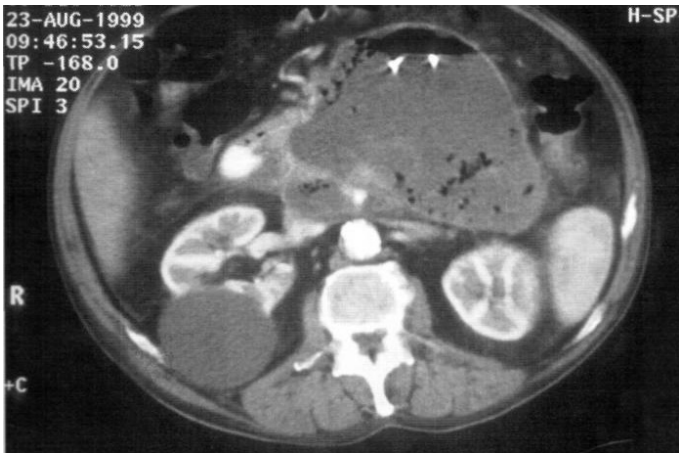

Figure 3.17. *CT confirming extensive necrosis with retroperitoneal gas*

OPERATIVE TECHNIQUES

Laparotomy, Retroperitoneal Exploration and Debridement. This is the most widely used approach to the management of infected necrosis. The process of blunt finger debridement of devitalized tissue evolved following recognition that attempts at formal pancreatic resection were associated with unacceptable mortality. The technique has been further developed with particular reference to the management of the postoperative retroperitoneal bed. Skeletization of the coeliac and mesenteric vessels may occur with extensive necrosis. The ease of debridement is dependent on the duration from onset of the acute pancreatitic episode, as the separation of necrotic and vital tissue is incomplete within the first few weeks of the illness. Debridement during this period results in increased oozing and often the need for packing for haemostasis. Some authors have argued that one advantage of antibiotic prophylaxis, is that while their use has often been shown not to influence the ultimate when exploration is less hazardous.

Despite variations regarding the approach to the postoperative management of the retroperitoneum, the initial exploration and surgical technique has changed little in the last 25 years. Most specialists employ a rooftop-subcostal incision, however midline exposure is favoured by some. Following initial laparotomy, the lesser sac is opened either through the gastrocolic omentum or by lifting the omentum from the colon. Both colonic flexures are mobilised to expose the retroperitoneum, and allow access to the paracolic gutters particularly in patients with extensive 'horseshoe' necrosis. The peritoneum at the base of the lesser sac is usually opened and the necrosis is exposed during this mobilization, but occasionally the peritoneum may remain intact and require incision to gain access to the necrosis behind.

The necrotic material, usually of a soft putty consistency is teased from the underlying viable tissue by a blunt finger dissection technique. This often leaves some adherent devitalized tissue, however overzealous clearance can lead to bleeding from the raw surface that becomes difficult to control. The procedure usually includes a cholecystectomy with an operative cholangiogram. Occasionally the vascular integrity of the colon is questionable and an extended right hemicolectomy with terminal ileostomy may be required. Many authors recommend a feeding jejunostomy although recently we have preferred naso-enteric feeding as this avoids the risks of jejunostomy complications particularly when further surgical exploration may be required. There are several approaches to the management of the residual abscess cavity, the ultimate choice being determined by a combination of personal preference, operative findings and extent of residual necrosis.

With Drainage. Simple drainage, often with multiple retroperitoneal tube drains was the original approach to the postoperative management of the debrided pancreatic and peri-pancreatic bed. Warshaw (1985) has reported respectable mortality figures using this technique, although over 40% of the patients treated had pancreatic abscess rather than true infected necrosis. Whilst mortality was less than with resective procedures, multiple second-look laparotomies were often required for residual sepsis. This led to the development of the packing or lavage techniques described below.

With Open Packing. Bradley and his colleagues (1993, 1997) have been the principal proponents of the open laparostomy technique, which was widely practiced in the 1980s as the treatment of choice. In this, at the conclusion of the debridement, the divided gastrocolic omentum is sutured to the wound edges leaving the wound

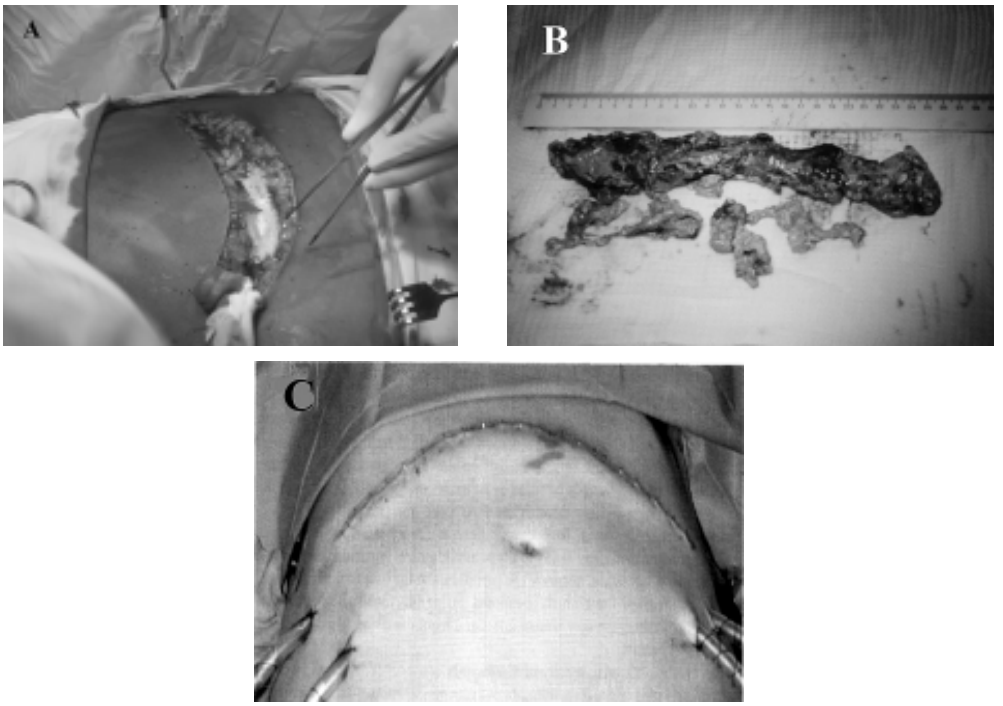

Figure 3.18. *Infected pancreatic necrosis: laparotomy (A), necrosectomy (B - pancreatic necrosis), pancreatic drainage (C)*

open. The lesser sac is packed with lubricated cotton gauze, allowing planned re-explorations/wound dressings, every few days until granulation tissue forms. This approach has the advantage that subsequent explorations/dressings can be carried out under sedation in the ITU/HDU without recourse to further anaesthetics. The technique may be modified closing part of the wound leaving the left side open to allow access for gentle manual exploration.

With Closed Lavage. Radical debridement combined with postoperative closed lavage as described by Beger et al. (1988), is now widely used for the management of infected pancreatic necrosis, the aim of the lavage being the continuous removal of devitalised necrotic material and bacteria. Having completed the debridement, the lesser sac is closed over multiple large diameter tube drains (Fig. 3.18). Continuous lavage is then commenced, our own preference being for peritoneal dialysis fluid (isoosmolar) warmed through a blood warmer and delivered at 500 ml/h. The lavage is continued, for around 3 to 4 weeks on average, until the return fluid is clear, and the patient has no residual signs of systemic sepsis. Re-exploration may still be required for residual sepsis, and for blocked or dislodged drains.

Minimally invasive approaches to infected necrosis. CT or ultrasound-guided aspiration and drainage has revolutionised the management of many surgical conditions and complications. In the presence of pancreatic necrosis, simple aspiration and percutaneous drainage alone rarely result in resolution in that it does not address the solid component within the abscess, and should therefore be discouraged as sole treatment. Percutaneous drainage may however have a role as a temporising measure in the hope of finding a "window of opportunity" in which to perform more definitive intervention. Freeny and his colleagues (1998), took this approach to its limits by

combining aggressive CT guided percutaneous drainage, tract dilatation and continuous post drainage lavage, using a median of four drains per patient. They confirmed that using this technique pancreatic sepsis may resolve, however nearly 75% of the patients will subsequently require surgical intervention for residual sepsis or necrosis. The logistic demands on the radiological department of this approach have restricted its appeal, although other groups have reported series managed by primarily conservative means.

The technique of endoscopic cystgastrostomy for pancreatic sepsis was first described by Baron et al. (1996) and developments in endoscopic ultrasound have greatly facilitated the ease and safety of performing a transmural drainage. Tract dilatation combined with naso-cyst lavage will adequately drain any fluid component however the residual necrotic material results in a significant failure rate in resolution. The same group subsequently reported that extensive necrosis was a contraindication, and highlighted that drainage must be combined with some form of surgical removal of the necrotic material.

Laparoscopy has transformed many surgical procedures and has been shown to reduce the inflammatory stimulus resulting from surgery. Some laparoscopic specialists have presented a small series of patients undergoing a laparoscopic necrosectomy, with encouraging results, however the surgical difficulty limits its universal application. In 2000, Carter et al. (2000) described the technique of percutaneous necrosectomy involving intra-operative dilatation of a percutaneous drain tract, and subsequent necrosectomy using a urological rigid rod lens system, usually from the left flank or right subcostal approach. To avoid contamination by bowel puncture, double contrast CT-guided FNA is performed. Autors preferred route is through the left fat plane between the spleen posteriorly and the colon anteriorly, although for right sided collections access can usually be obtained anterior to the duodenum, between colon and liver. The tract is first dilated using a balloon dilator, allowing insertion of a 34FG Amplatz sheath. Initial intermittent copious lavage and suction is performed until the irrigant clears sufficiently to see within the cavity. Devitalised tissue is easily identified and to can be removed by gentle traction in a piecemeal fashion (Fig. 3.19).

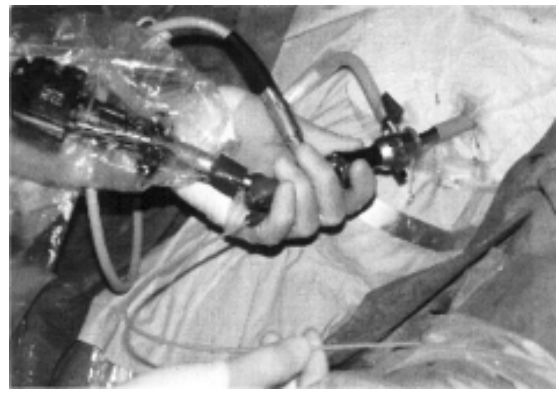

Figure 3.19. *Percutaneous necrosectomy*

Experience has suggested that overzeal-ous attempts at cavity clearance are unnecessary and can lead to bleeding. An 8FG umbilical catheter sutured to a 28FG tube drain is then passed to the far end of the cavity to allow continuous post-operative lavage (500 ml/h Dianil 7). Planned second look procedures are then performed every 7–10 days, until the cavity is clean.

Conclusion. Infected necrosis remains a major surgical challenge and the diversity of reported treatment strategies testifies to the lack of a universally agreed gold standard. Much of this controversy relates to the variability of clinical presentation, particularly of organ dysfunction, and the lack of uniformity in patient selection for procedures. Half of the patients who die as a result of severe acute pancreatitis do so

within the first few days of the illness, and may never reach a specialised unit, whilst others may not be considered fit for intervention. Background morbidity of patients within a series is difficult to determine and undoubtedly has a major influence on outcome. The potential effect of patient selection can be seen where overall mortality following surgery for infected necrosis has been reported as being less than the mortality for all-comers, with or without necrosis, recruited to trials in patients with predicted severe pancreatitis.

It is evident that no single treatment or technique is the answer. As these patients are relatively uncommon, most surgeons have adopted a favoured, but consistent, approach to infected necrosis. Within specialized units, it has become increasingly obvious that not all patients respond equally to a given surgical stimulus. As a generalisation, patients with infected necrosis but without significant organ dysfunction will do well regardless of the approach chosen, and in these an aggressive open debridement may be the most appropriate choice as it may hasten recovery.

A patient with multiple organ dysfunction and sepsis however, may be more appropriately managed by a carefully timed percutaneous drain followed by a delayed minimally invasive or even open necrosectomy.

The mortality associated with open surgical exploration in a patient with multi-organ dysfunction and sepsis is unacceptable. Undoubtedly the severity of systemic sepsis and organ dysfunction can be down-staged by percutaneous or endoscopic drainage, however the improvement tends to be temporary unless drainage is maintained together with some means of addressing the solid compo-nent of these abscesses. Tract dilatation and minimally invasive necrosectomy achieves drainage, addresses the solid component, whilst minimizing the surgical insult to the patient, and may be the optimum approach in a carefully selected high-risk group of patients. It is only through improved timing of intervention and understanding of the disease process, combining various different techniques tailored to the specific requirements of an individual patient, that the mortality in the high-risk group may fall.

3.10. GALLSTONE-INDUCED PANCREATITIS

All patients with pancreatitis should be evaluated for the presence of gallstones. Gallstones initiate the process of pancreatitis by increasing pancreatic ductal pressure and initiation of inflammatory cascade. It has been postulated that early removal of the impacted stone would attenuate the inflammatory response.

Gallstones are present in 60% of non-alcoholic patients with pancreatitis and if allowed to persist, 36–63% will develop recurrent bouts of pancreatitis. Cholecystectomy reduces this risk to 2–8%.

Timing of cholecystectomy. 75% of patients with acute abdominal pain, gallstones, and elevated amylase have no gross evidence of significant pancreatitis. Cholecystectomy is safe in this group. In patients with gross evidence of pancreatitis, 80% have mild disease and cholecystectomy is safe but does not alter the course of the pancreatitis.

In patients with severe pancreatitis there is an 82.6% morbidity and 47.8% mortality from cholecystectomy if performed within the initial 48 hours. If deferred until the signs of pancreatitis have subsided, morbidity and mortality fall to 17.8% and 11.8% respectively.

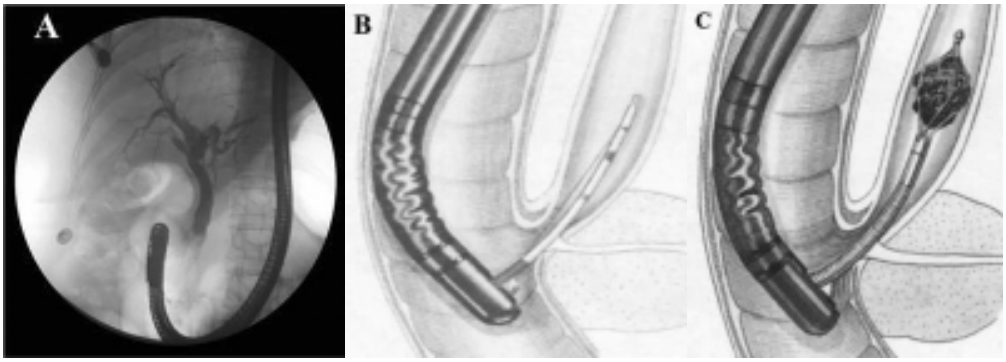

Figure 3.20. *Severe acute gallstone pancreatitis: A - endoscopic retrograde cholangiopancreatography; B, C - sphincterotomy and choledocholytotomy*

In patients with severe pancreatitis and an obstructed biliary tree secondary to choledocholithiasis, ERCP and sphincterotomy significantly reduce morbidity related to biliary complications but do not alter the course of the pancreatic inflammation (Fig. 3.20). Patients with a predicted severe attack of gallstone-associated acute pancreatitis need early endoscopic retrograde cholangiopancreatography.

Severe acute gallstone pancreatitis with obstructive jaundice should undergo urgent ERCP + endoscopic sphincterotomy (is more controvertial when obstructive jaundice is absent). A meta-analysis of four trials suggested a significant reduction in mortality and morbidity in subjects receiving early ERCP (< 24 of admission or < 72 hrs of symptom onset). For gallstone pancreatitis with obstructive jaundice, urgent ERCP should be performed within 72 hrs of onset of symptoms. If ERCP cannot be accomplished (not technically feasible or available) alternative methods of biliary drainage must be considered. When obstructive jaundice is absent, but with SAP due to suspected or confirmed gallstones, ERCP should be strongly considered within 72 hrs of onset of symptoms.

Fluid Collections. Fluid collections occur within or around the pancreas in up to 50% of patients with severe pancreatitis. The majority resolves spontaneously; collections that persist for more than 6 weeks develop a wall of granulation tissue and are then called pseudocysts. Collections that continue to expand or become infected require percutaneous drainage. Pancreatic abscesses contain liquid pus and may be considered to represent infected fluid collections.

A pancreatic abscess occurs 2 to 6 weeks after the initial attack, in contrast to infected necrosis, which occurs in the first few hours or days. The mechanism of delayed infection is not clear, but the treatment consists of external drainage, whether established by surgical methods.

Summary minimally invasive techniques:

- Laparoscopic retroperitoneal debridement.
- Percutaneous drainage.
- Endoscopic transgastric debridement and drainage.
- Some selected, relatively stable patients might be spared an operative necrosectomy.
- Offer advantages by minimizing the morbidity of laparotomy or temporizing until the retroperitoneal process has sufficiently demarcated.
- But the clinical scenario must be considered firstly.

- Recommend pancreatic debridement or drainage in patients with infected pancreatic necrosis and/or abscess confirmed by radiological evidence of gas or results of FNA.
- The gold standard for achieving this goal is open operative debridement.
- Laparoscopic and/or percutaneous interventions might be effective in selected patients.

Pancreatic Pseudocyst. Pancreatic pseudocysts (PPs) comprise more than 80% of the cystic lesions of the pancreas and cause complications in 7–25% of patients with pancreatitis or pancreatic trauma. The first step in the management of PPs is to exclude a cystic tumor. A history of pancreatitis, no solid components or mural calcification on CT scan and high amylase content at aspiration favor a diagnosis of PP. Endoscopic ultrasound (EUS)-guided FNAC is a valuable diagnostic aid. Intervention is indicated for PPs which are symptomatic, in a phase of growth, complicated (infected, hemorrhage, biliary or bowel obstruction) or in those occurring together with chronic pancreatitis and when malignancy cannot be unequivocally excluded. The current options include percutaneous catheter drainage, endoscopy and surgery. The choice depends on the mode of presentation, the cystic morphology and available technical expertise. Percutaneous catheter drainage is recommended as a temporizing measure in poor surgical candidates with immature, complicated or infected PPs. The limitations include secondary infection and pancreatic fistula in 10–20% of patients which increase complications following eventual definitive surgery. Endoscopic therapy for PPs including cystic-enteric drainage (and transpapillary drainage), is an option for PPs which bulge into the enteric lumen which have a wall thickness of less than 1cm and the absence of major vascular structures on EUS in the proposed tract or those which communicate with the pancreatic duct above a stricture. Surgical internal drainage remains the gold standard and is the procedure of choice for cysts which are symptomatic or complicated or those having a mature wall. Being more versatile, a cystojejunostomy is preferred for giant pseudocysts (>15cm) which are predominantly inframesocolic or are in an unusual location. In PPs with coexisting chronic pancreatitis and a dilated pancreatic duct, duct drainage procedures (such as longitudinal pancreaticojejunostomy) should be preferred to a cyst drainage procedure.

Percutaneous drainage:

- Continuous drainage until output < 50 ml/day + amylase activity ↓ (failure rate 16%; recurrence rates 7%).
- Complications (conversion into an infected pseudocyst (10%); catheter-site cellulitis; damage to adjacent organs; pancreatico-cutaneous fistula; GI hemorrhage).

Endoscopic drainage:

- Transenteric drainage: cystogastrostomy; cystoduodenostomy.
- Transpapillary drainage: 40–70% of pseudocysts communicate with pancreatic duct; ERCP with sphincterotomy, balloon dilatation of pancreatic duct strictures, and stent placement beyond strictures.

Surgical options:

- Excision: tail of gland & between proximal strictures – distal pancreatectomy & splenectomy; head of gland with strictures of pancreatic or bile ducts – pancreaticoduodenectomy.
- External drainage.

- Internal drainage: cystogastrostomy; cystojejunostomy (permanent resolution confirmed in 91%–97% of patients); cystoduodenostomy (can be complicated by duodenal fistula and bleeding at anastomotic site).

Laparoscopic management:

- The interface between the cyst and the enteric lumen must be ≥ 5cm for adequate drainage.
- Approaches: pancreatitis to biliary etiology → extraluminal approach with concurrent laparoscopic cholecystectomy; non-biliary origin → intraluminal (combined laparoscopic/endoscopic) approach.

Chapter 4

ACUTE INTESTINAL OBSTRUCTION

Intestinal obstruction exists when blockage prevents the normal flow of intestinal contents through the intestinal tract.

4.1. CLASSIFICATION AND DEFINITIONS

1. Types:

Mechanical obstruction:
- Obturation/obstruction;
- Strangulation ileus.
- Mixed (intussusception).

Functional obstruction:
- Dynamic ileus;
- Paralytic ileus, including ileus as a result of mesenteric thrombosis.

2. Stages of acute intestinal obstruction:

- Stage of acute disturbance of intestinal evacuation and peristalsis (12–16 hours from the beginning of the disease).
- Stage of hemodynamic disorders of the bowel wall and its mesentery (16–36 hours from the beginning of the disease).
- Stage of peritonitis (more than 36 hours from the beginning of the disease).

Mechanical obstruction. An intraluminal obstruction or a mural obstruction from pressure on the intestinal walls occurs. Examples are volvulus of the intestine, intussusception, polypoid tumors and neoplasms, stenosis, strictures, adhesions, hernias, etc (Fig. 4.1).

Functional obstruction. The intestinal musculature cannot propel the contents along the bowel. Examples are amyloidosis, muscular dystrophy, endocrine disorders such as diabetes mellitus, or neurologic disorders.

Conclusions. The obstruction can be partial or complete. Its severity depends on the region of bowel affected, the degree to which the lumen is occluded, and especially the degree to which the vascular supply to the bowel wall is disturbed.

Intestinal obstruction may be functional due to disruption of the intestinal wall or articular nerve dysfunction, or mechanical, due to a mechanical barrier. Obstruction may occur with obstruction of the small intestine (SBO) or large intestinal obstruction (LBO). Most intestinal obstructions occur in the small intestine. Adhesion is the most common cause of small intestinal obstruction accompanied by hernias and neoplasms. Other causes include intussusception, curvature (bowel twisting), and paralytic ileus. About 15% of intestinal obstructions are found in the large intestine. Most of them are in the left half of the large intestine.

Acute functional dilatation of the colon is referred to as "colonic pseudo-obstruction". Acute functional small bowel dilatation is referred to as "adynamic or

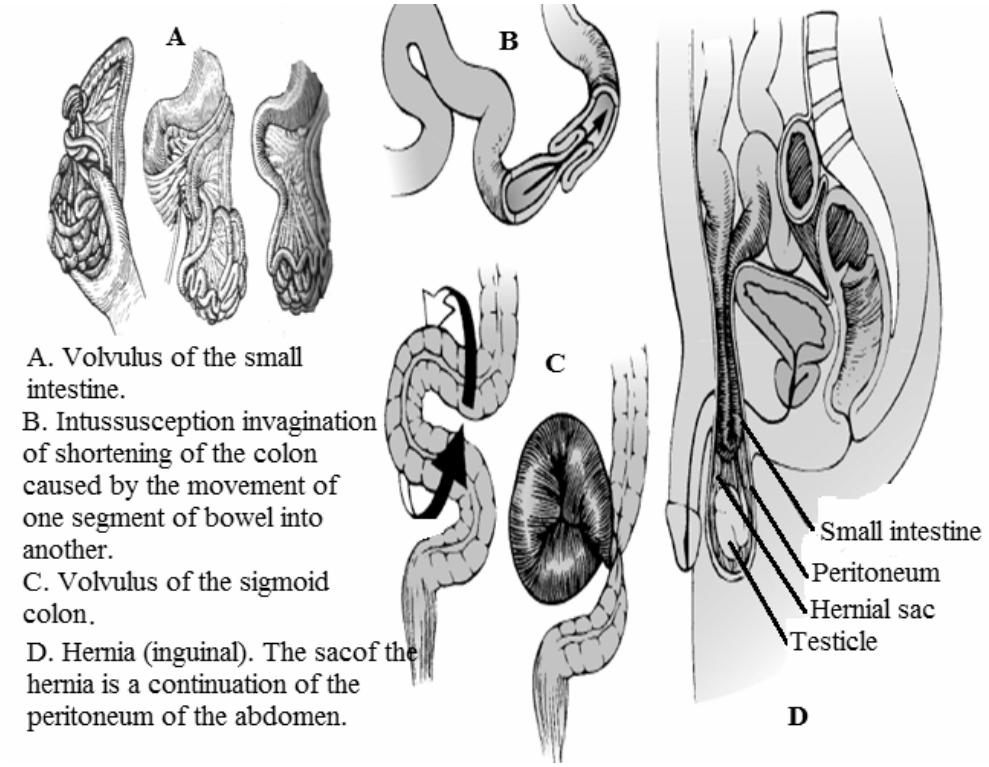

Figure 4.1. *Four causes of the intestinal obstruction.*

paralytic ileus". Small intestinal pseudo-obstruction describes a clinical syndrome characterized by manifestations of mechanical bowel obstruction in the absence of an obstructive lesion. A multitude of conditions cause functional bowel obstruction. Mechanical SBO may be due to a luminal, mural, or extra-mural mechanical barrier. Mechanical SBO may be proximal (high SBO) or distal (low SBO), closed loop or open-ended obstruction. In closed loop obstruction the lumen of the bowel is occluded at two points, thus, preventing prograde and retrograde movement of bowel contents. In open-ended obstruction a one-point obstruction interferes with the prograde propulsion of bowel contents.

Bowel obstruction may be partial or complete, simple or complicated. Partial obstruction allows some liquid contents and gas to pass through the point of obstruction, whereas complete obstruction impedes passage of all bowel contents. Unlike simple obstruction, complicated obstruction indicates compromise of the circulation to a segment of bowel with resultant ischemia, infarction, and perforation.

Intussusception is a unique type of obstruction that results from invagination of a segment of bowel into another. It may occur anywhere along the gastrointestinal tract distal to the gastric cardia. Intussusception may occur in a downward direction or may be retrograde. The exact mechanism of colic and enterocolic intussuception is not known but an organic lesion, diseased segment of bowel, or an adjacent area of normal bowel may serve as a lead point in initiating the process. Accordingly, intussusception is classified into idiopathic, postoperative, and intussusception due to an organic lesion. In adults, a neoplasm is the lead point in 80–90% of cases. A Meckel's diverticulum (MD) may invaginate into the ileum and sometimes, thence, into the colon. Volvulus is axial twist of the gastrointestinal tract around its mesentery resulting

in partial or complete luminal obstruction (closed loop) of the bowel and a variable degree of arterial or venous obstruction. Volvulus commonly occurs in the colon and may affect the stomach or SB. Volvulus occurs when the small bowel twists around a MD that is attached by a fibrous cord to the umbilicus, or when a closed loop obstruction twists along its long axis. Gallstone ileus is a mechanical bowel obstruction caused by migration of gallstones from the biliary system through a biliary-enteric fistula with impaction within lumen of the bowel. Littre's hernia is incarcerated MD in an external hernia.

4.2. PATHOPHYSIOLOGIC CHANGES

Acute small bowel obstruction (SBO) results in local as well as systemic physiologic and pathologic derangements. Significant partial or complete obstruction is associated with increased incidence of migrating clustered contractions (MCC) proximal to the site of obstruction. Such contractions are associated with abdominal cramps. With partial obstruction MCC propel intraluminal contents and allow them to pass distal to the point of obstruction. With complete unrelieved obstruction, bowel contents fail to pass distally, with resultant progressive accumulation of intraluminal fluids and distention of the proximal bowel. This eventually initiates retrograde giant contractions (RGC) in the small bowel (SB) as the first phase of vomiting. In adynamic ileus migratory motor complexes (MMC) (contractions initiated in the stomach and proximal SB occur almost simultaneously and propagate distally to clear the intestine of secretions and debris) and fed contractions (intermittent and irregular contractions that provide mixing and slow distal propulsion) are inhibited.

As intraluminal pressure in the bowel proximal to the obstruction increases, venous flow in the bowel wall and adjacent mesentery decreases, and ceases if pressure reaches systolic pressure. Blood flow to the mucosa decreases, followed by capillary rupture and hemorrhagic infiltration. A twist of the mesentery or direct pressure on the mesenteric vessels results in venous and/or arterial occlusion. Intestinal epithelium is very vulnerable to anoxia and is the first to suffer necrosis. Perforation may occur as a result of ischemic or pressure necrosis. Pressure necrosis may occur at site where a tight band adhesion passes across a loop of bowel, or where an impacted gall stone or fecoloma produces stercoral ulceration and subsequent perforation. In simple obstruction the bowel proximal to the obstruction appears heavy, edematous, and even cyanosed. In advanced cases serosal tears appear at the antimesenteric border of the bowel.

Acute SBO results in volume depletion and electrolyte disturbances. Intestinal contents are cut off from the absorptive surface of the colon. Further loss of volume occurs as bowel contents stagnate in the dilated loops of obstructed bowel, lost through vomiting, or sequestrated in the bowel wall or peritoneal cavity. Water loss is accompanied by electrolyte loss, and depending upon the level of obstruction specific electrolyte concentration changes. As intraluminal pressure increases, absorption of water and sodium decreases and luminal secretion of water, sodium, and potassium increases. In addition there is edema of the bowel wall and leakage of proteins. With strangulation, protein and electrolyte rich exudate accumulate in the peritoneal cavity, and with infarction sequestration of blood in bowel wall occurs. The peritoneal fluid exudate changes from plasma-like clear fluid, to bloody, then foul dark exudate. There is also change in the ecology of bacterial population with increase fecal type of bacterial colonies in the bowel proximal to the obstruction and altered proximal-to-distal gradient

change in bacterial flora. Bacterial breakdown of stagnant bowel contents results in formation of "feculent fluid". With strangulation physiologic changes are complicated by blood loss in the infarcted bowel, death of tissues, gut translocation of bacteria and toxins, and the final insult of perforation.

4.3. CLINICAL FEATURES

The initial symptom is usually crampy pain that is wavelike and colicky. The patient may pass blood and mucus, but no fecal matter and no flatus. Vomiting occurs. If the obstruction is complete, the peristaltic waves initially become extremely vigorous and eventually assume a reverse direction, with the intestinal contents propelled toward the mouth instead of toward the rectum. If obstruction is in the ileum, fecal vomiting takes place. The abdomen becomes distended. The lower the obstruction is in the GI tract, the more marked the abdominal distention. If the obstruction continues uncorrected, hypovolemic shock occurs from dehydration and loss of plasma volume.

1. Complains:

- Pain: suddenly arasing; paroxysmal.
- Nausea, voming.
- Abdominal distension is typical for mechanical and dynamic intestinal obstruction.
- Constipation, gas formation and absence of gas evolution.

2. Objective sings of disease:

- Inspection: hemodynamic parameters; state of nutrition, behavior, skin color, and turgor and warmth of the skin.
- Tongue is initially wet, with development of dehydration it becomes dry.
- Abdomen is asymmetric, distended.
- Palpation of the anterior abdominal wall: characteristics and localization of pain, symptoms corresponding pathognomonically to intestinal obstruction are evaluated.
- Percussion of the anterior abdominal wall: tympanitis across the whole abdominal wall.
- Auscultation: during attacks of colic, the sounds become loud, high-pitched and metallic.

3. Pathognomonic signs:

- **Wahl's sign:** asymmetry of the abdomen, high tympanic sound over the distended bowel.
- **Sklyarov's sign:** splashing sounds in the small or large intestine during balloting palpation of anterior abdominal wall.
- **Kywul's sign:** metallic sound over bloated intestinal loop during percussion of plesimer.
- **Schlange's sign:** visible peristals.

- **Spasokukotsky's sign:** during auscultation "sound of falling drop" above the stretched intestinal loops.
- **Tsege Manteuffel's sign:** when setting the siphon enema all entered the liquid (up to 500 ml) quickly leaves the intestine does not contain impurities of feces and gases). Positive at sigmoid volvulus.
- **Grekov's (Obukhov's hospital) sign:** empty and dilated anus and rectal ampoule when rectal examination of the patient.

4.4. DIAGNOSIS

RADIOLOGIC STUDIES.

X-ray. Plain radiograph of the abdomen is the most valuable initial diagnostic test in acute intestinal obstruction. This imaging method gives diagnostic information of in 50–60% of cases and provides enough information needed for clinical decision making (**Kloiber's cups**).

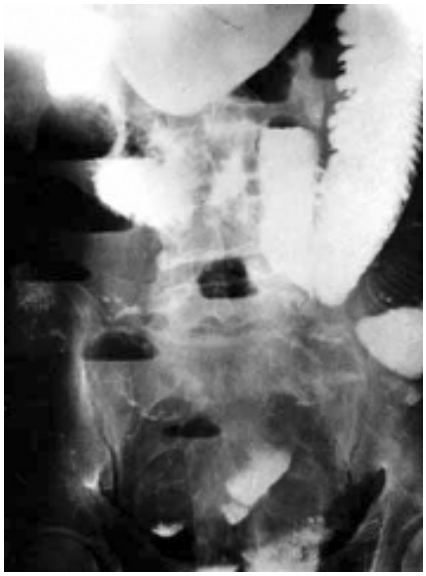

Figure 4.2. *X-ray abdominal: there are multiple intestinal air fluid level in erect film of abdomen (Kloiber's cups)*

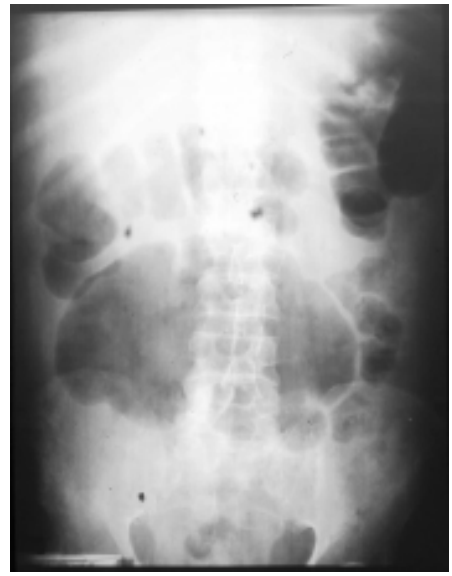

Figure 4.3. *X-ray: ileus involving small and large intestine*

The **Kloiber's cups** (air-fluid level) is the main sign of mechanical obstruction of the small intestine. In the small intestine they are low and wide, located closer to the spine. In the colon they are located in a circumferential direction of the abdominal cavity, their number is lesser, the cups are higher, but not wider, and on the gas background mucosal folds are seen in the loops of intestine (**Kerckring's folds**). In dynamic odstruction horizontal fluid levels are located in the loops of small intestine as well as large one.

In 20–30% the radiographic findings are equivocal, and in 10–20% are normal (Fig. 4.2, 4.3). The typical air fluid levels seen in the dilated bowel proximal to the obstruction may be absent in high acute intestinal obstruction, closed loop obstruction or late obstruction (Fig. 4.4). Low grade obstruction is difficult to assess with plain radiograph of the abdomen.

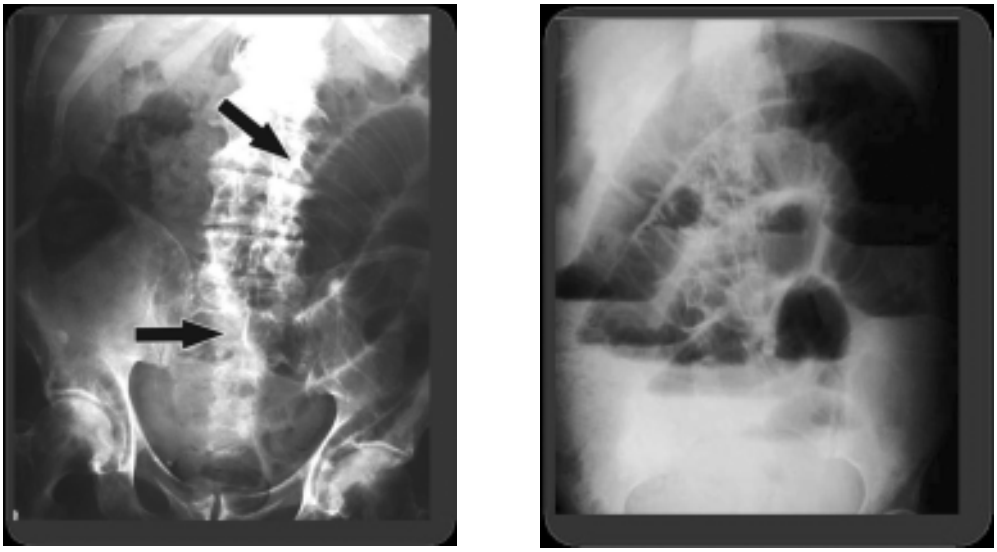

Figure 4.4. *Small bowel obstruction: multiple dilated loops of small bowel with air/fluid levels present at different heights*

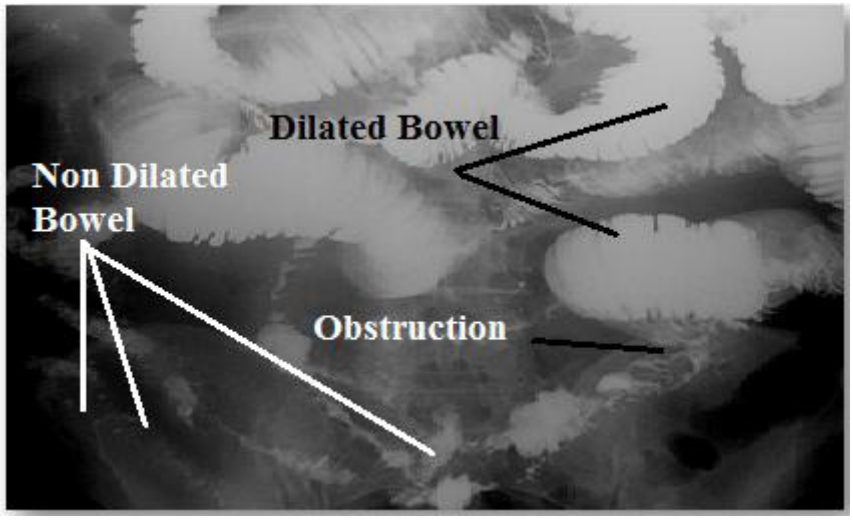

Figure 4.5. *Partial small bowel obstruction: proximal loops are dilated and distal loops are collapsed indicating an obstruction*

Intraluminal contrast studies (small bowel follow-through, enteroclysis, barium enema) are utilized in certain clinical situations (Fig. 4.5). Small bowel follow – through (SBFT) is indicated when: 1) clinical presentation of bowel obstruction is confusing; 2) plain radiograph of the abdomen is non-diagnostic, and 3) response to nonoperative management is inadequate, and more diagnostic accuracy is needed to aid in decision making i.e. to continue with nonoperative treatment or resort to surgical intervention.

The study is particularly indicated when a trial of medical treatment is warranted: postoperative or adynamic ileus, partial SBO, malignant SBO (carcinomatosis, intraabdominal recurrent or metastatic cancer), radiation enteritis, recurrent adhesive SBO, and SBO in Crohn's disease. Small bowel follow-through (SBFT) differentiates adynamic ileus from mechanical SBO. In adynamic ileus oral contrast moves to colon in 4–6 hrs.

Barium enema is not sensitive in the diagnosis of SBO except in distal SBO where LBO masquerades as SBO. Barium (or gastrografin, a water soluble hyperosmolar contrast) enema is utilized more frequently in LBO to differentiate pseudo-obstruction from mechanical obstruction, confirm the diagnosis of volvulus, and intussusception, and accurately determine site of obstruction.

Ultrasonography (US) is a valuable diagnostic tool in the evaluation of acute abdomen when used selectively. It is useful in the diagnosis of gallstone ileus, intussusception, pelvic disease, and gallbladder disease, and can aid in the exclusion of SBO. In gallstone ileus, US reveals diseased gall bladder (GB), gas in the GB or bile ducts or both, and fluid filled bowels that can be followed to the stone in the intestine. The presence of stones in the GB will modify the planned operative procedure in the treatment of gallstone ileus. In intussusception, US reveals the diagnostic "target sign", a mass with sonolucent periphery (due to edematous bowel) and a strongly hyperechoic center (from compressed center of intussusception). Paralytic ileus is differentiated from mechanical SBO by the presence of peristaltic movement that is easily observed by US. The location of obstruction is determined by analysis of dilated bowel loops in terms of location and valvulae conniventes. Adhesion is considered the cause of SBO when there is no apparent cause of obstruction.

4.5. SMALL BOWEL OBSTRUCTION (SBO)

Acute functional dilatation of the colon is referred to as "colonic pseudo-obstruction". Acute functional small bowel dilatation is referred to as "adynamic or paralytic ileus". Small intestinal pseudo-obstruction describes a clinical syndrome characterized by manifestations of mechanical bowel obstruction in the absence of an obstructive lesion. A multitude of conditions cause functional bowel obstruction. Mechanical SBO may be due to a luminal, mural, or extra-mural mechanical barrier. Mechanical SBO may be proximal (high SBO) or distal (low SBO), closed loop or open-ended obstruction. In closed loop obstruction the lumen of the bowel is occluded at two points thus preventing prograde and retrograde movement of bowel contents. In open-ended obstruction a one-point obstruction interferes with the prograde propulsion of bowel contents.

4.5.1. DIAGNOSIS

The diagnosis of majority of cases of bowel obstruction can be made based on clinical presentation and initial plain radiograph of the abdomen.

Abdominal x-ray studies show abnormal quantities of gas, fluid, or both in the bowel (Fig. 4.2, 4.3).

Laboratory studies:

- complete blood count;
- serum electrolytes and amylase determination;
- arterial blood gas analysis;
- urine test.

Clinical features:

Past surgical and medical history may shed light on etiology of SBO. In the absence of prior surgery and any apparent cause, or in presence of clinically confusing clinical picture, intussusception, MD, gallstone ileus, and neoplasms are suspects. The four cardinal symptoms of bowel obstruction are pain, vomiting, obstipation/ absolute constipation, and distention. Obstipation, change in bowel habits, complete

constipation, and abdominal distention are the predominant symptoms in LBO. Vomiting occurs late in the course of the disease. On the other hand, pain, vomiting, and distention are commonly seen in SBO. The pain is colicky in nature and becomes dull, late in the course of SBO. Vomiting is a pronounced symptom in high SBO. The vomitus is bilious or semi-indigested food in high SBO, and feculant in low SBO. Obstipation and constipation are present to a variable degree. "Tumbling SBO" describes intermittent symptoms of obstruction seen in patients with gallstone ileus. These episodes correspond to stone impaction, subsequent release, and reobstruction. Biliary symptoms are present before the onset of obstruction in 20–56% of cases. Intermittent partial bowel obstructive symptoms are also suggestive of intussusception.

The presence of strangulation/gangrene in SBO cannot always be reliably excluded or confirmed even in the hands of the most experienced clinician. Four classical findings are often used as indicators of strangulation: tachycardia, localized abdominal tenderness or pain, leuckocytosis, and fever. The absence of these four signs indicates simple obstruction, the development of any of the indicators raises the index of suspicion of strangulation, and the presence of multiple clinical parameters is correct in 70% of SBO with strangulation.

Radiologic studies:

X-ray. Abdominal ray studies show abnormal quantities of gas, fluid, or both in the bowel (Fig. 4.2, 4.3).

Ultrasonography (US) is a valuable diagnostic tool in the evaluation of acute abdomen. It is useful in the diagnosis of gallstone ileus, intussusception, pelvic disease, and gallbladder disease, and can aid in the exclusion of SBO. In gallstone ileus, US reveals diseased gallbladder (GB), gas in the GB or bile ducts or both, and fluid filled bowels that can be followed to the stone in the intestine. The presence of stones in the GB will modify the planned operative procedure in the treatment of gallstone ileus. In intussusception, US reveals the diagnostic "target sign", a mass with sonolucent periphery (due to edematous bowel) and a strongly hyperechoic center (from compressed center of intussusception). Paralytic ileus is differentiated from mechanical SBO by the presence of peristaltic movement that is easily observed by US.

With US for mechanical obstruction, dilated loops of small bowel with air/fluid levels present at different heights (Fig. 4.6).

Computed tomography (CT scan) is emerging as a valuable tool in the management of bowel obstruction (Fig. 4.7).

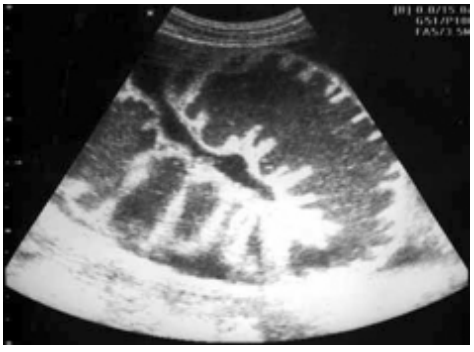

Figure 4.6. *Ultrasonography: dilated loops of small bowel with air/fluid levels present at different heights*

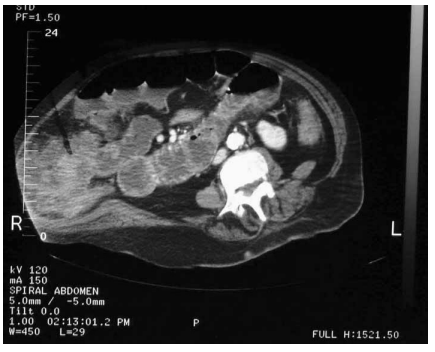

Figure 4.7. *CT scan: multiple dilated loops of small bowel with air/fluid levels present at different heights*

It confirms the diagnosis, differentiates between mechanical and functional obstruction, provides information about cause and site of obstruction, and helps differentiate between simple and complicated SBO. Furthermore, CT scan can diagnose other disease states. Hence CT scan helps in decision making for early surgical intervention, and prevents delay in treatment. CT scan may give false positive results and may be difficult to interprete when colonic abnormalities cause predominantly SB dilatation. CT scan is unable to identify location and cause of obstruction accurately in 18% of cases. Furthermore, CT scan cannot predict who will benefit from conservative treatment in cases of partial SBO. In these situations SBFT or enteroclysis are more helpful.

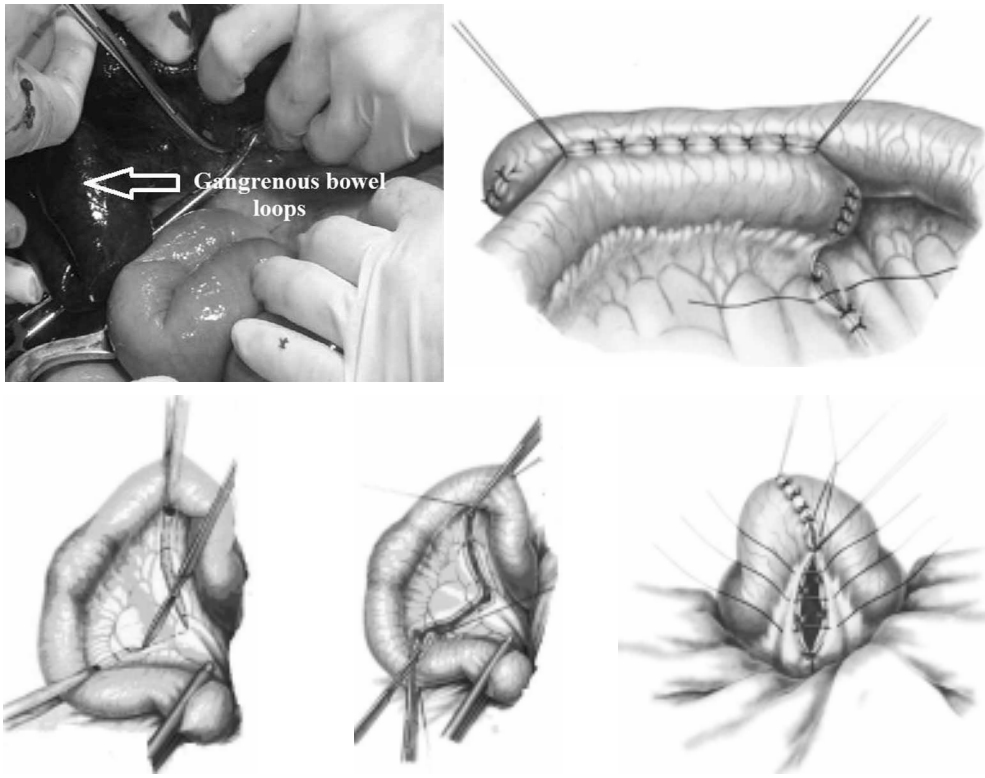

Figure 4.8. *Resection of the obstructing lesion or strangulated bowel with primary anastomosis*

Differential diagnosis with: perforated peptic ulcer; acute pancreatitis; acute appendicitis complicated by peritonitis; hepatic abscess; renal colic.

4.5.2. SURGICAL TREATMENT OF SBO

Time of operation:

- For strangulation and closed-loop obstruction the operation is required as soon as possible.
- Partial SBO: nonoperative management for 24-48 hrs; if no improvement, small bowel follow - through or CT to increase accuracy of diagnosis; laparotomy if radiologic evidence of high grade obstruction, or if clinical, laboratory, or radiologic evidence of strangulation.

The Procedure of Operation:

- Procedures not requiting opening of bowel.
- Enterotomy for removal of obturation obstruction.
- Resection of the obstructing lesion or strangulated bowel with primary anastomosis (Fig. 4.8).
- Gastrointestinal decompression.
- Bypass anastomosis around an obstruction.
- Formation of a cutaneous stoma proximal to the obstruction.

Postoperative Care: the principles are: fluid and electrolyte management, antibiotics and gastrointestinal decompression.

4.5.3. ADHESIVE SBO

With nonoperative treatment, complete SBO resolves far less frequently than partial SBO, 15–36% vs. 55–75%. Surgical intervention is indicated when strangulation is suspected to develop during nonoperative treatment, or when conservative treatment fails. The appearance of the bowel before and after release of adhesion is compared. Vascular compromise is recognized by bluish discoloration of intestinal wall, loss of arterial pulsation, subserosal and mesenteric hemorrhage, and lack of peristalsis (Fig. 4.9). If the bowel loop pinks up, resection is avoided, otherwise resection is indicated.

To prevent subsequent adhesion formation various mechanical and chemical methods have been employed. Mechanical methods include plication (small bowel and mesenteric), and stenting with long intestinal tubes. In addition to failure to prevent re-obstruction, plication is time consuming and tedious, and carries the risk of injury to the bowel or mesenteric vessels. Similarly, long intestinal tubes, in addition to difficulty in positioning distal to ligament of Treitz, are not without complications, and long term results are not adequately evaluated. Although high dose steroids with or without promethazine, antihistamines, and dextran-70 proved to reduce adhesion formation in animals, the potential for disastrous complications prevented their use in humans. A variety of other chemicals have been used to prevent adhesions with mixed results and associated significant complications. Sodium hyaluronate based bioabsorbable membrane has been shown to reduce adhesion formation in human, but its effect on intestinal obstruction is yet to be determined.

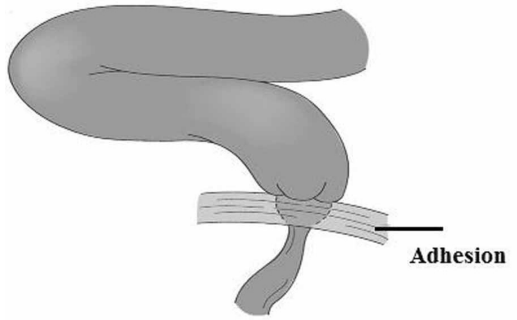

Figure 4.9. *Schematic illustration: simple obstruction is most often due to adhesion*

4.5.4. GALLSTONE ILEUS

Obstruction of the intestinal tract by gallstones, commonly known as "gallctone ileus", is a unique clinical entity, not particularly uncommon but so infrequently encountered that the average surgeon rarely observes more then a few in his professional

lifetime. Bartholin is credited with observing and describing the first case of gallstone ileus in a patient examined at autopsy in 1654. Courvoisier focused attention on the entity in 1890 by recording 131 cases in the first published paper on the subject. Gallstone Ileus is an infrequent cause of mechanical bowel obstruction. It is caused by an impaction of a gallstone in the terminal ileum by passing through a billiary-enteric fistula (often from duodenum) (fig 4.10).

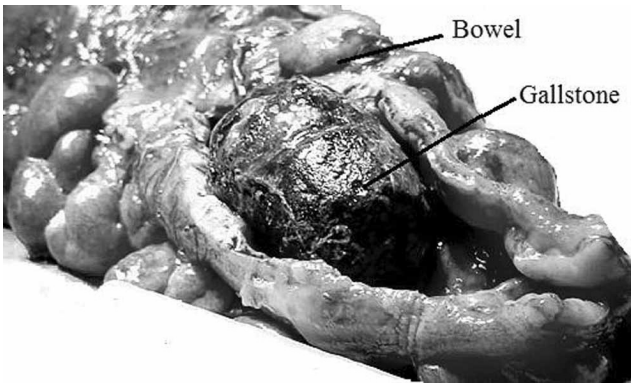

Figure 4.10. *Gallstone Ileus*

It occurs more frequently in women with average age of 70 years. The diagnosis of gallstone ileus is often difficult to make. Time from onset of symptoms to surgical intervention is often long, and correct diagnosis is made preoperative only in 13–60% of cases. In the small bowel, the site of obstruction is usually the distal ileum, and multiple stones are present in 3–15%.

The clinical symptoms of gallstone ileus are by no means diagnostic. A typical history of cholecystic disease is obtained in less than half of the patients, probably due to the difficulty in obtaining an accurate story from an elderly seriously ill patient. The gradual onset of colicky abdominal pain, distension, nausea and vomiting, and the recurrent nature of the attacks are characteristic of partial or intermittent intestinal obstruction from any cause. Symptoms lack consistency and may even simulate early cholecystitis, pancreatitis or early diverticulitis. There is usually no suggestion of an acute inflammatory episode coincident with fistula formation and passage of the stone, but this may be over shadowed by obstructive symptoms. One facet of the history if carefully evaluated may be important, namely the progressive nature of the symptoms and shifting of the primary site of pain. Bearing in mind the mechanism of propulsion of the obstructing stone, it produces what one might call a "tumbling" obstruction, the episodes of cramping pain and nausea corresponding to the halting passage of the stone along the alimentary tract.

Physical findings are inconsistent, Tenderness, distension, tympany, and hyperpeperistalsis may be present in variable degree, but no more pronounced than one would expect from any other type of obstruction or, in fact, other nonsurgical conditions. The tenderness in the gallbladder area which might be expected from the acute inflammation and edema associated with the fistula formation is usually absent or masked by abdominal distension. Dehydration is usually present, insidious in its development. Palpation of a large gallstone through the abdominal wall or on rectal examination has been reported but there is nothing to indicate the nature of the mass and to the absence of other confirmatory, findings, is of little significance.

X-ray demonstration of distended loops of bowel, an opaque shadow suggestive of gallstone, a changing obstructive level and air in the biliary tree is almost positive confirmatory evidence.

Abdominal radiography showed a hyperdense lesion with calcified margins in the right upper quadrant (Fig. 4.11, panel 1, arrow). The upper gastrointestinal series,

which was performed with the use of barium contrast material, showed a fistulous communication between the gallbladder and the duodenum, with multiple filling defects in the jejunum (Fig. 4.11, panel 2, arrows). Cholecystoduodenal fistulas can occur through erosion of gallstones (typically, more than 2.5 cm in diameter) into the intestinal lumen.

If some but not all of these are present, one is justified to entertaining a high index of suspicion and instituting therapy on a presumptive diagnosis. Since successful treatment of the disease is so dependent upon early diagnosis it is imperative that these factors be evaluated as early as possible in its course. Fortunately our concept of diagnosis has, during recent years, assumed a positive attitude in contrast to the pessimistic outlook voiced by earlier investigators.

CT of the abdomen showed a **Rigler's triad** (Fig. 4.12):

1. Pneumobilia.
2. SBO.
3. Impacted gallstone-usually in the terminal ileum at ileocecal valve.

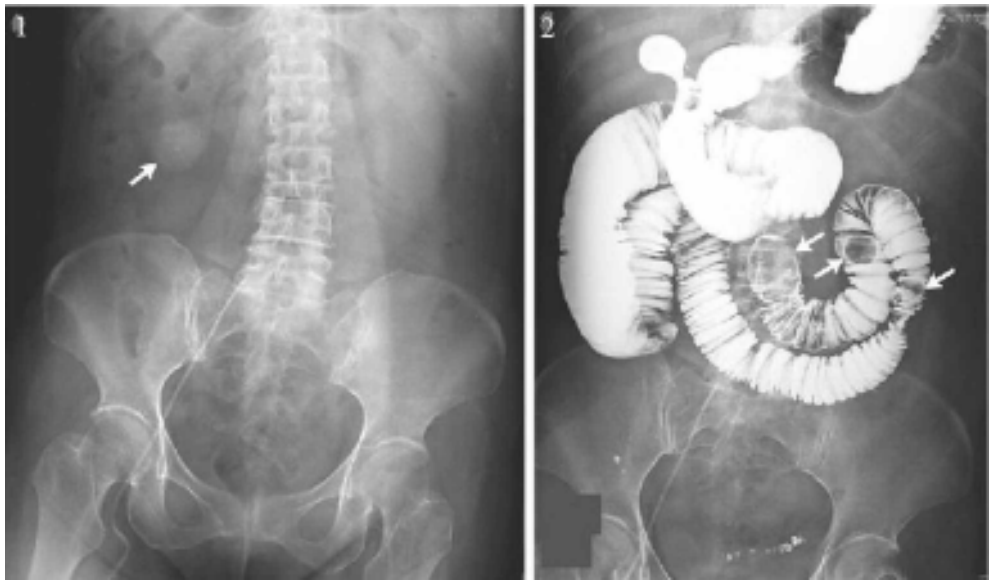

Figure 4.11. *Abdominal radiography*

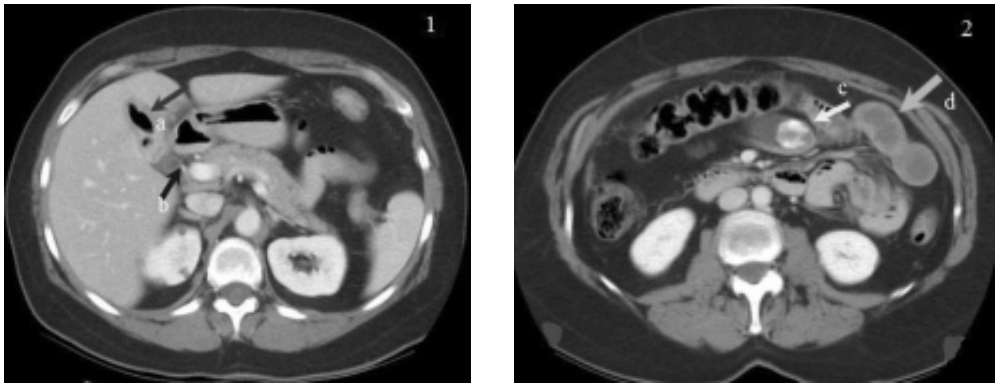

Figure 4.12. *CT scan: panal 1 CT of the abdomen show air in the gallbladder (arrow - a) and air in the common bile duct (arrow - b) representing pneumobilia; panal 2 the gallstone in the small bowel lumen (arrow - c) and dilated and fluid-filled loops of small bowel from small bowel obstruction (arrow - d)*

Treatment consists of relief of the obstruction at the earliest optimal time. Inasmuch as these patients are usually elderly, seriously ill, victims of concomitant disease such as obesity, diabetes, and myocardial disease, and are dehydrated and electrolytica depleted:, it is of paramount importance to restore the patient to optimal condition before proceeding with definitive operation. This includes restoration of electrolyte balance, hydration, and decompression with nasogastric suction so long as one does not rely too long on intubation. Exploration and removal of the abstracting lesion should be carried out as soon as the patient's condition permits. Attempts to move or crush the stone are not advisable since this entails: the risk of injury to an already devitalized bowel. A longitudinal incision with removal of the stone and transverse closure is usually sufficient. If the bowel appears injured beyond the point of spontaneous recovery, resection of the segment should be done or in extremely critical cases, exteriorization of the diseased portion. The options are enterolithotomy, cholecystectomy, and fistula division, with or without common bile duct exploration (one-stage procedure), with definitive repair performed at a second operation (two-stage procedure). The treatment of choice is the enterolithotomy wich consits in localize and extract the gallstone. Often the cholecystectomy is contraindicated by comorbidities and the general state of the patient.

4.5.5. MALIGNANT SBO

This refers to obstruction occurring after treatment of a primary malignancy. Obstruction is due to benign causes (adhesion, radiation enteritis, internal hernia) occurs in 18–38% of cases (Fig. 4.13).

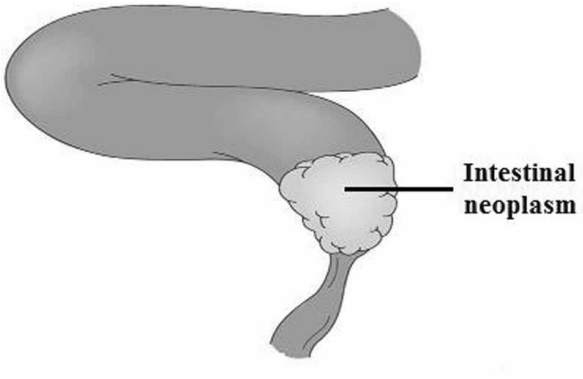

Figure 4.13. *Schematic illustration: simple obstruction is most often due to neoplasm*

Ten percent to 30% of patients will have relief of obstruction with nonoperative management alone, and about 40% will eventually require surgery. Resolution with nasograstric decompression occurs in 68% of cases and within 3 days. About 35–80% of patients will obtain relief of symptoms with surgery depending on nature of obstruction. Patients presenting in shock, with carcinomatosis, ascites, or palpable mass, have 54% to 100 % mortality. Hence, patients with known cancers should be treated as any other patient presenting with SBO, and final decision making regarding surgical intervention must be individualized. Early surgical intervention is indicated in patients with no known recurrence or long interval to the development of SBO. In patients with carcinomatosis, ascites, or palpable masses, more prolonged course of nonoperative treatment is justifiable. Surgical intervention is indicated if nasogastric decompression fails or if re-obstruction develops after removal of nasogastric tube. Selection of surgical procedure, resection, bypass, gastrostomy, or tube jejunostomy is based on extent of the disease. Used selectively, percutaneous gastrostomy can improve quality of life.

4.5.6. INTUSSUSCEPTION

In adults 85–90% of intussusceptions are associated with a discrete, pathologic process leading the intussusception, and neoplasms account for majority of cases (Fig. 4.14). Malignant lesions are being recognized with increasing frequency. A recently recognized subtype is postoperative intussusception. The point of origin of the intussusception is the small bowel and more specifically, the jejunum, particularly proximal jejunum, and dense desmoplastic inflammatory reaction within the mesentery may be the underlying mechanism precipitating the intussusception.

Treatment of intussusception in adults is surgical without attempts at hydrostatic reduction. Optimal surgical procedure depends on the anatomic location, presence of a lead point, and local factors, such as edema, inflammation, and ischemia of involved bowel. While resection is the treatment of colic and enterocolic intussusception, the choice in enteric type i.e. attempt at operative reduction vs. resection without attempt at reduction, depends on presence of underlying lesion, chances that the lesion is malignant, and viability of involved bowel.

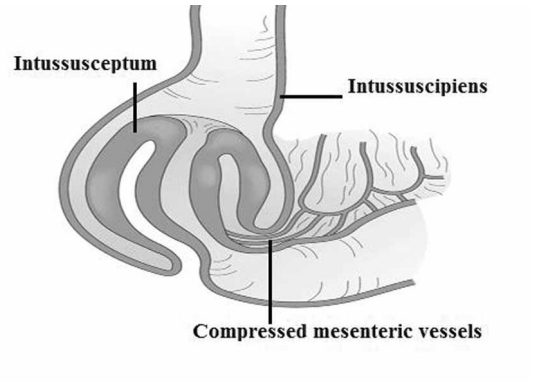

Figure 4.14. *Schematic illustration: simple obstruction is most often due to intussusception*

4.5.7. EARLY POST-OPERATIVE OBSTRUCTION

This is defined as SBO within 30 days after celiotomy. In this clinical situation bowel activity may not return (prolonged ileus) or there is initial temporary return of bowel function (Fig. 4.15).

The obstruction is due to adhesions (92%), phlegmon or abscess, intussusception (2.5–4%), or internal hernia. The treatment is conservative in the absence of bowel ischemia or mechanical obstruction. Nasogastric decompression, intravenous fluid therapy, and even parenteral nutrition for up to 10–14 days is indicated if the patient is stable and exhibiting clinical and radiologic improvement continues. After this time further improvement is unlikely and operation should be performed.

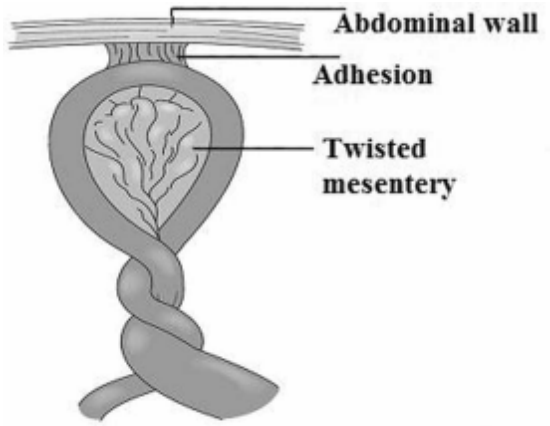

Figure 4.15. *Schematic illustration of a closed-loop obstruction. The small intestine twists around its mesentery, compromising inflow and outflow and outf low of luminal contents from the loop. Also, the vascular supply to the loop may be compromised because of the twisting of the mesentery*

4.6. LARGE BOWEL OBSTRUCTION (LBO)

Obstruction mechanical interruption of the flow of intestinal contents (adults):

1. Neoplasms (60% of cases).

Cancer of the colon and rectum is predominantly (95%) adenocarcinoma it may start as a benign polyp but may become malignant, invade and destroy normal tissues, and extend into surrounding structures (Fig. 4.16). Cancer cells may break away from the primary tumor and spread to other parts of the body (most often to the liver).

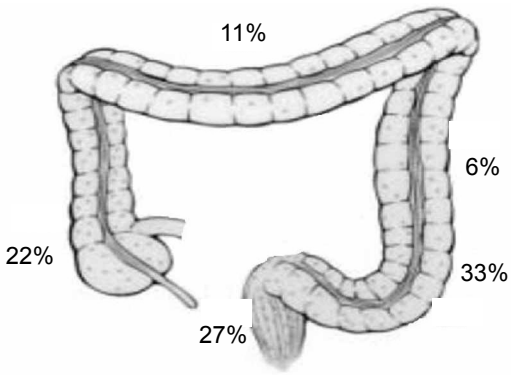

Figure 4.16. *Cases of neoplasms of the colon and rectum*

2. Volvulus: the sigmoid; the cecal ; others are rare.

3. Diverticulitis with stricture: the sigmoid; the cecal; others are rare.

4. Intussuception.

4.6.1. PATHOPHYSIOLOGY

As in small bowel obstruction, large bowel obstruction (LBO) results in an accumulation of intestinal contents, fluid, and gas proximal to the obstruction. Obstruction in the large bowel can lead to severe distention and perforation unless some gas and fluid can flow back through the ileal valve. Large bowel obstruction, even if complete, may be undramatic if the blood supply to the colon is not disturbed. If the blood supply is cut off, however, intestinal strangulation and necrosis (ie, tissue death) occur; this condition is life threatening. Dehydration occurs more slowly than in the small intestine because the colon can absorb its fluid contents and can distend to a size considerably beyond its normal full capacity.

4.6.2. CLINICAL MANIFESTATIONS

Large bowel obstruction differs clinically from small bowel obstruction in that the symptoms develop and progress relatively slowly. In patients with obstruction in the sigmoid colon or the rectum, constipation may be the only symptom for days. Loops of large bowel become visibly outlined through the abdominal wall, and the patient has crampy lower abdominal pain. Finally, fecal vomiting develops. Symptoms of shock may occur.

4.6.3. ASSESSMENT AND DIAGNOSTIC FINDINGS

Diagnosis is based on symptoms and on X-ray studies. Abdominal X-ray studies (flat and upright) show a distended colon. Barium studies are contraindicated (Fig. 4.17, 4.18).

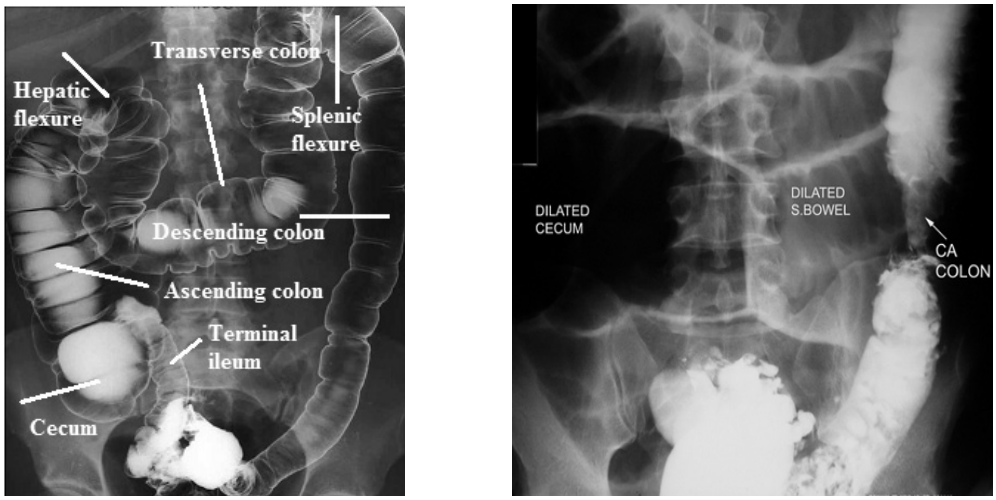

Figure 4.18. *Large bowel obstruction cancer of the descending colon (arrow)*

4.6.4. MEDICAL MANAGEMENT OF THE NEOPLASMS OF THE COLON

- A colonoscopy may be performed to untwist and decompress the bowel.
- Neoplasms of the right colon: a cecostomy: in which a surgical opening is made into the cecum, may be performed for patients who are poor surgical risks and urgently need relief from the obstruction (Fig. 5.19).

Surgical resection of the right colon right hemicolectomy (Fig. 4.20). Colic resection is easy to perform because of the mobility of the right colon.

- Neoplasms of the transverse colon: surgical resection of the colon and a cecostomy.
- Surgical resection to remove the obstructing lesion. A temporary or permanent colostomy may be necessary. An ileoanal anastomosis may be performed if it is necessary to remove the entire large colon. When gangrene is associated with peritonitis a **Hartmann's procedure** may be performed (Fig. 4.21).

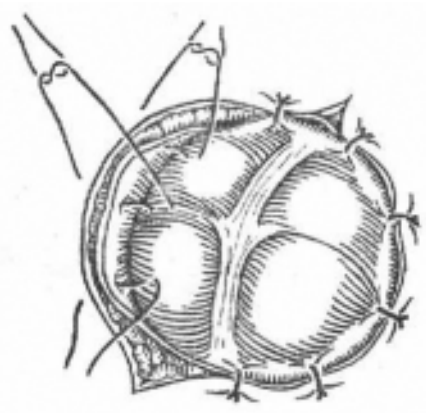

Figure 4.19. *Cecostomy*

4.6.5. VOLVULUS OF THE COLON

Obstruction caused by twisting of the intestines more than 180 degrees about the axis of the mesentery (Fig. 4.22).

Pathophysiology of obstruction:

- With mechanical obstruction, air and fluid accumulate in the bowel lumen. Results in increase of intestinal intraluminal pressure.
- This further inhibits absorption and stimulates influx of water and electrolytes into lumen.

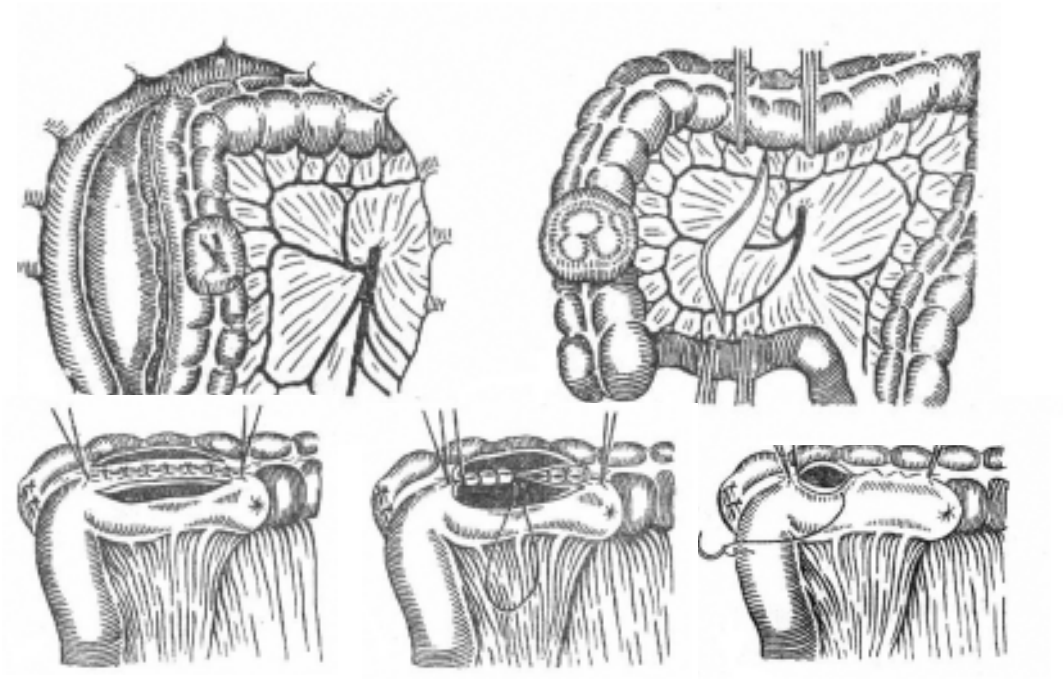

Figure 4.20. *Right*

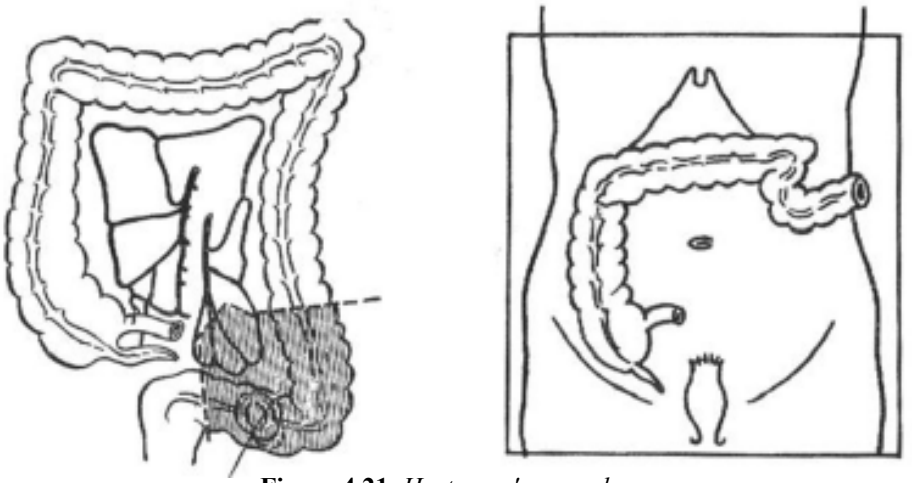

Figure 4.21. *Hartmann's procedure*

- Initially, there is increase in peristaltic activity. But as process progresses, coordinated peristaltic activity diminishes along with contractile function.
- Gives rise to dilated and atonic bowel proximal to point of obstruction.
- With progression, patient may actually appear to improve clinically with less
- Frequent and crampy pain.
- Effect of mechanical obstruction causes an initial increase in blood flow.
- With unrelieved obstruction, blood flow diminishes leading to breakdown of mucosal barriers and increased susceptibility to bacterial invasion and ischemia.

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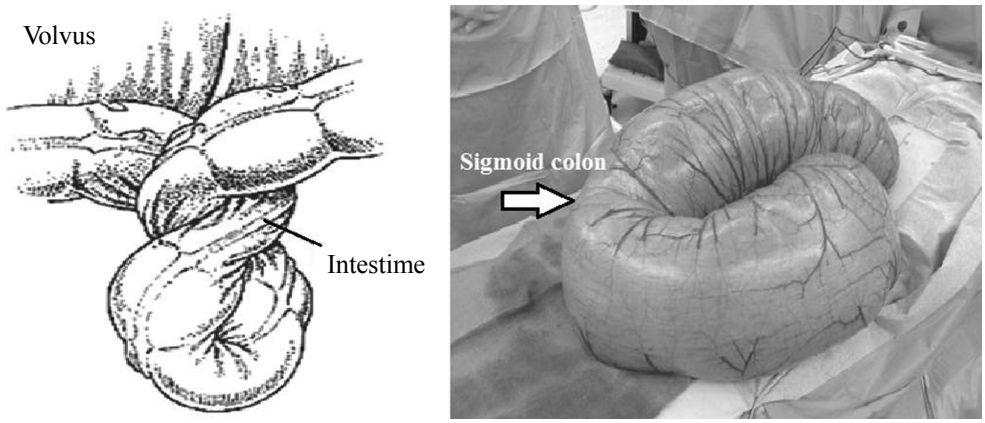


Figure 4.22. *Volvulus of the colon*

- Initially, there is increase in peristaltic activity. But as process progresses, coordinated peristaltic activity diminishes along with contractile function.
- Gives rise to dilated and atonic bowel proximal to point of obstruction.
- With progression, patient may actually appear to improve clinically with less.
- Frequent and crampy pain.
- Effect of mechanical obstruction causes an initial increase in blood flow.
- With unrelieved obstruction, blood flow diminishes leading to breakdown of mucosal barriers and increased susceptibility to bacterial invasion and ischemia.

Volvulus of the right colon. Acute volvulus of the right colon and terminal ileum (cecal volvulus) accounts for 1–10% of all intestinal obstructions and 18–44% (~25%) of all cases of colonic volvulus. Autopsies have shown that in 11–22% of the population the right colon is sufficiently mobile to allow the development of volvulus. Adhesions from previous surgery, congenital band, pregnancy, malrotation and obstructing lesions of the left colon have been discussed as triggers (Fig. 4.23).

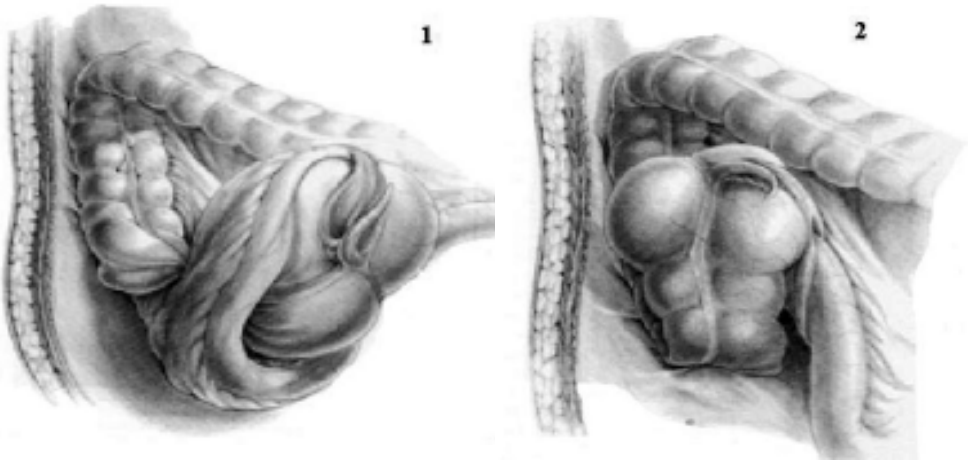


Figure 4.23. *Types of volvulus of the right colon: 1 – axial torsion type (twist 180–360 degrees on longitudinal axis of ascending colon (distal ileum and ascending colon); associated with bowel compromise, ischemia, and perforation); 2 – cecal bascule (cecum folds anteriorly on ascending colon; may result in intermittent obstructive symptoms)*

Female predominance has been frequently reported; many authors observed a significantly higher frequency in elderly women.

Clinical features. The diagnosis is not easy; the clinical picture is that of bowel obstruction with a tympanic mass extending from the right lower to the right upper quadrant. Radiological examination of the abdomen remains the key to diagnosis: typically, the caecal shadow is absent from the lower quadrant, the caecal air fluid level may be seen in the left upper quadrant mimicking the stomach shadow (Fig. 4.24).

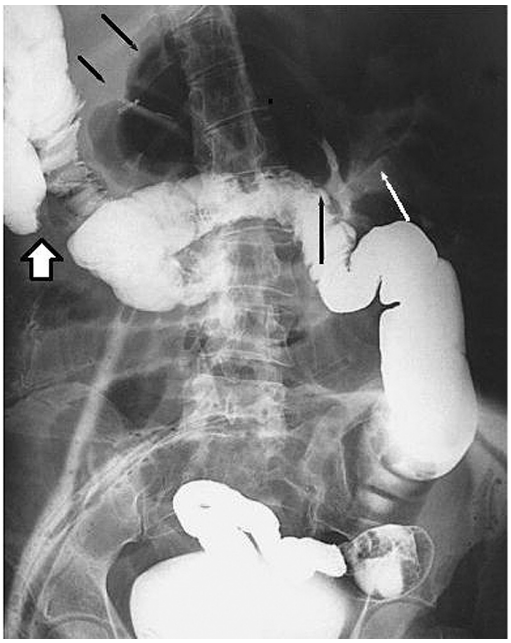


Figure 4.24. *Barium enema of cecal volvulus: contrast stops at hepatic flexure (arrow head) and air filled cecum crosses midline of abdomen in LUQ*

Cecum is greatly dilated, distended small bowel loops are often present, and the terminal ileum may be filled with air and visualized in an abnormal position to the right of the distended cecum. A single, long air-fluid level is usually noted in the distended cecum, there may be a relative absence of gas in the transverse and left colon.

Many authors advocate a preoperative water-soluble contrast enema examination to confirm the diagnosis and to exclude a concomitant obstructing lesion of the left colon, but this examination is potentially dangerous.

Treatment. The treatment of choice for volvulus of the right colon without gangrene is discussed controversially. Cecostomy and cecopexy (Fig. 4.25) have been recommended but postoperative mortality and recurrence rates were high. Total resection of the right colon eliminates the risk of recurrence. Colic resection is easy to realise because of the mobility of the right colon. Postoperative mortality after resection is about 8% without gangrene and 26% with gangrene.

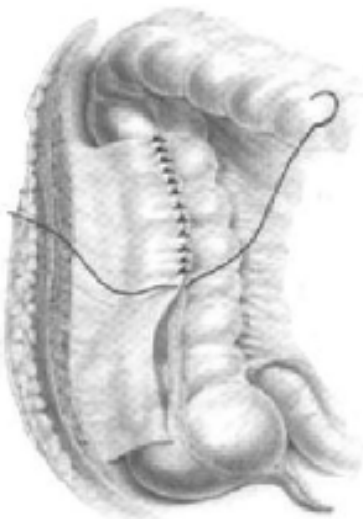


Figure 4.25. *Cecopexy*

Many authors advocate right hemicolectomy as the method of choice for the treatment of volvulus of the right colon, even in the absence of gangrene.

Volvulus of the transverse colon and the splenic flexure. Volvulus of the transverse colon and the splenic flexure is uncommon. Less common area for volvulus (~ 4%). Transverse colon volvulus associated with mobile right colon, distal obstruction, chronic constipation, congenital malrotation of the midgut.

Diagnosis is generally made at laparotomy. The principles of treatment resection of transverse colon with colostomy.

Volvulus of the sigmoid. The sigmoid colon is the most common site for colonic volvulus occurring often in patients over 60 years with a history of chronic constipation. Other contributing factors may include neurologic or psychiatric disorders (neuropsychotropic drugs alter the bowel motility), adhesions from previous surgery, and pregnancy. Acute volvulus sigmoid colon accounts for 1–10% of all intestinal obstructions and 60% of all cases of colonic volvulus. Rotation of a segment of bowel around its mesenteric axis that is sufficient to cause a complete or partial obstruction of the lumen and a variable degree of impairment to its vascular supply. Can only occur if the two ends of the segment that are twisted are in close approximation.

Clinical features. The symptoms of volvulus of the sigmoid are colicky abdominal pain, complete constipation, and gross, usually asymmetric distention of the abdomen. Physical examination reveals marked abdominal tenderness frequently with a palpable tympanitic mass. Rectal examination commonly shows absence of feces. If gangrenous changes have occurred, tachycardia, toxicity and peritonitis may be present.

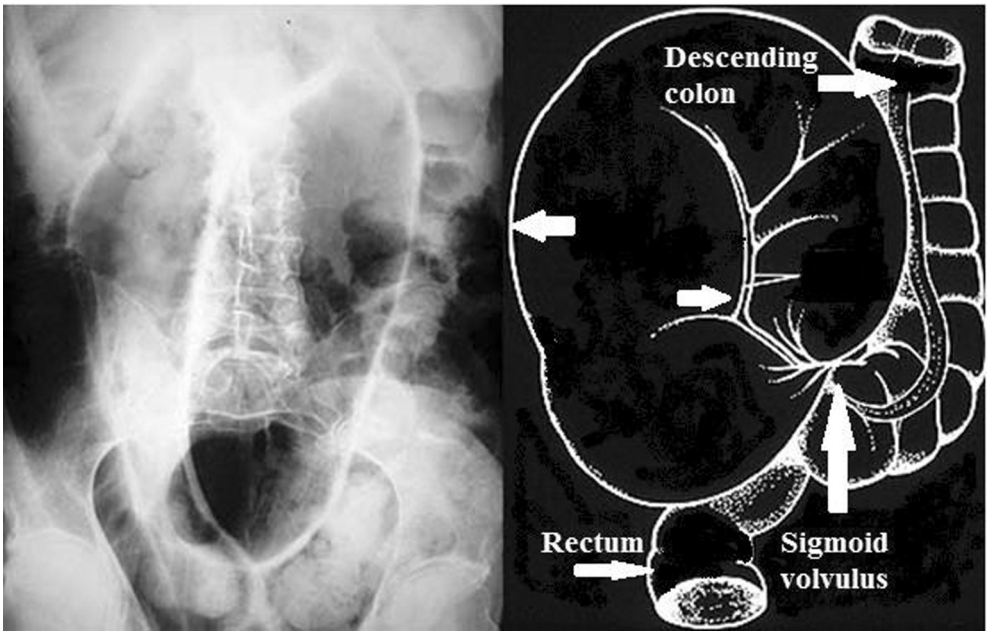


Figure 4.26. *Plain-film radiograph of abdomen: shows a very dilated single loop of colon in the left abdomen with both ends toward the pelvis and the center superiorly positioned – looking like a "bent innertube"*

Diagnosis. A plain X-ray examination of the abdomen reveals dilated loops of large bowel forming the "omega loop" sign, with the convexity of the loop lying away from the site of obstruction. Pointing towards the obstruction is the "bird's beak". Volvulus can be confirmed in 80% by a plain-film of the abdomen (Fig.4.26) and barium enema ("bird's beak" or ace of spades – pathognomonic of volvulus) (Fig. 4.27).

Mesocolon "whirl" sign around the superior mesenteric artery is pathognomonic of volvulus on CT scan (Fig. 4.28).

Treatment. The initial treatment is an attempt at deflation and untwisting of the sigmoid loop by the passage of a well lubricated rubber rectal tube through a sigmoidoscope with the patient in the lateral knee-chest position. After decompression,

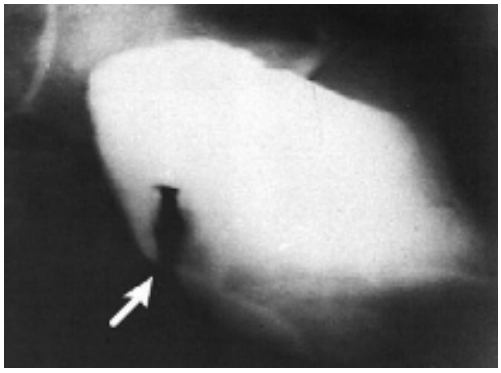


Figure 4.27. *Radiograph of abdomen: "bird's beak" on barium enema (arrow)*

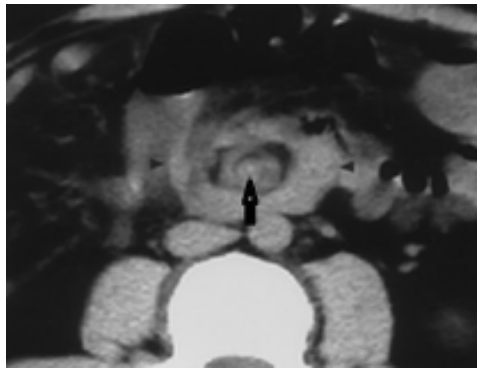


Figure 4.28. *CT scan: mesocolon whirl (arrow)*

the involved segment usually undergoes spontaneous detorsion, the immediate escape of flatus and liquid faeces through the sigmoidoscope or catheter indicates that the obstruction has been removed. The tube is left in place for 48 hours. This treatment can be expected to be successful in 80% of patients. If the segment is viable at exploration simple detorsion should be performed, further treatment should be considered because of the high recurrence rate (35–60%). Elective resection of the sigmoid loop following adequate preparation with primary anastomosis should be performed through laparotomy or laparoscopic approach.

If decompression is unsuccessful or if mucosa of doubtful viability is seen, immediate laparotomy is mandatory. The resection may be accompanied by primary anastomosis with peroperative colonic lavage in most of the cases. When gangrene is associated with peritonitis a Hartmann's procedure may be performed, eventually accompanied by Mikulicz's drainage. Colostomy alone is contraindicated because it will not prevent recurrent volvulus.

Differential diagnosis:

- Colon cancer.
- Diverticular disease.
- Extrinsic compression from metastatic carcinoma.
- Hernia
- Intussusception .
- Fecal impaction.
- Paralytic ileus.
- Toxic megacolon.

4.6.6. OTHERS

Endometriosis. Endometriosis may affect rectum and sigmoid of fertile female patients. The intestine is involved in 12–37% of cases. Intestinal endometriosis is usually asymptomatic and complete obstruction of the bowel lumen occurs in less than 1% of cases. Endometriosis usually does not produce complete obstruction but endometriosis may extend circumferentially round the bowel producing a stenosis. Symptoms related to the menstrual cycle and the length of the history may suggest the diagnosis of endometriosis. At laparotomy endometriosis may mimick a constricting colon carcinoma. Establishing an accurate pre- and peroperative diagnosis is very difficult.

Fecal impaction. Fecal impaction is a common cause of obstruction in elderly, chronically ill, bed-ridden patients and can be detected by rectal examination. When the colon has been emptied of fecal material by enemas repeated over a period of several days, colonoscopy or barium contrast enema may be necessary to exclude other causes of obstruction.

4.7. MESENTERIC ISCHEMIA

Mesenteric ischemia is a condition characterized by high mortality. It does not occur very frequently but when it does, the diagnosis is often made too late. Several new approaches have been suggested but mortality still remains in the same high range for the last decades.

4.7.1. DEFINITION, PATHOGENESIS AND EPIDEMIOLOGY

Mesenteric ischemia is defined as a condition in which the supply of oxygen is too small to satisfy the needs of the intestines.

Ischemia can affect the small intestine, the colon or both. It can be acute and chronic.

Most often mesenteric ischemia is classified as occlusive or non-occlusive.

Probably the most common cause of acute occlusive mesenteric ischemia is strangulation. This variant will, however, not be dealt with in this chapter. Other common causes of occlusive intestinal ischemia are arterial emboli, arterial thrombosis, complications of aortoiliac surgery and venous thrombosis. Unusual causes of occlusive mesenteric ischemia include trauma and small vessel disease. Acute non-occlusive mesenteric ischemia occurs as a consequence of other critical diseases such as shock and heart failure. Non-occlusive intestinal ischemia can also be induced pharmacologically by e.g. digitalis and vasoactive drugs. Chronic ischemia can be caused by atherosclerosis, fibromuscular dysplasia, inflammatory disease (Takayasu), and be a consequence of radiation injury. It can also be congenital as a part of aortic coarctation.

The pathogenesis in occlusive ischemia is most often an abrupt occlusion of a major vessel resulting in a significant reduction in intestinal blood flow. It is, however, not unusual that one finds two of the three supplying major arteries to be occluded without signs of mesenteric ischemia. That probably requires that the process has taken some time to allow collateral blood flow to develop and that the remaining vessels are healthy. In the elderly patient with atherosclerotic vessels, which is a common situation in the western world, sudden obstruction of blood flow by an embolus in the superior mesenteric artery is likely to impair blood flow enough to cause bowel infarction. If ischemia is total or near total it takes 8–16 hours to develop transmural infarction. This is, thus, the time frame during which the diagnosis has to be made and appropriate actions started in order to allow measures to prevent bowel infarction. After this period removal of dead bowel is all that can be achieved surgically and the prognosis is then dependent on the extent of bowel necrosis.

In the non-occlusive forms time course and extent of bowel injury is less easy to predict. Small intestinal blood flow can be reduced to about half of normal and the bowel can compensate for this reduction in oxygen delivery by increased oxygen extraction. (As a consequence the liver might become quite hypoxic). If blood flow is

about 5% of normal or less then ischemia is total or near total. In between, e.g., reduction down to 10–40% of the normal level, ischemia causes mucosal injury but not transmural infarction even if it becomes quite prolonged. This mucosal injury is considered important in the sense that it further exaggerates the underlying shock situation by release of various toxic factors from the intestine including bacteria and endotoxin – a process often referred to as translocation. Impairment of the intestinal immune function and the associated liver ischemia may also contribute to the aggravation of the underlying disease. The mucosal injury caused by this degree of intestinal ischemia, is likely to be exacerbated at reperfusion by increased generation of oxygen free radicals.

The colon seems to be affected less often by ischemia than the small intestine. One exception is focal non-occlusive colonic ischemia affecting the splenic flexure. The ischemic episode often remains undiscovered and these patients are later found to have a stricture at barium enema. This stricture sometimes can be mistaken for a neoplastic one. Colon is also affected in ischemia caused by surgery for abdominal aortic aneurysm.

The frequency of acute mesenteric ischemia is low. It has been reported that less than 1% of all acute laparotomies are performed because of acute mesenteric ischemia. Arterial thrombosis and embolus make up the two most common occlusive causes of acute mesenteric ischemia (approximately 50 and 33%, respectively). Most often the patient with a mesenteric embolus has an atrial fibrillation as the source of the embolus. Venous mesenteric thrombosis is the cause of acute mesenteric ischemia in about 10–15% of all cases. The onset of ischemia may be significantly prolonged in some patients with venous thrombosis.

4.7.2. CLINICAL STAGES

Irrespective of etiology four clinical stages are usually recognized. As mentioned above in arterial embolism the onset of symptoms is often very quick and the progression of symptoms rapid while the process can take several days following venous thrombosis.

The first stage is **the hyperactive stage**. This is characterized by the intermittent severe pain which starts immediately after occlusion of the vessel. Frequently there is passage of loose stools, sometimes with blood, and vomiting. Usually there is a discrepancy between the often very severe pain and the few findings on physical abdominal examination. Ischemia causes hyperperistalsis reflected in hyperactive bowel sounds on auscultation.

The paralytic stage. The pain is usually diminishing but becomes more continuous and diffuse. During this stage the size of the abdomen increases and it becomes generally tenderer and there are no bowel sounds at auscultation.

The stage of disarranged fluid balance. Fluids containing proteins and electrolytes start to leak through the mucosal as well as the serosal side of the gut. When the bowel becomes necrotic peritonitis develops. The fluid loss is usually massive. In this stage the patient does not differ much from other patients suffering from peritonitis of other causes.

The Shock stage. In this stage patients are rapidly deteriorating with severe alterations in the fluid balance and the situation soon goes over into irreversible shock.

4.7.3. DIAGNOSIS

Rapid diagnosis is essential in order to improve the high rate of detrimental outcome in acute mesenteric ischemia. A high degree of suspicion is the single most important factor in order to achieve diagnosis while treatment still could be corrective.

When the patient is in the hyperactive symptomatic stage, at laparotomy the gut could still be saved if an embolectomy, thrombectomy or a reconstruction could be performed.

Plain X-ray is unspecific until very late when gas can be seen in the bowel wall and in the mesenteric veins.

Duplex ultrasonography can be diagnostic in the hyperactive stage but in the later stages there is usually too much gas to allow reliable readings.

Angiography can be diagnostic and has been advocated strongly by some. Others have argued angiography may cause unacceptable delays in the handling of these patients. If angiography is performed and if there is a non-occlusive disease the catheter can be used for local infusion of vasodilating drugs as papaverin as advocated by *Boley* and co-workers.

Ischemia following aortoiliac reconstructive surgery constitutes a special situation. Clinical colonic ischemia occurs in 2.3% of all cases increasing to 7.3% after surgery for ruptured aortic aneurysm with the patient in shock. If the patients are routinely followed up by endoscopy mucosal ischemia is seen in about 10% of the cases.

Early passage of stools postoperatively, especially if bloody, is an important warning sign. Other signs that might indicate ischemia include failure to improve as expected, increasing creatinine concentrations in serum and extensive thrombocytopenia. The vast majority of the ischemic lesions following surgery for aortic aneurysm are within reach by the sigmoidoscope.

4.7.4. TREATMENT

If the diagnosis is made before bowel gangrene has developed (or if there is only a minor localized gangrenous area which could be resected although surrounded with ischemic but not yet gangrenous intestine) and if there is a localized obstruction an embolectomy or thrombectomy is the best treatment, although not supported by RCTs (Fig. 4.29).

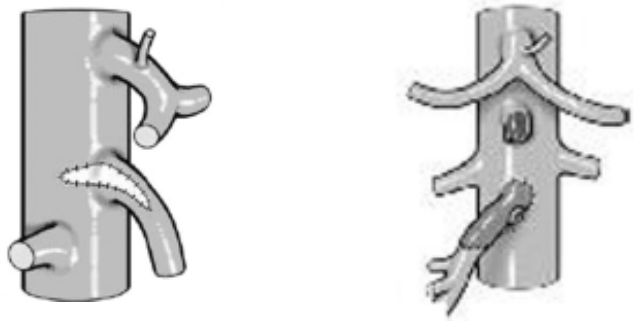


Figure 4.29. *Open endarterectomy and reconstruction by piece of autologous vein (1). Cutting and reinplantation with reconstruction by autologous vein (2) by M. Betzler, 1998*

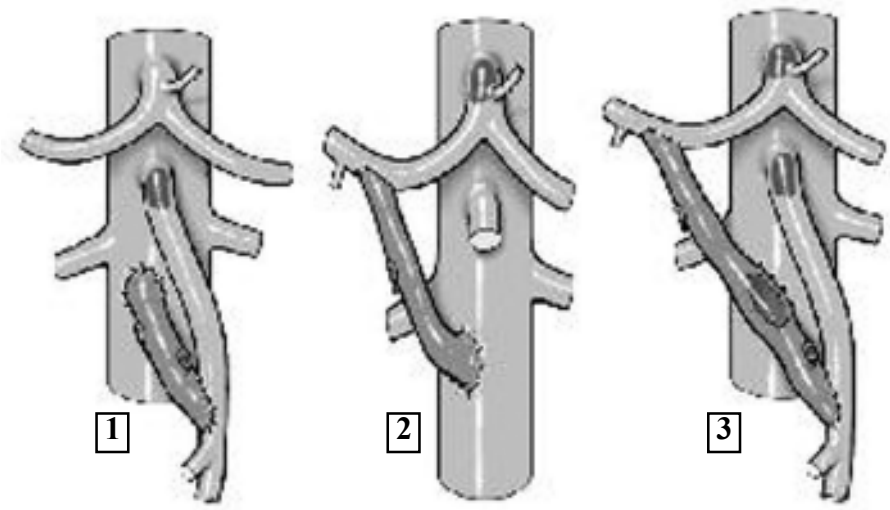


Figure 4.30. *Variants of autologous vein shunts by M. Betzler, 1998: 1 – between aorta and superior mesenteric artery; 2 – between aorta and common hepatic artery; 3 – aorta-hepatico-mesenteric shunts*

If there is a central stenosis in the superior mesenteric artery with low flow after thrombectomy, this is best treated by implanting the infrapancreatic part of the artery end to side in the infrarenal aorta or by a short bypass. Reconstruction of the superior mesenteric artery close to the aorta is warned against because of the difficult anatomic position (Fig. 4.30).

Following this type of surgery, with or without simultaneous bowel resection, the rule should be to perform a second look 12–24 – hours later. This decision should be made at the time of the first operation and should basically not be discussed thereafter.

Mortality following second look surgery is generally 65–85%. It is, however, 100% if nothing is done.

In transmural colonic ischemia following aortic aneurysm surgical resection of the affected bowel segment should be performed. Closing the distal end of the colon blindly and an end colostomy (the Hartmann procedure) is then a safe operation. If the bowel segment is removed before signs of peritonitis are visible the prognosis is excellent.

If there is significant bowel infarction at the time of surgery all that could be done is removal of the dead gut.

In case that the remaining part of the gut would allow oral nutrition and a normal life, resection should be performed. It is often advised to perform a "second look" 24 hours later and this decision should be made at the time of primary surgery, as stated above. If, however, the entire small intestine is gangrenous and the patients has peritonitis it is most often too late to save the life of the patient and often nothing is done at laparotomy in such cases.

4.8. PSEUDOOBSTRUCTION (OGILVIE'S SYNDROME)

Ogilvie's syndrome – dilation of the bowel in the absence of a causative anatomic lesion. Distention of colon with signs and symptoms of colonic obstruction without a mechanical cause for the obstruction. May be acute or chronic: *acute:* usually

involves only colon, and more commonly effects patients with chronic renal, respiratory, cerebral or cardiovascular disease; *chronic:* can effect other parts of the GI tract and tends to recur.

Pseudoobstruction may be primary or secondary: *primary* pseudoobstruction – a motility disorder (familial visceral myopathy; diffuse disorder involving autonomic innervation of intestinal wall); *secondary* associated with neuroleptics, opiates, metabolic illness, myxedema, uremia, lupus, scleroderma, Parkinson's, traumatic retroperitoneal hematomas.

4.8.1. DIAGNOSIS

- Water soluable contrast enema (can differentiate between mechanical and pseudoobstruction).
- Colonoscopy (can also be used for treatment).

4.8.2. INITIAL TREATMENT

- Resuscitation.
- Parasympathomimetic.
- Rectal tube/enemas /exams (work in most).
- Colonoscopic decompression (80–90% effective).
- Surgery (cecostomy vs. resection) – cecum >12 cm or peritoneal signs.

Chapter 5

HERNIAS. COMPLICATIONS OF HERNIAS

A hernia is the protrusion of an organ or the fascia of an organ through the wall of the cavity that normally contains it. A hernia occurs when the contents of a body cavity bulge out of the area where they are normally contained. These contents, usually portions of intestine or abdominal fatty tissue, are enclosed in the thin membrane that naturally lines the inside of the cavity. Although the term hernia can be used for bulges in other areas, it most often is used to describe hernias of the lower torso (abdominal-wall hernias).

HISTORICAL

The early history of interest in hernia is that of the discipline of surgery.

15th century – Castration with wound cauterization or hernia sac debridement

The Egyptian papyri do not contain reference to the operative treatment of hernia, but the Papyrus (1552 B.C.) recommended diet and externally applied pressure (truss) for its treatment. The word barbaric is frequently used in terms of surgery during the Middle Century and no less so for the treatment of hernia. Major developments in the knowledge of hernia anatomy and treatment occurred during the eighteenth century.

The nineteenth century brought anesthesia, hemostasis, and antisepsis, which made modern surgery possible. As in every area of surgery, these advances allowed rapid development of the science of hernia surgery (first of all inguinal hernia). Wide acceptance was soon attained in Europe and America for the operation; consisting of ligature and excision of the sac at the external ring and suturing of the pillars around the cord to reduce the size of the ring. This procedure was described in 1877 by Czerny. It is to Marcy of Boston that the modern era of hernial surgery is credited. His understanding of the importance of the transversalis fascia and of the anatomic contribution of fascial repair of the internal ring was reported in 1871. Parenthetically, this was 12 years before Bassini did his first operation for hernia, and 16 years before Bassini published his first paper on the subject.

It remained for Bassini to present a reconstruction technique of the inguinal floor with transposition of the cord. His operation (1884) included high ligation of the sac and reinforcement of the floor of the canal by suturing the con-joined tendon to the inguinal ligament beneath the cord, thus placing the cord under the external oblique aponeurosis. Bassini at this time held the chair of clinical surgery at the University of

Padua. Independently and almost simultaneously, Halsted (1852–1922), developed an operation similar to that of Bassini. The Halsted operation (Halsted I) transposed the cord above the external oblique aponeurosis. This procedure was first mentioned in 1889.

Eduardo Bassini – Father of Modern Inguinal Hernia Repair

5.1. HERNIA: EPIDEMIOLOGY DEFINITIONS

- Latin for *"rupture"*.
- An abnormal protrusion of an organ or tissue through a defect in its surrounding walls.
- Occur at sites where aponeurosis and fascia are not covered by striated muscle

Epidemiology:

- 700,000 hernia repairs yearly.
- Inguinal hernias – make up 75% of all hernias.
- 2/3 indirect, remainder are direct hernias.
- Incisional hernias – make up 15 to 20% all hernias.
- Umbilical and epigastric – make up10% all hernias.
- Femoral – 5% of all hernias.
- Prevalence of hernias increases with age.
- Most serious complication – *strangulation*.
- 1 to 3% of groin hernias.
- Femoral hernias have the highest rate of complications 15% to 20%.
- It is recommended that all hernias be repaired at the time of discovery.

Hernia is the protrusion of the visceral peritoneum or part of the visceral peritoneum through an abnormal opening. This opening can be natural, for example – Umbilical, Inguinal channel, Femoral ring, Petit's triangle or can develop after trauma, operations and diseases.

Parts of Hernia: the *port (gate)*, the *sac* and the *contents*. The port is the opening in muscle apeneurosis layer. The form of the port can be different; it can be ovoid, ring-like, and triangular and so on. The size of the port can be 20–30 cm. (Fig. 5.1).

The sac is part of the parietal peritoneum, which is subdivided into neck, body and top. The sac can be Uni-chamber and Multi-chamber. As usual, the contents of sac can be motile organs of abdomen cavity (loops of small intestine, omentum, transverse colon, sigmoid colon, Uterus e.t.c).

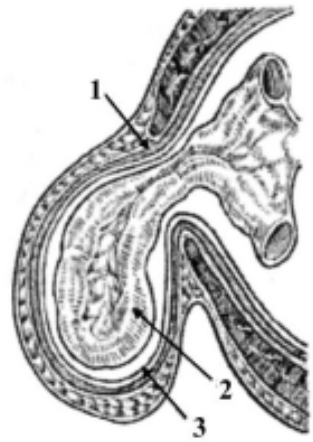


Figure 5.1. *Parts of Hernia: 1 – port; 2 – contents; 3 – sac*

5.2. ETIOLOGY AND PATHOGENESIS

The main etiological period of development of hernia is the disturbance of dynamic balance between intra-abdominal pressure and capacity of the abdominal wall to counteract it.

A powerful muscular effort or strain occasioned by lifting a heavy weight, or indeed any condition which raises intra-abdominal pressure, is liable to be followed by a hernia. Whooping cough is a predisposing cause in childhood, while a chronic cough, straining on micturition or on defecation may precipitate a hernia in an adult. It should be remembered that the appearance of a hernia in an adult can be a sign of intra-abdominal malignancy. Premature infants have a high incidence of hernia.

Stretching of the abdominal musculature because of an increase in contents, as in obesity and in pregnancy, can be another factor. Fat acts as a kind of "pile-driver" for it separates muscle bundles and layers, weakens aponeuroses, and favors the appearance of paraumbilical, direct inguinal, and hiatus hernias.

5.3. LOCALIZATIONS OF HERNIAS

External: the groin (inguinal: indirect and direct; femoral hernia); the middle of the abdomen (epigastric hernia; umbilical hernia; paraumbilical hernia); abdominal wall around a previous incision (ventral hernia); spigelian hernia; lumbar hernia (Petit's Triangle hernia and Grynfeltt's hernia); perineal hernia (Fig. 5.2).

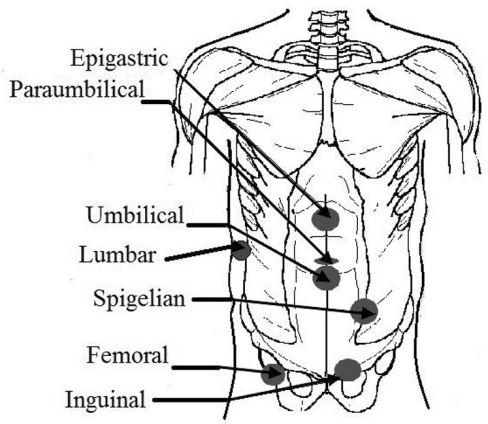


Figure 5.2. *External hernias*

Internal: around the esophagus (hiatal hernia) (Fig. 5.3); diaphragmatic hernia; obturator hernia; gluteal and sciatic hernias.

Hernia can be: congenial and acquired; reducible (the hernia either reduces itself) and irreducible; sliding; strangulated (obstructed); inflamed.

Acute complications: coprostasis; hernia inflammation; strangulated hernia.

Reducible hernia. The hernia either reduces itself when the patient lies down, or can be reduced by the patient or by the surgeon. Note that *intestine* gurgles on reduction, and the first portion is more difficult to reduce than the last. *Omentum* is doughy, and the last portion is more difficult to reduce than the first. A reducible hernia imparts an impulse on coughing.

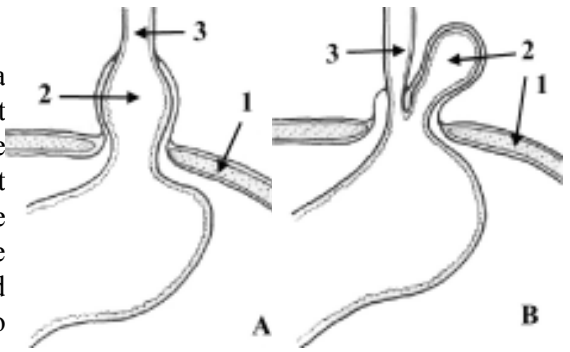


Figure 5.3. *Hiatus hernia: A – sliding hernia; B – paraesophagus hernia (1 – diaphragm, 2 – hiatus hernia, 3 – esophagus)*

Irreducible hernia. Here the contents cannot be returned to the abdomen and there is no evidence of other complications. It is brought about by adhesions between the sac and its contents or from overcrowding within the sac. Irreducibility without other symptoms is almost diagnostic of an omentocele especially in femoral and umbilical hernia.

Note: any degree of irreducibility predisposes to strangulation.

Sliding hernia. As a result of slipping of the posterior parietal peritoneum on the underlying retroperitoneal structures, the posterior wall of the sac is not formed of peritoneum alone, but by the sigmoid colon and its mesentery on the left, the caecum on the right and, sometimes, on either side by a portion of the bladder. It should be clearly understood that the caecum, appendix, or a portion of the colon *wholly within* a hernial sac does not constitute a sliding hernia. A small-bowel sliding hernia occurs once in 2000 cases; a sac less sliding hernia once in 8 000 cases.

A sliding hernia occurs almost exclusively in males. Five out of six sliding hernias are situated on the left side; bilateral sliding hernias are exceedingly rare. The patient is nearly always over 40, the incidence increasing with the weight of years. There are no clinical findings that are pathognomonic of a sliding hernia, but it should be suspected in every large globular inguinal hernia descending well into the scrotum. Large intestine is commonly present in a sliding hernia (or caecum and appendix in a right-sided case).

Occasionally large intestine is strangulated in a sliding hernia; more often nonstrangulated large intestine is present behind the sac containing strangulated small intestine.

Strangulated hernia. A hernia becomes strangulated when the blood supply of its contents is seriously impaired, rendering gangrene imminent. Gangrene may occur as early as 5 or 6 hours after the onset of the first symptoms of strangulation. Although inguinal hernia is four times more common than femoral hernia, a femoral hernia is more likely to strangulate because of the narrowness of the neck of the sac and its rigid walls.

The intestine is obstructed (except in a Richter's hernia, see below) and in addition its blood supply is constricted. At first only the venous return is impeded. The wall of the intestine becomes congested and bright red, and serous fluid is poured out into the sac. As the congestion increases, the intestine becomes purple in color. As a result of increased intestinal pressure the strangulated loop becomes distended, often to twice its normal diameter. As venous stasis increases, the arterial supply becomes more and more impaired. Blood is extravasated under the serosa (an ecchymosis) and is effused into the lumen. The fluid in the sac becomes bloodstained. The shining serosa becomes dull and covered by a fibrinous, sticky exudate. By this time the walls of the intestine have lost their tone; they are flabby, and are very friable. The lowered vitality of the intestine favors migration of bacteria through the intestinal wall, and the fluid in the sac teems with bacteria. Gangrene appears first at the rings of constriction which become deeply furrowed and grey in color, and then it appears in the antimesenteric border and spreads upwards, the color varying from black to green according to the decomposition of blood in the subserosa. The mesentery involved by strangulation also becomes gangrenous. If the strangulation is unrelieved, perforation of the wall of the intestine occurs, either on the convexity of the loop or at the seat of constriction. Peritonitis spreads from the sac to the peritoneal cavity.

5.4. CLINICAL FEATURES

Sudden pain, at first situated over the hernia, is followed by generalized abdominal pain, paroxysmal in character and often located mainly at the umbilicus. Vomiting is forcible and usually repeated. The patient may say that the hernia has recently become larger.

Risk factors. When a hernia is not repaired, it may become incarcerated or strangulated. When strangulation occurs, there is a danger that part of the intestine be caught in the hernia cutting off blood supply to the tissue. Also, when a bowel obstruction occurs, it leads to severe pain, vomiting, nausea and inability to have a bowel movement or pass gas. Men are more prompt to suffer inguinal hernias than women, and they risk a damage to their testicles if a hernia becomes strangulated. Also, the pressure caused on the hernia's surrounding tissues may extend into the scrotum causing pain and swelling.

On examination, the hernia is tense, extremely tender, irreducible and there is no impulse on coughing.

Unless the strangulation is relieved by operation, the paroxysms of pain continue until peristaltic contractions cease with the onset of gangrene when paralytic ileus (often the result of peritonitis) and toxic shock develop. Spontaneous cessation of pain is therefore of grave significance.

It is necessary to distinguish: typical infringement (**elastic and feacal**) and atypical (Richter's hernia, Littre's hernia, Maidl's hernia).

Richter's hernia is a hernia in which the sac contains only a portion of the circumference of the intestine (usually small intestine).

Strangulated **Richter's hernia** (Fig. 5.4) is particularly dangerous as operation is frequently delayed because the strangulated knuckle of bowel is small and there is no obstruction to the bowel lumen. A femoral site is common for this hernia and in a fat woman; the local signs of strangulation are often not obvious. The patient may not vomit, or vomits only once or twice. Intestinal colic occurs, but the bowels are often opened normally or there may be diarrhea; absolute constipation is delayed until paralytic ileus supervenes. Peritonitis often occurs before operation is undertaken.

The Maidl's hernia is seldom met (Fig. 5.5).In this form of hernia, hernial ring is restrained not only by **mesentery**; a loop being in a hernial bag, but, that is especially dangerous, mesenteric gut which is in the abdominal cavity.

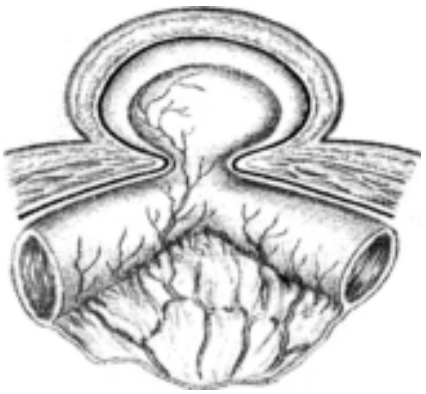


Figure 5.4. *Richter's hernia*

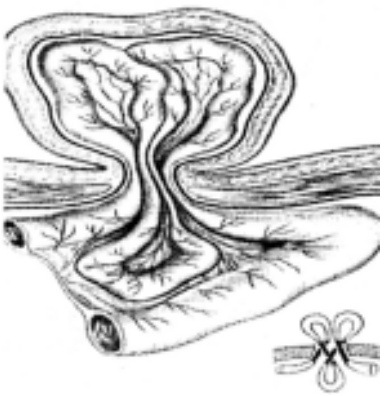


Figure 5.5. *Maidl's hernia*

Littre's hernia contains Meckel's diverticulum.

Inflamed hernia. Inflammation can occur from irritation or sepsis of the contents within the sac, e.g. acute appendicitis or salpingitis, also from external causes, e.g. from a sore caused by an ill-fitting truss. The hernia is tender but not tense, and the overlying skin becomes red and oedematous. Operation is necessary to deal with the cause.

EXTERNAL ABDOMINAL HERNIA

5.5. INGUINAL HERNIA

Indirect inguinal hernia. An indirect hernia follows the pathway that the testicles made during fetal development, descending from the abdomen into the scrotum (Fig. 5.6). This pathway normally closes before birth but may remain a possible site for a hernia in later life. Sometimes the hernia sac may protrude into the scrotum. An indirect inguinal hernia may occur at any age.

Direct inguinal hernia. The direct inguinal hernia occurs slightly to the inside of the site of the indirect hernia, in an area where the abdominal wall is naturally slightly thinner. It rarely will protrude into the scrotum. Unlike the indirect hernia, which can occur at any age, the direct hernia tends to occur in the middle-aged and elderly because their abdominal walls weaken as they age.

The inguinal canal, which is a tubular opening through the lower part of the abdominal wall, is one of those areas and the region where most hernias occur. In males, this canal contains the spermatic cord; in females, where the canal is not as developed, it contains the uterine round ligament (Fig. 5.7, 5.8).

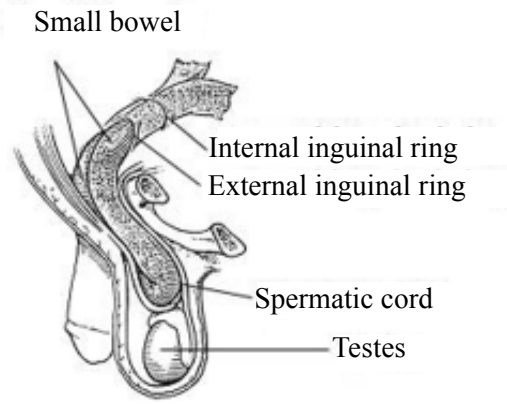


Figure 5.6. *Inguinal hernia*

An inguinal hernia can be of the "indirect" or "direct" variety; the former is most common. **Indirect hernias** begin at the deep inguinal ring where several abdominal muscles overlap; in these hernias the tissue migrates through the inguinal canal and into the scrotum. **In direct hernias** the deep inguinal ring is intact, but the tissue protrudes through a weakness in the floor of the inguinal canal above the pubic crest. All types of hernial repair basically involve pushing the tissue back into the abdominal cavity and repairing the defect.

Clinical presentation:

- Groin bulge (Fig. 5.9, 5.10, 5.11, 5.12).
- Often asymptomatic.
- Dull feeling of discomfort or heaviness in the groin.
- Focal pain – raise suspicion for incarceration or strangulation.
- Symptoms of bowel obstruction.

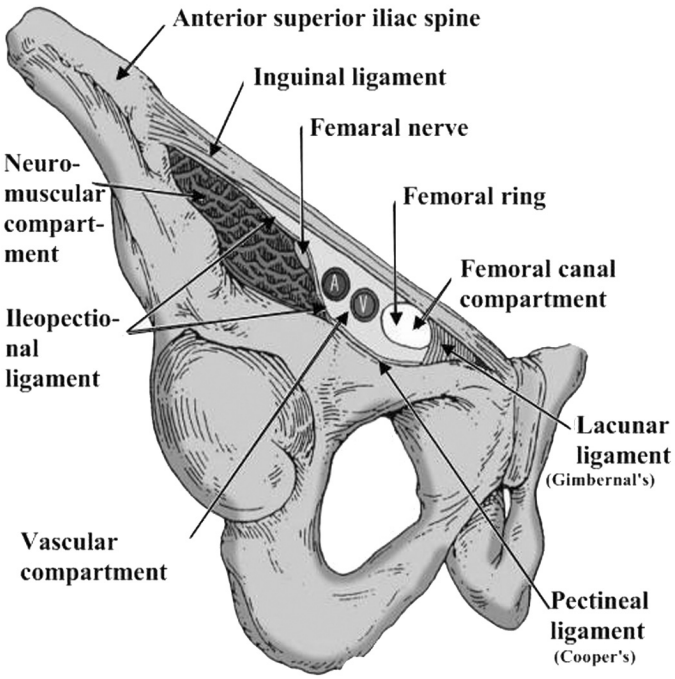


Figure 5.7. *Inguinal canal*

- Inguinal ligament (Poupart's) – inferior edge of **external oblique**.
- Lacunar ligament – triangular extension of the **inguinal ligament** before its insertion upon the pubic tubercle.
- Conjoined tendon (5–10%) – Internal oblique fuses with transversus abdominis aponeurosis.
- Cooper's Ligament – formed by the periosteum and fascia along the superior ramus of the pubis.

The characteristic of indirect inguinal hernia (Fig. 5.11):

- Is a congenital lesion.
- Occurs when bowel, omentum or other abdominal organs protrudes through the abdominal ring within a patent processus vaginalis.
- If the processus vaginalis does not remain patent an indirect hernia cannot develop.
- Most common type of hernia.

Note: the hernia sac passes outside the boundaries of Hesselbach's triangle and follows the course of the spermatic cord.

The characteristic of direct inguinal hernia (Fig. 5.12):

- Proceeds directly through the posterior inguinal wall.
- Direct hernias protrude medial to the inferior epigastric vessels and are not associated with the processus vaginalis.
- They are generally believed to be acquired lesions.
- Usually occur in older males as a result of pressure and tension on the muscles and fascia.

Note: the hernia sac passes directly through Hesselbach's triangle and may disrupt the floor of the inguinal canal.

Causes of groin hernias: divided into two categories congenital and acquired defects.

Physical examination:

- The patient should be standing and facing the examiner (Fig. 5.13).
- Visual inspection may reveal a loss of symmetry in the inguinal area or bulge.
- Having the patient perform valsalva's maneuver or cough may accentuate the bulge.

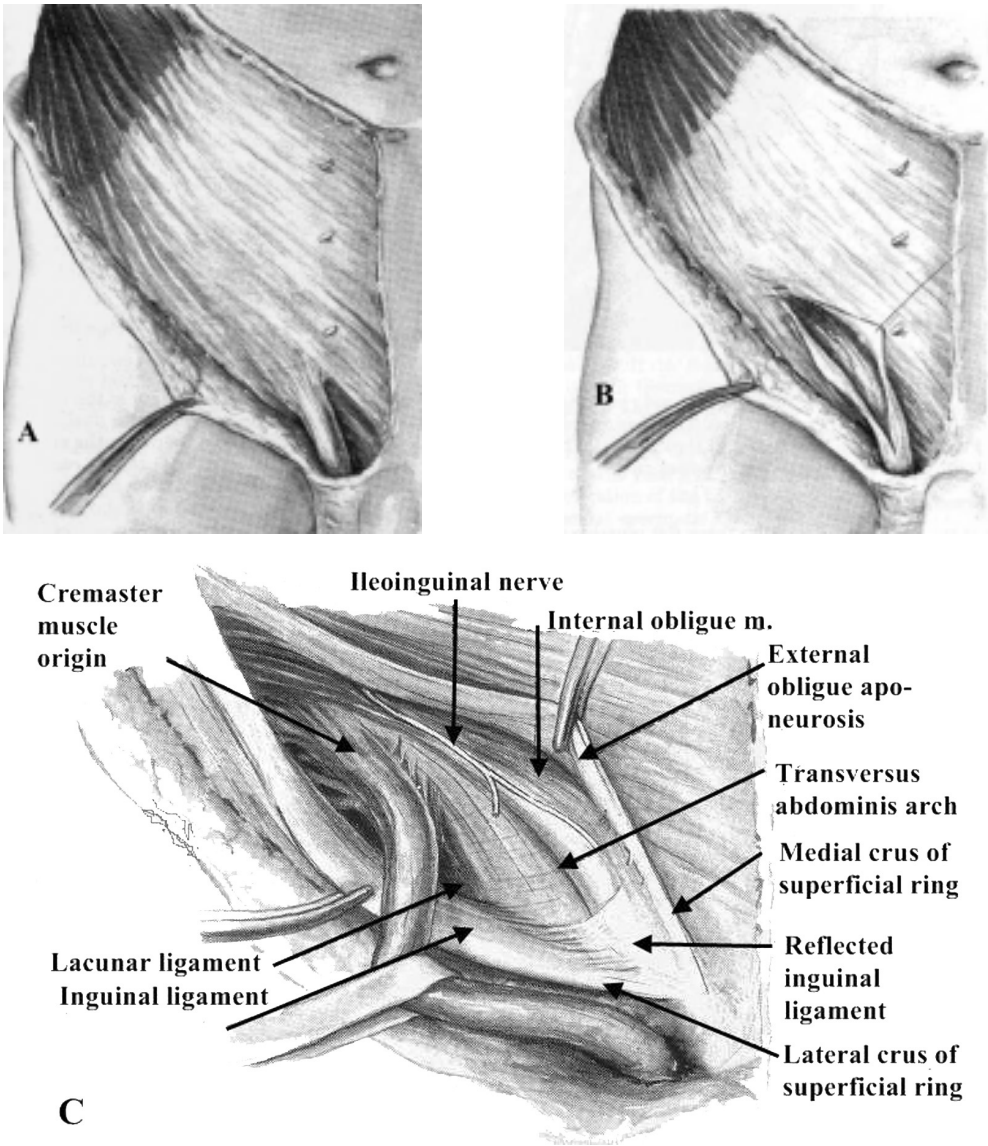


Figure 5.8. *Dissection of the inguinal canal:* ***A*** *– The intact external oblique lamina is depicted.* ***B*** *– The external spermatic fascia and innominate fascia have been incised through the superficial inguinal ring.* ***C*** *– The external oblique aponeurosis has been opened widely and the spermatic cord mobilized by transection of many of its areolar (cremasteric fascia) attachments to the walls of the inguinal canal*

- A fingertip is then placed in the inguinal canal; Valsalva maneuver is repeated.
- Differentiation between indirect and direct hernias at the time of examination is not essential. Allows the hernia contents to fill the sac, making the hernia obvious on examination:

Invaginate the scrotum near the external ring and direct the finger medial towards the pubic tubercle. The finger will thus lie on the spermatic cord with the tip of the finger within the external ring. The patient is then asked to cough or perform a Valsalva maneuver. Will be felt as a silk-like sensation against the gloved finger of the examiner… **"cough impulse"** sign.

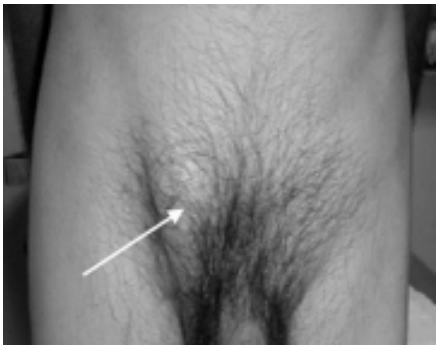


Figure 5.9. *Male inguinal hernia*

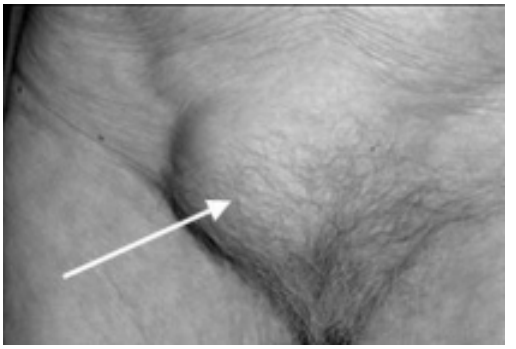


Figure 5.10. *Female inguinal hernia.*

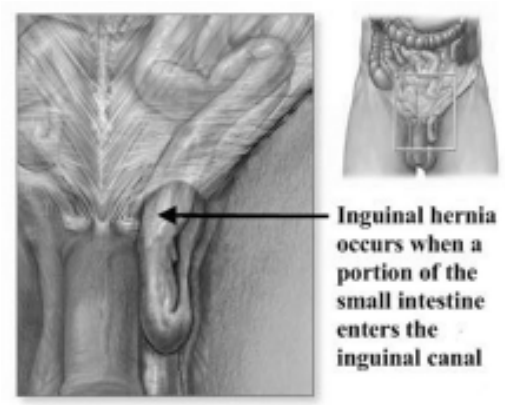


Figure 5.11. *Inguinal hernia (indirect)*

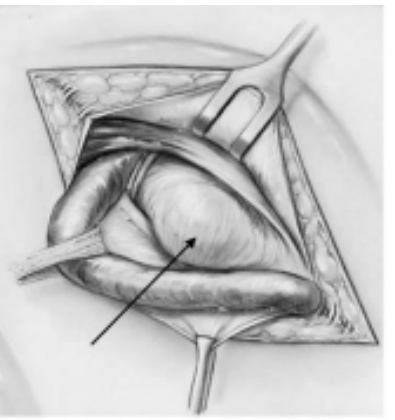


Figure 5.12. *Inguinal hernia (direct)*

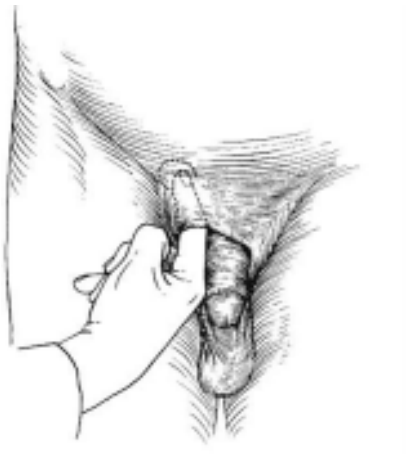


Figure 5.13. *Examine the patient in the standing positions*

The female patient does not have the long and stretched spermatic cord to follow with the examiner's finger during the physical examination. Instead, two fingers can be placed along the inguinal canal, and the patient is asked to cough or strain.

As an oblique inguinal hernia increases in size it becomes more obvious when the patient coughs, and persists until reduced. As time goes on the hernia comes down as soon as the patient assumes the upright position. In large hernias there is a sensation of weight, and dragging on the mesentery may produce epigastric pain. If the contents of the sac are reducible, the inguinal canal will be found to be commodious. During examination surgeon understands: Is the hernia right, or left, or bilateral? Is it an inguinal or a femoral hernia? Is it a direct or an indirect inguinal hernia? Is it reducible or irreducible? (Patient has to lie down for this to be ascertained). What are the contents – bowel, or omentum?

Note: examination using finger and thumb across the neck of the scrotum will help to distinguish between a swelling of inguinal origin and one which is entirely intrascrotal.

Radiologic investigations – herniography:

- Suspected hernia, but clinical diagnosis is unclear.
- Procedure done under flouroscopy following injection of contrast medium.
- Frontal and oblique radiographs are taken with and without increased intra-abdominal pressure.
- Ultrasonography.
- MRI.
- CT.

5.5.1. TREATMENT OF INGUINAL HERNIA

Indications for Operative Repair:

- Early repair is justified when potential for strangulation is weighed against minimal risks for surgery.
- Not warranted in terminally ill without incarceration.
- Patients with ascites should have it controlled before surgery.
- Incarceration, strangulation.

Surgical Techniques:

- Open anterior repair (Bassini, McVay, Shouldice).
- Open posterior repair (Nyhus, preperitoneal).
- Tension-free repair with mesh (Liechtenstein).
- Laparoscopic.

Operative treatment.

Operation is the treatment of choice. It must be remembered that patients who have a bad cough from chronic bronchitis should not necessarily be denied operation, for these are the very people who are in danger of getting a strangulated hernia. As these patients are often elderly, the surgeon should consider giving extra strength to the repair by a synchronous orchidectomy and full closure of the internal ring. In adults, local, epidural or spinal, as well as general anesthesia can be used.

Inguinal herniotomy is the basic operation which entails dissecting out and opening the hernial sac, reducing any contents and then transfixing the neck of the sac and removing the remainder. It is employed either by itself or as the first step in a repair procedure (herniorrhaphy).

Herniotomy and repair (herniorrhapy). This operation consists of (1) excision of the hernial sac (above), plus (2) repair of the stretched internal inguinal ring and the transversalis fascia, and (3) further reinforcement of the posterior wall of the inguinal canal. Stages (2) and (3) must be achieved without tension, usually by 'darning' with a monofilament suture material such as polypropylene. Fascial flaps, or synthetic mesh implants, are employed when the deficiency of the posterior wall is extensive.

5.5.2. OPERATIVE PROCEDURES

General principles. Excision *of the hernial sac* (*adult herniotomy)*. Before the skin incision in large inguinoscrotal hernias, the usual antiseptic preparation of the skin should not be extended to the perineal aspect of the scrotum, for, by so doing, severe bacterial contamination of the operation site is likely. The operation approach should be confined to the anterior inguinal and scrotal aspects. An incision is made in

the skin and subcutaneous tissues 1.25 cm above and parallel to the medial two-thirds of the inguinal ligament. In large irreducible herniae the incision is extended into the upper part of the scrotum. After dividing the superficial fascia and securing complete haemostasis, the external oblique aponeurosis and the superficial inguinal ring are identified. The external oblique aponeurosis is incised in the line of its fibers, and the structures beneath are carefully separated from its deep surface before completing the incision through the superficial inguinal ring. In this way the ilioinguinal nerve is safeguarded. With the inguinal canal thus opened, the upper leaf of the external oblique aponeurosis is separated by blunt dissection from the internal oblique. The lower leaf is likewise dissected until the inner aspect of the inguinal ligament is seen. The cremasteric muscle fibers are divided longitudinally to open up the subcremasteric space and display the spermatic cord, which is then lifted out.

Excision of the sac. The indirect sac is easily distinguished as a pearly white structure lying on the upper side of the cord and, when the internal spermatic fascia has been incised longitudinally, it can be dissected out and then opened between haemostats.

Variations in dissection. If the sac is small (e.g. bubonocele) it can be freed *in toto*. If it is of the long funicular or scrotal type, or is extremely thickened and adherent, the fundus need not be sought. The sac is freed and divided in the inguinal canal. Care must be taken to avoid damage to the vas deferens and the spermatic artery, including the blood supply to the epididymis.

An adherent sac can be separated from the cord by first injecting saline under the posterior wall from within. A similar tactic is used when dissecting the gossamer sac of infants and children.

Reduction of contents. Intestine or omentum is returned to the peritoneal cavity. Omentum is often adherent to neck or fundus of the sac; if to the neck, it is freed, and if to the fundus of a large sac, it may be transfixed, ligated and cut across at a suiTable site. The distal part of omentum, like the distal part of a large scrotal sac, can be left *in situ* (the fundus should not, however, be ligated).

Whatever type of sac is encountered, it is necessary to free its neck by blunt and gauze dissection until the parietal peritoneum can be seen on all sides. Only when the extraperitoneal fat is encountered and the inferior epigastric vessels are seen on the medial side has the dissection reached the required limit. If it has not been done already, the sac is opened. The finger is passed through the mouth of the sac in order to make sure that no bowel or omentum is adherent. The neck of the sac is transfixed and ligated as high as possible, and the sac is excised 1.25 cm below the ligature (Fig. 5.14).

Figure 5.14. *Isolation and ligation of the neck of the sac*

Reinforcement of the posterior inguinal wall is achieved by approximating without tension; the tendinous and aponeurotic part of the conjoined muscle to the pubic tubercle and to the undersurface of the inguinal ligament. The suturing method includes a choice between: simple interrupted stitches (Bassini type). After extraction of the hernial sac, the spermatic duct is clamped

and held. Sutures are placed between the borders of transverse muscle, internal oblique muscle, transverse fascia and inguinal ligament interrupted. Except that, few sutures are placed between the border of abdominal rectus muscle sheath and pubic bone periosteum. In the way, inguinal space is closed and posterior wall strengthened. Spermatic duct is placed on the new-formed posterior wall of the inguinal canal. Over the spermatic duct the aponeurosis is restored by interrupted sutures.

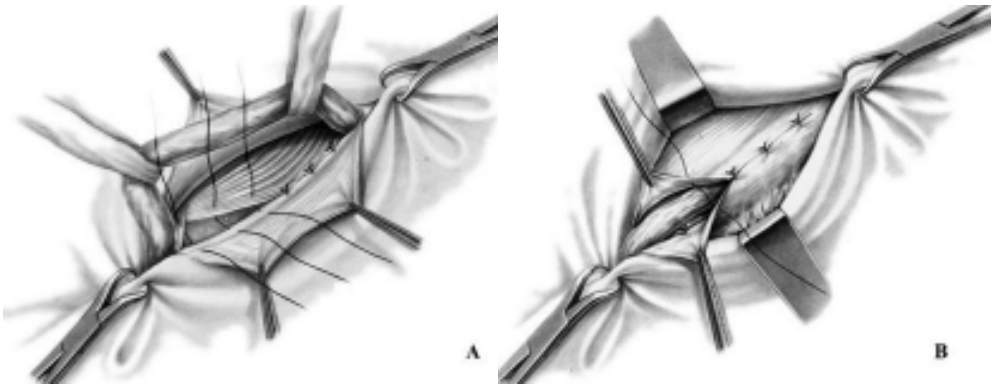


Figure 5.15. *Bassini repair*

Bassini Repair (early 20th Century):

- Is frequently used for indirect inguinal hernias and small direct hernias.
- The conjoined tendon of the transversus abdominis and the internal oblique muscles is sutured to the inguinal ligament (Fig. 5.15, A).
- It is sewn up the aponeurosis of external oblique abdominis muscle (Fig. 5.15, B).

Bassini's operation epitomized the essential steps for an ideal tissue repair. He opened the external oblique aponeurosis through the external ring, then resected the cremasteric fascia to expose the spermatic cord. He then divided the canal's posterior wall to expose the preperitoneal space and did a high dissection and ligation of the peritoneal sac in the iliac fossa. Bassini then reconstructed the canal's posterior wall in 3 layers. He approximated the medial tissues, including the internal oblique muscle, transversus abdominus muscle and transversalis fascia to the shelving edge of the inguinal ligament with interrupted sutures. He then placed the cord against that newly constructed wall and closed the external oblique aponeurosis over it, thereby restoring the step-down effect of the canal and reforming the external inguinal ring (Fig. 5.15).

There have been numerous modifications of Bassini's original technique, although many of the less detailed renditions have yielded poor results. Those that avoided opening the posterior wall, for example, resulted in suture-line tension between tissues at the most medial part of the inguinal canal just cephalad to the pubic bone. Some help was afforded the Bassini technique and other tissue repairs by the introduction of relaxing incisions by surgeons such as Wolfer, Halsted, Tanner, and McVay.

McVay Repair:

- Is for the repair of large inguinal hernias, direct inguinal hernias, recurrent hernias and femoral hernias.
- The conjoined tendon is sutured to Cooper's ligament from the pubic tubercle laterally (Fig. 5.16).

Note: this repair reconstructs the inguinal canal without using mesh prosthesis.

McVay and Anson revived Lothiessen's operation in 1942 (Fig. 5.16). They considered the superior pubic ligament to be the ideal structure for reconstructing the posterior wall of an inguinal hernia, since it shares the same tissue plane and is derived from the same tissue origin as the transversus aponeurosis and the transversalis fascia. However, many surgeons who attempted this procedure found that it was sometimes difficult to approximate the transversus arch to the Cooper ligament. Doing so frequently resulted in considerable suture-line tension – enough to require one or more relaxing incisions. Patients complained of considerable and prolonged postoperative pain, and failure rates became unaccep table. This procedure has, however, had value to surgeons by demonstrating the strength of the superior pubic ligament and showing its utility in large and difficult hernia repairs, including incisional hernias. It is a reliable structure to which prosthetic material can be fixed when a large defect must be spanned.

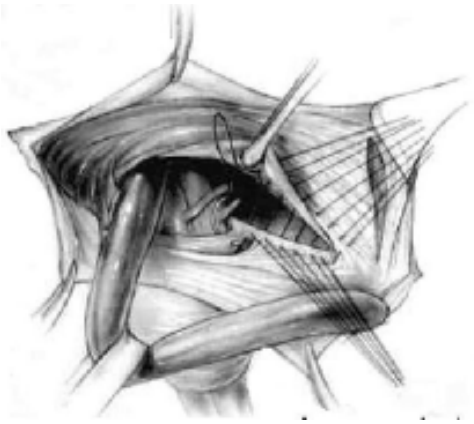


Figure 5.16. *McVay repair*

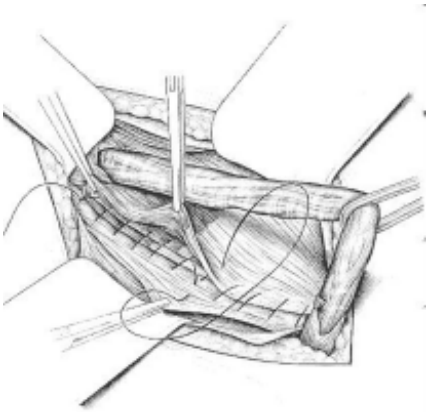


Figure 5.17. *Shouldice repair*

Shouldice repair. The Shouldice repair (Fig. 5.17) has been refined over several decades and is the gold standard for the prosthesis-free treatment of inguinal hernias. A recurrence rate around 1% has been consistently demonstrated over the years. The objective of this paper is to outline and highlight the key principles, including the dedicated pre-operative preparation, the use of local anesthesia, a complete inguinal dissection and the eponymous four-layered reconstruction. A knowledge and understanding of inguinal hernia anatomy and the pathophysiology of recurrence are vital to achieving a long-term success and patient satisfaction for a pure tissue repair.

Canadian surgeon E.E. Shouldice contributed substantially to hernia surgery in the second half of the 20th century. He founded a clinic that has since become a hospital devoted exclusively to the treatment of abdominal wall hernias. The Shouldice operation for hernia repair revitalizes Bassini's original technique. It applies the principle of an imbricated posterior wall closure with continuous monofilament suture. Local anesthesia is routinely used and bilateral hernias are usually repaired separately, 2 days apart. Patients walk to and from the operating room, begin exercise therapy on the day of surgery, and resume their usual activities within a reasonable time after the operation.

An unusual feature of the procedure is the routine sacrifice of the lateral cremasteric bundle, a structure that contains the external spermatic vessels and the

genital branch of the genitofemoral nerve. Shouldice surgeons have not reported any ill effects related to this step. In fact, before using this technique, pubic tubercle recurrences were unacceptably high. The minor sensation loss that results from dividing that nerve has not proven to be a substantial or longstanding disability. However, when the lateral flap of the cremasteric fascia is sacrificed, ptosis of the testicle will occur. This can be prevented by fixing the distal pedicle of the cremasteric fascia to the external oblique when the canal is restored.

The Shouldice repair has been considered the gold standard of hernia repairs for the last 4 decades, although its use has declined since the introduction of various tension-free prosthetic repairs. The Shouldice repair remains an excellent option, however, and has produced the best and most enduring results of any other pure tissue repair.

Use of prosthetics in hernia repair. The need for a satisfactory prosthesis for hernia repair has been recognized for more than a century. Various materials, including autografts (the patient's own tissue), have been tried. The most successful of the autografts is fascia lata, which has been used as suture material, a pedicle graft, and as a free transplanted graft. However, in addition to requiring a second operation to harvest it, fascia lata weakens and fails over time and dissolves in the presence of infection.

Artificial prostheses. Many authors have attempted to define characteristics of the ideal prosthetic material for hernia repairs (Table 5.1), although attempts to achieve this "ideal" have met with varying degrees of success (see Tables 5.2 , 5.3). No currently available prosthesis is perfect or free of problems, and the choice of material thus requires compromise. Surgeons do, however, have a large array of products from which to choose.

Table 5.1 – Characteristics of an ideal prosthesis

The ideal prosthetic mesh should:
Not be physically modified by tissue fluids
Be chemically inert
Not excite inflammatory or foreign body reaction
Be noncarcinogenic
Not produce allergy or hypersensitivity
Be capable of resisting mechanical strain
Be capable of being fabricated in the form required, and constructed in a way such that sutures or cutting will not cause the mesh to unravel or fray
Be sterilizable
Be permeable and allow tissue ingrowth within it
Stimulate fibroblastic activity to allow incorporation into tissue rather than sequestration or encapsulation
Be sufficiently pliable so as not to cause stiffness or to be felt by the patient
Strong enough to resist bursting by the maximum forces that can be created by intra-abdominal pressure or from an outer force

Table 5.2. – Metal prosthetic graft material

Silver filigree mesh (1900)	Became brittle and fractured and eventually extruded causing multiple sinuses and fistulas.
	Fractured and caused sinus formation.
Toilinox (stainless steel)	Set up electrolyte reactions between ingredients if composition varied.

Table 5.3. – Nonmetal synthetic prosthesis

Nylon (1944)	Replaced rubber, metals and animal products. Initially used for sutures, later knitted or woven into patches for hernia repair; disintegrates in tissue and loses most of its tensile strength within 6 months.
Polyethylene mesh (1958) Polypropylene mesh (1962)	High-density polyethylene mesh (*Marlex*, 1958) resistant to chemicals and sterilizable, but unraveled after being cut. Modified to polypropylene mesh (1962). Available under various trade names (*Hertra-2, Marlex, PROLENE, Surgipro, Tramex, Trelex*). Available as a flat mesh as well as 3-dimensional devices (*Altex, Hermesh3, PerFix Plug, PROLENE Hernia System*).
Polyester mesh (*MERSILENE*) (1984)	Composed of polyester fiber with the characteristics of filigree; can be inserted into narrow spaces without distortion.
Expanded polytetrafluoroethylene	Teflon product; produces minimal adhesions when placed intraperitoneally. Does not allow significant fibroblastic or angiogenic ingrowth; must be removed if infection occurs.
Polyglycolic acid mesh (*Dexon*) Polyglactin 910 mesh (*Vicryl*)	Absorbable mesh; loses strength after 8–12 weeks; should not be used as a sole prosthesis for the repair of abdominal or groin hernias

Complications related to the use of prosthetics. Materials composed of polypropylene and polyester insight a prompt and strong fibroblastic tissue response with minimal inflammation. This response consists of macrophages and giant cells, most of which eventually disappear. Fibroblastic activity allows rapid integration of the prosthesis into tissues; however, contraction of the enveloping scar tissue creates undesirable deformation of unsecured pieces of the monofilament; its free margins tend to curl, and small pieces roll up. There also have been some reports in the literature of freeform and preformed prosthetic mesh products migrating.

Serum or blood that accumulate in dead spaces surrounding any prosthesis becomes an excellent media for infection. Suction drainage is therefore advisable to

eliminate dead space as well as to remove serum collections. Intestinal obstruction and fistula formation are serious complications and often require removal of the mesh/ prosthesis. When a prosthesis is placed inside the peritoneal cavity, various degrees of visceral adhesions form depending upon the type of material used. When this is unavoidable, omentum or an absorbable prosthesis should be interposed between the mesh and the bowel.

Treatment of infection involves the application of basic surgical principles. Although most infections occur acutely, delayed infections involving nonabsorbable prostheses can occur months or years later. In the case of an acute infection of a groin hernia repair, it is advisable to quickly and widely open the wound (including the subcutaneous layer down to the external oblique) to avoid chronic sinus formation. A specimen should be taken for culture and sensitivity, irrigation and antibiotics started and healing observed by secondary intention. Frequent wound check to remove accumulated fluid is advisable.

If a prosthetic mesh had been used in the repair, it can usually be left in place if the above measures are employed promptly. If the wound closes, but a sinus continues to drain, it is likely that the mesh and all old suture material will need to be removed. Unlike early infection, when the mesh can be salvaged, late infection involving mesh requires the complete removal of the unincorporated material, although the incorporated mesh may be left undisturbed.

If the surgeon encounters an inflammatory granuloma in the course of repairing a recurrent inguinal hernia, it is prudent to avoid using a new prosthesis. Gram staining of the inflammatory granuloma at the time of surgery is not sufficiently reliable to exclude subsequent infection. In most cases of persistent infection related to a prior prosthetic repair, the culprit is the nature of the suture material rather than the graft itself. Multifilament and braided sutures, such as silk and cotton should be avoided.

Tension-free hernia repair. The most important advance in hernia surgery has been the development of tension-free repairs. In 1958, Usher described a hernia repair using *Marlex* mesh. The benefit of that repair he described as being "tension-eliminating" or what we now call "tension-free". Usher opened the posterior wall and sutured a swatch of *Marlex* mesh to the undersurface of the medial margin of the defect (which he described as the transversalis fascia and the conjoined tendon) and to the shelving edge of the inguinal ligament. He created tails from the mesh that encircled the spermatic cord and secured them to the inguinal ligament.

Every type of tension-free repair requires a mesh, whether it is done through an open anterior, open posterior, or laparoscopic route. The most common prosthetic open repairs done today are the *Kugel patch* repair, the *Lichtenstein* onlay patch repair, the *PerFix plug* and patch repair, and the *Prolene Hernia System* bilayer patch repair (Fig. 5.18, 5.19).

Lichtenstein Repair (1989):

- One of the most commonly performed procedures.
- A mesh patch is sutured over the defect with a slit to allow passage of the spermatic cord.

Techniques:

- Central feature is polypropylene mesh over unrepaired floor.
- Gilbert repair uses a cone shaped plug placed thru deep ring.
- Slit placed in mesh for cord structures.

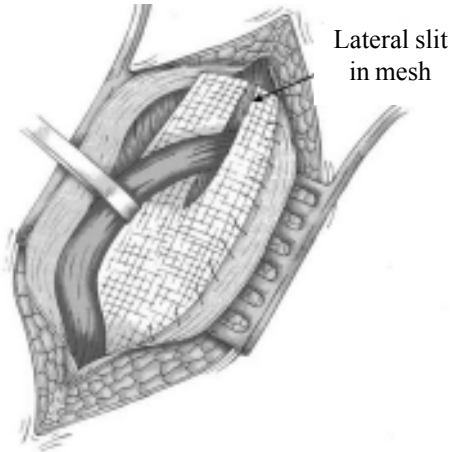


Figure 5.18. *Lichtenstein repair (1989): mesh is sutured from the transversus arch to the shelving edge of the inguinal ligament creating a "tension-free" repair*

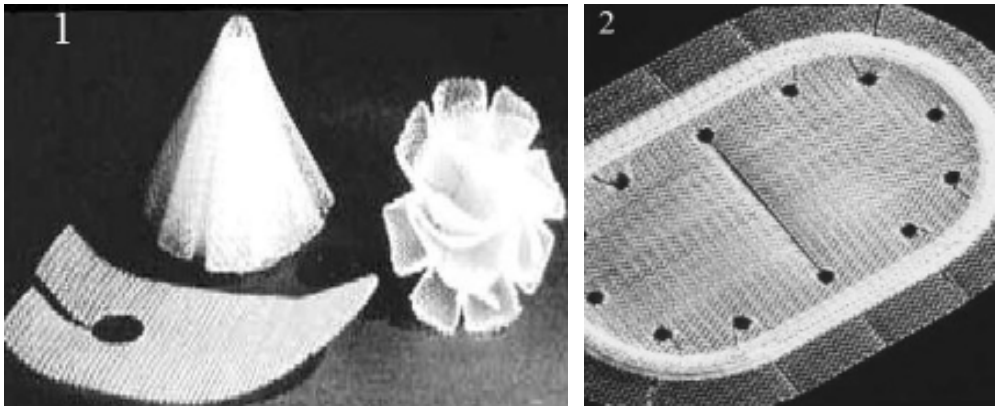
The mesh is attached to the inguinal ligament, the coverings of the pubis, the rectus sheath and the conjoined aponeurosis and tendon. It is also fashioned by a slot laterally above and below the cord at the internal ring (Fig. 5.18).

The PerFix Plug repair of direct and indirect hernias is an adaptation of Gilbert's free-formed umbrella-shaped plug, which was initially used as a plug in the internal ring for treatment of indirect inguinal hernias (Fig. 5.19 – 1). Currently, the PerFix Plug is manufactured and placed in the internal ring and fixed with sutures to the surrounding tissues. When a direct hernia is present, the PerFix Plug is used in a similar fashion. When pantaloon or unusually large direct hernias are present, multiple plugs are sewn together to repair the defect. In addition to the plugs, an onlay patch is provided, which can be used with or without sutures over the posterior wall and around the spermatic cord lateral to the internal ring.

The Kugel patch is a polypropylene, oblong-shaped mesh with a thickened polypropylene thread that encourages the mesh to flatten. The Kugel approach is through a small incision above the internal ring. The preperitoneal space is entered and dissected free. An indirect sac is retracted or transsected, and the threshold of the internal ring is thereby freed of the peritoneal sac. The prosthesis is inserted and fixed in place with a single suture and then held in place by the natural intra-abdominal forces (Pascal's principle) (Fig. 5.19 – 2). In this procedure, the spermatic cord anterior to the interior ring is not handled. This approach is designed to protect the internal ring and posterior groin wall as well as the femoral canal.

The Prolene Hernia System bilayer patch device (Fig. 5.19 – 3) has a combined onlay graft (like a Lichtenstein repair) and underlay graft (like a Stoppa or Kugel patch); these are held together by a connector (like a plug). The external oblique aponeurosis is opened through a 5 cm groin incision, the spermatic cord is elevated, and an anterior space is created for placement of the onlay component of the device. If an indirect hernia sac is present, it is invaginated through the internal ring. If a posterior wall hernia is present (direct hernia), the defective tissue is circumscribed. In either case, the preperitoneal space (space of Bogros) is dissected free with a 4 x 4 sponge (Fig. 5.20).

The PerFix Plug repair of direct and indirect hernias is an adaptation of Gilbert's free-formed umbrella-shaped plug, which was initially used as a plug in the internal ring for treatment of indirect inguinal hernias (Fig. 5.19 1). Currently, the PerFix Plug is manufactured and placed in the internal ring and fixed with sutures to the surrounding tissues. When a direct hernia is present, the PerFix Plug is used in a similar fashion. When pantaloon or unusually large direct hernias are present, multiple plugs are sewn together to repair the defect. In addition to the plugs, an onlay patch is provided, which can be used with or without sutures over the posterior wall and around the spermatic cord lateral to the internal ring.



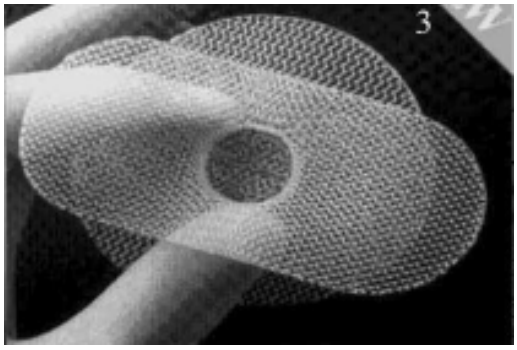


Figure 5.19. *PerFix plug (1) – flower-shaped polypropylene mesh plug with multiple petals, and onlay graft with slit to accommodate the spermatic cord; Kugel Patch (2) – "Race-track" oval shaped polypropylene mesh graft with pocket for insertion and larger gauge polypropylene ring to hold graft's flat shape; Prolene hernia system (3) bilayer patch repair. Bilayer polypropylene mesh. Three-in-one device with round disc for properitoneal repair, plug effect of connector, and oblong shaped onlay component*

The Kugel patch is a polypropylene, oblong-shaped mesh with a thickened polypropylene thread that encourages the mesh to flatten. The Kugel approach is through a small incision above the internal ring. The preperitoneal space is entered and dissected free. An indirect sac is retracted or transsected, and the threshold of the internal ring is thereby freed of the peritoneal sac. The prosthesis is inserted and fixed in place with a single suture and then held in place by the natural intra-abdominal forces (Pascal's principle) (Fig. 5.19 – 2). In this procedure, the spermatic cord anterior to the interior ring is not handled. This approach is designed to protect the internal ring and posterior groin wall as well as the femoral canal.

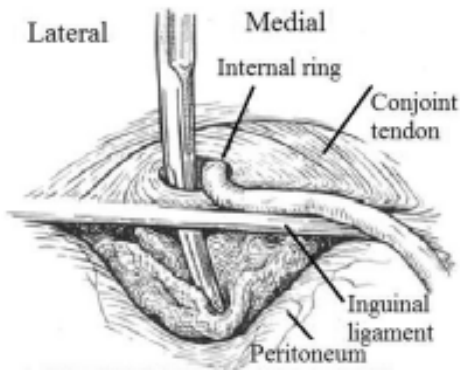


Figure 5.20. *Sponge dissection, posterior space. Sponge traction in the properitoneal space is more effective in creating the space than is the gloved finger of the surgeon*

The Prolene Hernia System bilayer patch device (Fig. 5.19 – 3) has a combined onlay graft (like a Lichtenstein repair) and underlay graft (like a Stoppa or Kugel patch); these are held together by a connector (like a plug). The external oblique aponeurosis is opened through a 5 cm groin incision, the spermatic cord is elevated, and an anterior space is created for placement of the onlay component of the device. If an indirect hernia sac is present, it is invaginated through the internal ring. If a posterior wall hernia is present (direct hernia), the defective tissue is circumscribed. In either case, the preperitoneal space (space of Bogros) is dissected free with a 4x4 sponge (Fig. 5.20).

The Prolene Hernia System is inserted through either of these defects. If a pantaloon hernia exists, the deep epigastric vessels are divided, and the two defects are converted to a single defect. The entire Prolene Hernia System is inserted through the posterior wall defect or internal ring. The underlay component is deployed so that the edge of the graft is at complete distraction from the connector (Fig. 5.21). The lateral portion of the underlay graft descends caudad to the Cooper ligament, thereby protecting the femoral canal. The onlay graft is extracted and placed against the posterior wall into the anterior space beneath the external and internal oblique muscles and laid against the transversus arch down to and over the pubic tubercle. A few sutures are placed in the onlay graft. At minimum, one is placed at the pubic tubercle, one at the mid portion of the transversus arch, and one at the mid portion of the inguinal ligament. The spermatic cord is accommodated with a central or lateral slit in the onlay component.

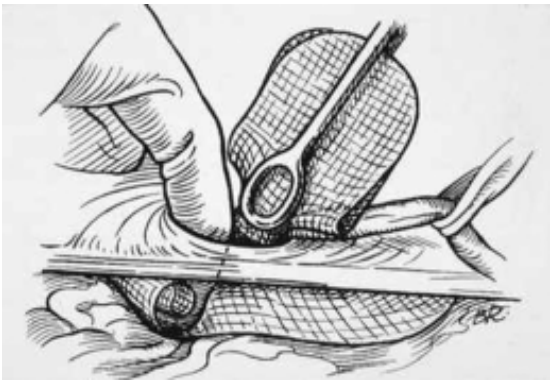


Figure 5.21. *The forefinger unfurls the underlay component of the Prolene Hernia System device*

Laparoscopic hernia repair
(Fig. 5.22):

- Early attempts resulted in exceptionally high reoccurrence rates.
- Current techniques include.

1. Transabdominal preperitoneal repair (TAPP).
2. Totally extraperitoneal approach (TEPA).
3. Intraperitoneal onlay mesh approach (IPOM).

1. Transabdominal Preperitoneal Repair (TAPP):

- Infraumbilical incision.
- Incise peritoneum from within abdomen.
- Direct visualization of hernia.
- Mesh placed over entire area, secured with staples or tacks.
- Peritoneum returned to line repair.

2. Totally Extraperitoneal Repair (TEP):

- Balloon entered into preperitoneum and inflated; creates space for scope.
- Mesh repair.
- Benefits.
- No risk of damage to peritoneal structures.
- No adhesive complications.

3. Intraperitoneal Onlay Mesh Approach (IPOM):

- Less popular.
- Diagnostic laproscopy.
- Mesh stapled over visible defect w/o dissection of peritoneum or establishment of defect borders.
- Quick, but high risk of adhesion of intestinal content to mesh.

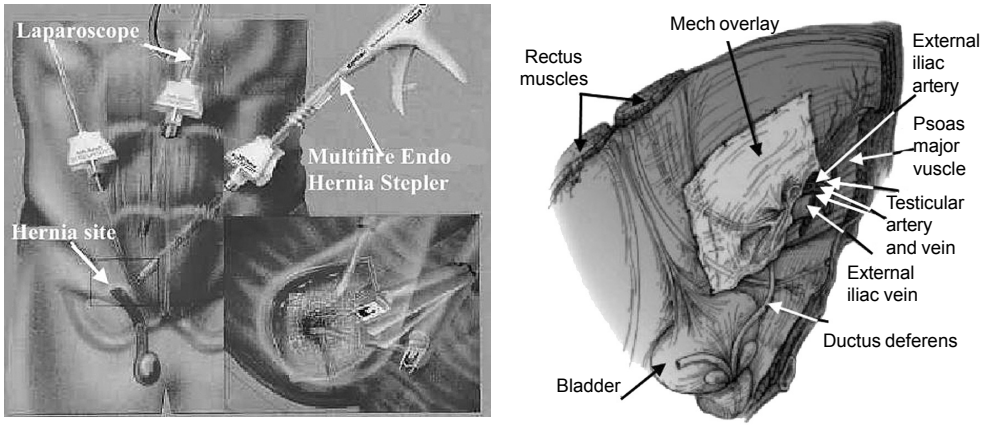


Figure 5.22. *Laparoscopic mesh repair*

Complications of herniorrhaphy: wound sepsis (especially due to faulty technique); hematoma; lymphocele (commoner after operations for femoral hernia); wound sinus (especially when foreign tissue is used for the repair); division of spermatic cord (especially in infantile hernia operation); testicular ischemia (especially after large or recurrent hernia repairs); testicular atrophy; hydrocele; nerve entrapment: pain, parasthesia or numbness; recurrence (especially after operations for large hernias in elderly males or sepsis); general: retention of urine; respiratory complications; thromboembolic complications.

Treatment of strangulated inguinal hernia. The treatment of strangulated hernia is by *emergency operation.* ("The danger is in the delay, not in the operation" – Astley Cooper.)

If dehydration and collapse are present, intravenous fluid replacement and gastric aspiration for 1–3 hours are invaluable. It is absolutely essential to make sure that the stomach is emptied just before commencing the anesthetic. The passing of a large-bore stomach tube is the best way of preventing vomiting, drowning, and cardiac arrest during the induction. The bladder must also be emptied, if necessary by a catheter. SuiTable broad-spectrum antibiotics are given i.v.

Operative complications. Approximately 7% of people undergoing surgical hernia repair will have complications.

- These are short-term and usually trea Table.
- The hernia that comes back after initial surgical repair can be repaired by the same or an alternate method.
- Complications include the following: recurrence (most common); urinary retention; wound infection; fluid build-up in scrotum (called hydrocele formation); scrotal hematoma (bruise); testicular damage on the affected side (rare).

5.6. FEMORAL HERNIA

The femoral canal is the path through which the femoral artery, vein, and nerve leave the abdominal cavity to enter the thigh. Although normally a tight space, sometimes it becomes large enough to allow abdominal contents (usually intestine) to protrude into the canal. A femoral hernia causes a bulge just below the inguinal crease in roughly the mid-thigh area. Usually occurring in women, femoral hernias are particularly at risk of becoming irreducible (not able to be pushed back into place)

and strangulated. Not all hernias that are irreducible are strangulated (have their blood supply cut off), but all hernias that are irreducible need to be evaluated by a health-care provider.

Femoral hernia is the third most common type of hernia (incisional hernia comes second). It accounts for about 20 per cent of hernias in women, and 5 per cent in men.

Surgical anatomy. The femoral canal occupies the most medial compartment of the femoral sheath, and it extends from the femoral ring above to the saphenous opening below. It is 1.25 cm long and 1.25 cm wide at its base, which is directed upwards. The femoral canal contains fat, lymphatic vessels, and the lymph node

The femoral ring is bounded:

- anteriorly by the inguinal ligament;
- posteriorly by Astley Cooper's (iliopectineal) ligament, the pubic bone, and the fascia over the pectineus muscle;
- medially by the concave knife-like edge of Gimbernat's (lacunar) ligament, which is also prolonged along the iliopectineal line as Astley Cooper's ligament;
- laterally by a thin septum separating it from the femoral vein.

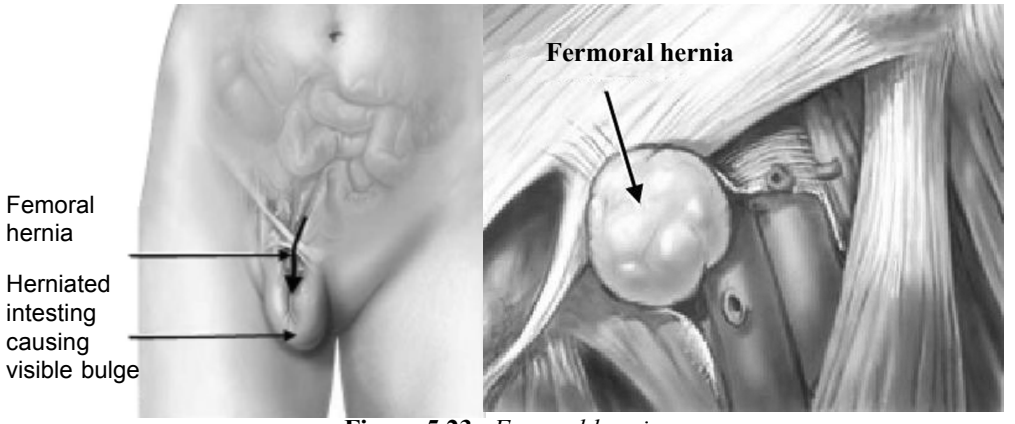


Figure 5.23. *Femoral hernia*

Pathology. A hernia passing down the femoral canal descends vertically as far as the saphenous opening. While it is confined to the inelastic walls of the femoral canal the hernia is necessarily narrow, but once it escapes through the saphenous opening into the loose areolar tissue of the groin, it expands, sometimes considerably. A fully distended femoral hernia assumes the shape of a retort, and its bulbous extremity may be above the inguinal ligament. By the time the contents have pursued so tortuous a path they are usually irreducible and apt to strangulate, a circumstance which is also favored by the rigidity of the surrounds of the femoral ring (Fig. 5.23).

Clinical features. Femoral hernia is rare before puberty. Between 20 and 40 years of age the prevalence rises, and continues to old age. The right side is affected twice as often as the left, and in 20 per cent of cases the condition is bilateral. The symptoms to which a femoral hernia gives rise are less pronounced than those of an inguinal hernia.

Differential diagnosis. A femoral hernia has to be distinguished from the following:

- *An inguinal hernia*. An inguinal hernia lies above and medial to the medial end of the inguinal ligament at its attachment to the pubic tubercle. Occasionally the fundus of a femoral hernia sac overlies the inguinal ligament.

● *A saphena varix.* A saphena varix is a saccular enlargement of the termination of the long saphenous vein and it is usually accompanied by other signs of varicose veins. The swelling disappears completely when the patient lies down, while a femoral hernia sac usually is still palpable.

● *An enlarged femoral lymph node.* If there are other enlarged lymph nodes in the region the diagnosis is tolerably simple, but when lymph node alone is affected the diagnosis may be impossible unless there is a cause, such as an infected wound or abrasion on the corresponding limb or on the perineum. Doubt should be removed by immediate surgery.

● *Lipoma.*

● *Hydrocele of a femoral hernial sac.*

Treatment. The constant risk of strangulation is sufficient reason for urging operation. A truss is contraindicated because of this risk.

OPERATIVE TREATMENT

The low operation (Lockwood). The sac is dissected out below the inguinal ligament via a groin-crease incision. It is essential to peel off all the anatomical layers which cover the sac. These are often thick and fatty. After dealing with the contents the neck of the sac is pulled down, ligated as high as possible and allowed to retract through the femoral canal. The canal is closed by suturing the inguinal ligament to the iliopectineal line using unabsorbable sutures.

The high (McEvedy) operation. A vertical incision is made over the femoral canal and continued upwards above the inguinal ligament. Through the lower part of the incision the sac is dissected out. The upper part of the incision exposes the inguinal ligament and the rectus sheath. The superficial inguinal ring is identified, and an incision 2.5 cm above the ring and parallel to the outer border of the rectus muscle is deepened until the extraperitoneal space is found. By gauze dissection in this space the hernial sac entering the femoral canal can be easily identified. Should the sac be empty and small, it can, be drawn upwards; if it is large, the fundus is opened below, and its contents, if any, dealt with appropriately before delivering the sac upwards from its canal. The sac is then freed from the extraperitoneal tissue and its neck is ligated. An excellent view of the iliopectineal ligament is obtained and the conjoined tendon is sutured to it with non-absorbable sutures.

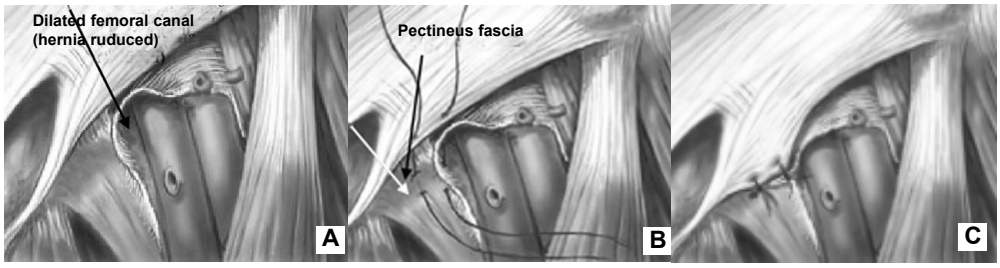


Figure 5.24. *Bassini repair*

Bassini Repair (Fig. 5.24).

Original approach was inferior to inguinal ligament with excision of sac and closure of inguinal ligament to pectineal fascia and Cooper's ligament from below. Hernia is approached via inguinal incision and Coopers Ligament repair performed.

Transverse incision 4 cm above pubic tubercle. Expose the Femoral ring preperitonealy. Coopers ligament sutured to ileopubic tract.

5.7. UMBILICAL HERNIA

These common hernias (10%–30%) are often noted at birth as a protrusion at the bellybutton (the umbilicus). This is caused when an opening in the abdominal wall, which normally closes before birth, doesn't close completely (Fig. 5.25). If small (less than half an inch), this type of hernia usually closes gradually by age 2. Larger hernias and those that do not close by themselves usually require surgery at age 2–4 years. Even if the area is closed at birth, umbilical hernias can appear later in life because this spot may remain a weaker place in the abdominal wall. Umbilical hernias can appear later in life or in women who are pregnant or who have given birth (due to the added stress on the area). This is a hernia through a weak umbilical scar. The ratio of males to females is 2:1. Most do not become obvious until the infant is several weeks old. The hernia is often symptomless, but increase in the size of the hernia on crying causes pain, which makes the infant cry the more. Small hernias are spherical; those that increase in size tend to assume a conical shape and are present apart from crying. Obstruction or strangulation below the age of three years is extremely uncommon.

Treatment. Conservative treatment by **masterly inactivity** is successful in about 93 per cent of cases. When the hernia is symptomless, reassurances of the parents is all that is necessary, for in a very high percentage of cases the hernia will be found to disappear spontaneously during the first few months of life.

Herniorrhaphy. In cases where masterly inactivity fails, operation is required, and it should be carried out, preferably, about the age of 2 years.

Mayo's Operation. A transverse elliptical incision is made around the umbilicus. The subcutaneous tissues are dissected off the rectus sheath to expose the neck of the sac. The neck is incised to expose the contents. Intestine is returned to the abdomen. Any adherent omentum is freed, and ligated by transfixion if it is bleeding. Excess adherent omentum can be removed with the sac if necessary. The sac is then removed and the peritoneum of the neck closed with catgut. The aponeurosis on both sides of the umbilical ring is incised transversely for 2.5 cm or more sufficiently to allow an overlap of 5 to 6 cm. Three to five mattress sutures of fine, unabsorbable material are then inserted into the aponeurosis. If the patient has a tendency to bronchitis, it is wise to prescribe antibiotic therapy and breathing exercises.

5.8. LUMBAR HERNIA

There are three types of lumbar hernia (Fig. 5.26). The first two are primary lumbar hernia which come out through the superior lumbar triangle and the inferior lumbar triangle. The 3rd one is an acquired lumbar hernia and is better called an incisional lumbar hernia.

Superior lumbar hernia. This is rarer than the inferior lumbar hernia. Superior lumbar hernia is protrusion of abdominal contents through the *superior lumbar triangle*, which is bounded above by the 12^{th} rib, medially by the sacrospinalis and laterally by the posterior border of the obliquus internus abdominis.

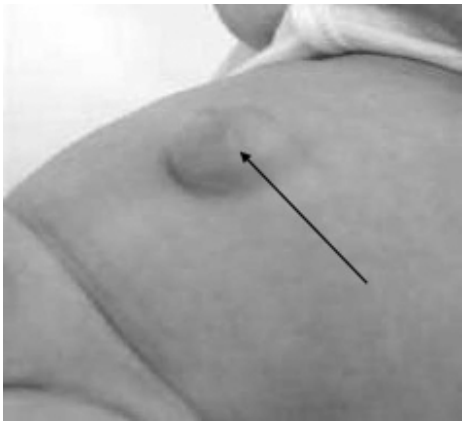


Figure 5.25. *Umbilical hernia*

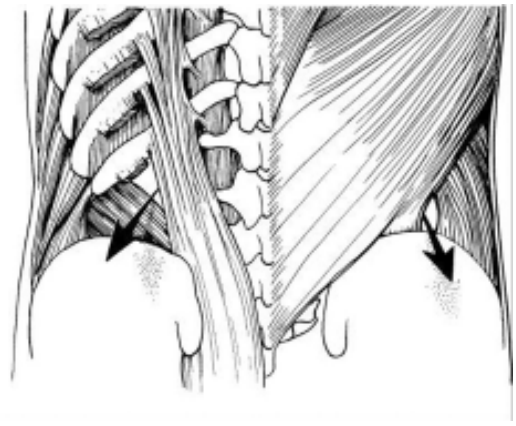


Figure 5.26. *Lumbar hernia*

Inferior lumbar hernia is more common than the superior one and the hernia protrudes through the *inferior lumbar triangle or lumbar triangle of Petit*. This triangle is bounded below by the crest of the ilium, medially by the anterior border of the latissimus dorsi and laterally by the posterior border of the obliquus externus abdominis.

Incisional lumbar hernia may occur in the lumbar region (a) following an operation or an infected kidney or drainage of lumbar abscess, in which the wound gets infected post-operatively or (b) following paralysis of the muscles of the lumbar region either due to injury of the nerve supplying this region or due to poliomyelitis. This is also known as *phantom hernia.*

Clinical features. Patient complains of a swelling in the typical lumbar region.

On examination there is soft swelling which corresponds to the superior or inferior lumbar triangle. The swelling is reducible. It may be reduced by itself when the patient lies down. Impulse on coughing is also present.

Differential diagnosis. When there is reducibility and impulse on coughing diagnosis may not be difficult, yet the following conditions should be kept in mind.

- *Lipoma.* It is a firm or soft swelling, which moves easily over the taut muscles. It is not reducible and there is no impulse on coughing. On moving the swelling one will see puckering of the skin.
- *Cold abscess* pointing to this region. Paravertebral cold abscess may become superficial through the lumbar triangle. This condition very much mimics lumbar hernia. It gives rise to a soft *cystic* swelling. Fluctuation test is positive. This swelling is not reducible. But cough impulse may be present. X-ray of the thoracic and lumbar spine is confirmatory.
- *Phantom hernia* due to local muscular paralysis – discussed above.

Treatment. Primary lumbar hernia is treated by herniotomy and repair of the gap i.e. hemiorrhaphy.

5.9. OBTURATOR HERNIA

This extremely rare abdominal hernia develops mostly in women. This hernia protrudes from the pelvic cavity through an opening in the pelvic bone (obturator foramen). This will not show any bulge but can act like a bowel obstruction and cause nausea and vomiting. Because of the lack of visible bulging, this hernia is very difficult to diagnose. It means hernia occurs through the obturator foramen traversed by the

obturator vessels and nerve. This obturator foramen is wider in females and that is why it is about 6 times commoner in women (Fig. 5.27).

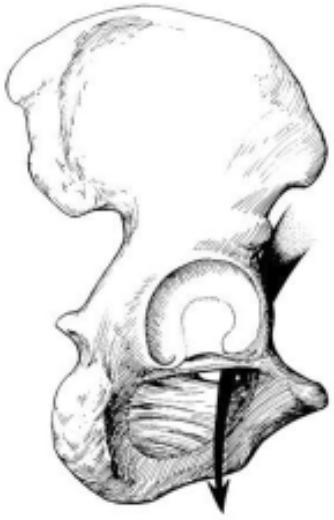


Figure 5.27. *Obturator hernia*

It is a condition of:

- Rare form of hernia.
- Protrusion of intra-abdominal contents through *obturator foramen.*
- Female/male ratio 6:1.
- The obturator foramen is formed by the ischial and pubic rami.
- Obturator vessels and nerve lie posterolateral to the hernia sac in the canal.
- Small bowel is the most likely intraabdominal organ to be found in an obturator hernia.

Clinical features. Most of the patients are old women above 60 years of age who have lost considerable fat.

The condition is difficult to diagnose, since the swelling is covered by the pectineus and no definite swelling can be seen even in the Scarpa's triangle. The hernia becomes only apparent when the limb is flexed, abducted and rotated outwards. The patient usually keeps the limb in the semiflexed position and movements increase pain.

Obturator hernia often gets strangulated as it comes out through an opening surrounded by osseoaponeurosis. In about half of the cases of strangulation, pain is referred along the obturator nerve to the knee joint of the corresponding side by its articulate branch.

Only rectal or vaginal examination can detect the tender swelling in the region of the obturator foramen.

Richter's hernia is quite common.

Treatment. Operation should always be performed as strangulation is very common.

Abdominal approach is usually preferred by lower paramedian incision as the condition is often discovered only after laparotomy which has been performed for intestinal obstruction. The obturator foramen is widened and the hernia is pulled in after the abdomen has been properly mopped to prevent contamination from the toxic fluid of the hernial sac. The obturator vessels are always vulnerable of being injured. If the hernia cannot be released from the abdomen or the diagnosis has been made before operation, the femoral approach may be employed.

Femoral approach. A vertical incision is made extending downwards from the inguinal ligament 2 cm medial to the femoral vessels. The adductor longus is retracted medially. The pectineus muscle is separated or divided to expose the obturator externus. The hernial sac is usually found lying on the surface of this muscle having emerged along its superior border. The sac and the contents are dealt with in the similar manner as strangulated hernia and hemiorrhaphy is performed by repairing the gap in the obturator foramen.

5.10. OTHER UNIMPORTENT RARE HERNIA

Epigastric hernia. Occurring between the navel and the lower part of the rib cage in the midline of the abdomen, epigastric hernias are composed usually of fatty tissue and rarely contain intestine. Formed in an area of relative weakness of the abdominal wall, these hernias are often painless and unable to be pushed back into the abdomen when first discovered.

Gluteal hernia. This hernia extrudes out through the greater sciatic foramen either above or below the piriformis muscle.

Sciatic hernia. This hernia occurs through the lesser sciatic foramen. In the differential diagnosis of gluteal and sciatic herniae one should remember a cold abscess, a lipoma, a gluteal aneurysm, fibrosarcoma beneath the gluteus maximus.

Spigelian hernia. It is a type of interparietal hernia occurring at the level of the arcuate line just lateral to the rectus muscle (Fig. 5.28).

The extraperitoneal fat along with the hernial sac lies just deep to the internal oblique muscle or may advance to reach the gap between the external and internal oblique muscles. Only a slight swelling can be detected.

Treatment. Operation is the treatment. Incision is made on the swelling just lateral to the rectus muscle. After incision of skin and subcutaneous tissue, external aponeurosis is split to expose the hemial sac. The sac is isolated, the contents reduced. The sac is ligated and excised. The transversus and oblique muscles are repaired.

Perineal hernia. In this case hernia occurs through the pelvic floor. It is extremely rare and occasionally seen in:

1. *Postoperative hernia* through the perineal scar following excision of rectum.

2. *Pudenda hernia* when the hernia occurs as a swelling of the labium majus.

3. *Ischiorectal hernia*, in which the hernia passes through the levator ani into the ischiorectal fossa through hiatus of Schwalbe.

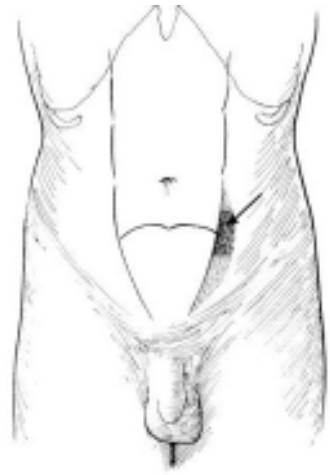


Figure 5.28. *Spigelian hernia (arrow)*

5.11. INCISIONAL HERNIA

Abdominal surgery causes a flaw in the abdominal wall. This flaw can create an area of weakness in which a hernia may develop. This occurs after 2%–10% of all abdominal surgeries, although some people are more at risk. Even after surgical repair, incisional hernias may return.

An incisional hernia (synonyms: ventral hernia or postoperative hernia) is one which occurs through an acquired scar in the abdominal wall caused by a previous surgical operation or an accidental trauma.

Scar tissue is inelastic and can be stretched forcibly if subjected to constant strain. When a ventral hernia occurs, it usually arises in the abdominal wall where a previous surgical incision was made. A hernia involves the weakening of a patient's abdominal muscles, resulting in a bulge or tear (Fig. 5.29).

Etiology

Defect of the patient:

- Obese individuals with lax muscles.
- Patients suffering from chronic cough, which may continue in the early postoperative period and will lead to incisional hernia.
- Undue abdominal distension in the early postoperative period.
- Malnutrition patients with severe anemia, hypoproteinaemia or Vitamin C deficiency may predispose to incisional hernia.

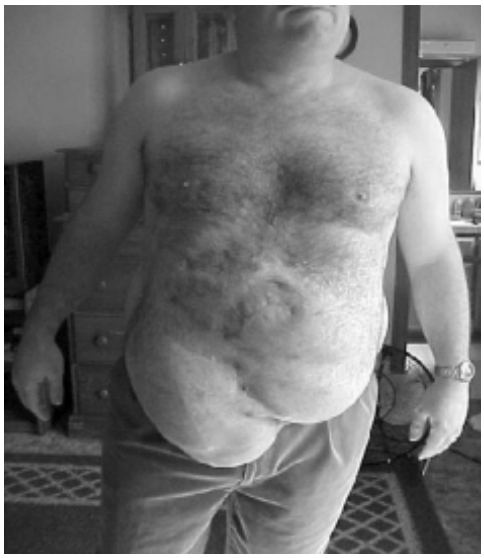


Figure 5.29. *Postoperative hernia*

Fault during operation:

- Injury to the motor nerves supplying the area. Certain incisions are vulnerable to cause nerve injury e.g. Kocher's subcostal incision for cholecystectomy often inflicts injury to the 8th, 9th and 10th intercostal nerves; Battle's pararectal incision for appendicectomy; McBurney's incision for appendicectomy may injure the subcostal or ilioinguinal nerve.
- Particular care was not taken during closure of the wound particularly the deeper layers.
- Haemostasis was not perfect or the tissues were manhandled, so that early postoperative infection was the result.
- Tube drainage through the laparotomy wound.
- Certain incisions are more liable to cause incisional hernia e.g. midline infraumbilical incision for caesarean section.

3. Postoperative causes.

- Infection. This seems to be the commonest cause.
- Postoperative cough and distension.
- Postoperative peritonitis due to more chance of wound infection.
- Too early removal of sutures.
- Steroid therapy in the postoperative period.
- Pathology.

Often the incisional hernia starts unnoticed and symptomless with partial disruption of the deeper layers of a laparotomy wound during very early postoperative period. So careful closure of the wound is extremely important to prevent incisional hernia.

Wound infection often causes disruption of sutures thus the muscles are separated by weak scar tissue. A portion of the muscles may also be destroyed by infections which are resolved afterwards by fibrosis. This also causes weak scar. Incisional hernia occurs through this weak scar.

SWR (Situation, wide, recurrence) classification of incisional hernia

1. Localization (S):

- median (M): M1 – supraumdilical hernia; M2 – periumbilical hernia; M3 – subumbilical hernia; M4 – pubic hernia.
- lateral (L): L1 – subcostal hernia; L2 – transversal hernia; L3 – inguinal hernia; L4 – lumbar hernia.

2. The Width of hernia port (W):

- W1 – till 5 cm; W2 – 5–10 cm; W3 – 10–15 cm; W4 – more than 15 cm

3. Relapses (R): R – no relapse; R1 – first relapse; R2 – second relapse etc.

Clinical features

A previous operation or a trauma is often noticed. Patient may give a history of wound infection. Incisional hernia may occur at any age, but more common in fatty elderly females.

The commonest symptoms are the swelling and the pain. Sometimes attacks of subacute intestinal obstruction may occur; leading to abdominal colic, vomiting, constipation and distension of the abdomen. Strangulation, though uncommon, is liable to occur at the neck of a small sac or in a locule of a large hernia.

On examination the old scar is seen with the swelling. The hernia may occur through a small portion of the scar often the lower end. Often a diffuse bulge may occur involving the whole length of the scar. Usually the swelling is reducible and an expansile cough impulse is present. The defect in the abdominal wall is often palpable.

It may so happen that the hernia is irreducible. Such cases become difficult to diagnose. These cases must be differentiated with (differential diagnosis):

- A deposit of tumor.
- An old abscess.
- A hematoma.
- A foreign body granuloma.

Treatment

A few preoperative measures should be carefully adopted to
lessen the chance of incisional hernia. These are:

1. If the patient is obese, weight should be reduced by dieting if an elective operation has to be performed.

2. If the patient has a tendency of chronic bronchitis, it should be treated first.

3. During operation one must be very careful in closure of the abdomen. Deeper layers must be sutured with due respect.

4. All precautions should be adopted to prevent immediate postoperative wound infection.

Operation

Ventral hernias can be repaired by the following techniques: an "open" method or a laparoscopic minimally invasive procedure. Like other hernias, a ventral hernia may become worse if left untreated. Moreover, ventral hernias can be dangerous, because abdominal structures, such as the intestines, can become stuck or twisted in the hernia, leading to a more complex and riskier operation. The only known treatment is to have the ventral hernia repaired through surgery. Any continuous or severe discomfort, redness, nausea, or vomiting resulting from the bulge associated with a hernia; are signs the hernia may be entrapped or strangulated.

The incision is deepened to the aponeurosis. The unhealthy skin is gradually dissected off the sac, which is nothing but a redundancy of peritoneum. The sac is not opened. If the sac is loculated and very adherent it is better to open the sac around its neck. The contents are freed. Adherent omentum may be ligated and removed along with the sac. Any adhesions involving the bowel should be separated as far as practicable before the hernial contents are returned to the abdomen. Repair of the hernia depends on the type of hernia and its size.

Mesh closures. These are becoming increasingly popular. The incision and the sac are dealt with similarly as done in the previous operations. The deficiency in the abdominal wall is easily made good without tension by laying and stitching a sheet a Mesh made of Dacron or polypropylene to the surrounding aponeurosis. Repair with synthetic mesh is only advised when the defect is very large and cannot be closed effectively by autogenous tissue. It cannot be used routinely as there is increased chance of infection. Collection of oozing fluid inside the wound acts as a good nidus of infection. The following points should be considered whenever a synthetic mesh is used:

1. Asepsis should be maintained at all costs.
2. Too much handling of the tissues should be minimized.
3. Haemostasis must be carefully maintained.
4. Even the pre-sterilized mesh should be handled as little as possible.
5. The mesh should be placed as deeply as possible. It should be used as an onlay on the sutured peritoneum. Under no circumstance should the intestine be allowed to come in contact with the mesh lest dense adhesions should form. The mesh should not be used just under the skin.
6. The mesh should be sutured all round with fine prolene sutures.
7. A suction drainage should always be used to aspirate the oozing fluid and, thus, to prevent infection.
8. Early ambulation is not encouraged, but movements of the legs and exercises of the legs are encouraged.
9. Patient must not resume work or strenuous exercise for 6 months.

A minimally invasive laparoscopic surgical technique includes small, dime-sized incisions that allow the use of a miniature camera, or videoscope, and specialized instruments to perform the procedure. The procedure will avoid a conventional incision. The main advantages of a laparoscopic ventral hernia procedure over open surgery include:

- Less time in hospital.
- Smaller incisions.
- Less recovery time – a traditional open procedure may take approximately three to four weeks of recovery compared to one to two weeks if performed laparoscopically.

Risks and complications. All surgical procedures have risks. The risk for serious complications depends on a patient's medical condition and age, as well as their surgeon's and anesthesiologist's experience. Patients should ask their doctor or surgeon about what to expect after surgery, as well as the risks that may occur with any surgery, including:

Risks of any surgery:

- Reactions to medications or anesthesia.
- Breathing problems.
- Bleeding.

- Infection.
- Injury to blood vessels.
- Injury to internal organs.
- Blood clots in the veins or lungs.
- Death (rare).
- If the surgeon uses a special mesh, or screen, to help repair the hernia, an added potential complication can be infection in the mesh. If this happens, the mesh may need to be removed or replaced.
- For patients undergoing surgical repair on a hernia that has reoccurred, there is a greater chance that the hernia will reoccur again.

Risks of conventional surgery. Additionally, conventional surgery for Ventral hernia repair has a greater potential for:

- Muscle injury.
- Recurrence of the hernia.

INTERNAL ABDOMINAL HERNIA

Higher in the abdomen, an (internal) "diaphragmatic hernia" results when part of the stomach or intestine protrudes into the chest cavity through a defect in the diaphragm. A hiatus hernia is a particular variant of this type, in which the normal passageway through which the esophagus meets the stomach (esophageal hiatus) serves as a functional "defect", allowing part of the stomach to (periodically) "herniate" into the chest. Hiatus hernias may be either "sliding", in which the gastroesophageal junction itself slides through the defect into the chest, or non-sliding (also known as paraesophageal), in which case the junction remains fixed while another portion of the stomach moves up through the defect. Non-sliding or paraesophageal hernias can be dangerous as they may allow the stomach to rotate and obstruct. Repair is usually advised. A congenital diaphragmatic hernia is a distinct problem, occurring in up to 1 in 2000 births, and requiring pediatric surgery. Intestinal organs may herniate through several parts of the diaphragm, posterolateral (in Bochdalek's triangle, resulting in Bochdale"s hernia), or anteromedial-retrosternal (in the cleft of Larrey/Morgagni's foramen, resulting in Morgagni-Larrey hernia, or Morgagni's hernia).

5.12. HIATAL HERNIA

A hiatal hernia is an anatomical abnormality in which part of the stomach protrudes through the diaphragm and up into the chest. Although hiatal hernias are present in approximately 15% of the population, they are associated with symptoms in only a minority of those afflicted.

Normally, the esophagus or food tube passes down through the chest, crosses the diaphragm, and enters the abdomen through a hole in the diaphragm called the esophageal hiatus. Just below the diaphragm, the esophagus joins the stomach. In individuals with hiatal hernias, the opening of the esophageal hiatus (hiatal opening) is larger than normal, and a portion of the upper stomach slips up or passes (herniates) through the hiatus and into the chest. Although hiatal hernias are occasionally seen in infants where they probably have been present from birth, most hiatal hernias in adults are believed to have developed over many years.

Allocate:

- Sliding hiatal hernia (90% of cases).
- Rolling hiatal hernia (paraesophageal).

Sliding hiatal hernia, the most common type of hernia, are those in which the junction of the esophagus and stomach, referred to as the gastro-esophageal junction, and part of the stomach protrude into the chest (Fig. 5.30). The junction may reside permanently in the chest, but often it juts into the chest only during a swallow. This occurs because with each swallow the muscle of the esophagus contracts causing the esophagus to shorten and to pull up the stomach. When the swallow is finished, the herniated part of the stomach falls back into the abdomen.

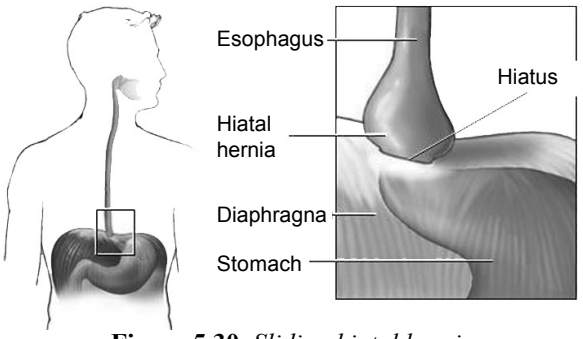


Figure 5.30. *Sliding hiatal hernia*

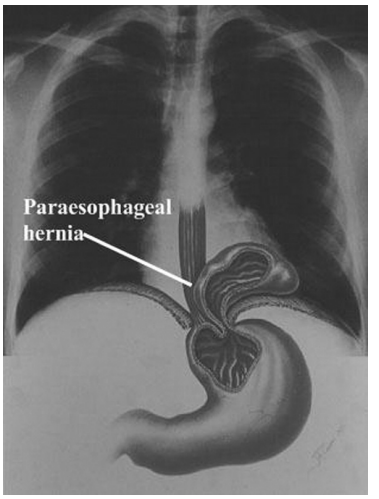


Figure 5.31. *Rolling hiatal hernia*

Paraesophageal hiatal hernia.

Paraesophageal hernias are hernias in which the gastro-esophageal junction stays where it belongs (attached at the level of the diaphragm), but part of the stomach passes or bulges into the chest beside the esophagus. The paraesophageal hernias themselves remain in the chest at all times and are not affected by swallows (Fig. 5.31). A paraesophageal hiatal hernia that is large, particularly if it compresses the adjacent esophagus, may impede the passage of food into the stomach and cause food to stick in the esophagus after it is swallowed. Ulcers also may form in the herniated stomach due to the trauma caused by food that is stuck or acid from the stomach. Fortunately, large paraesophageal hernias are uncommon.

Clinical features of hernias:

- Sliding hiatal hernia: heartburn; regurgitation; chest pain; dysphagia; belching.
- Paraesophageal hernia: feeling of fullness and breathlessness after eating; feeling of suffocation; cheat pain that mimics angina; symptoms worse in recumbent position.

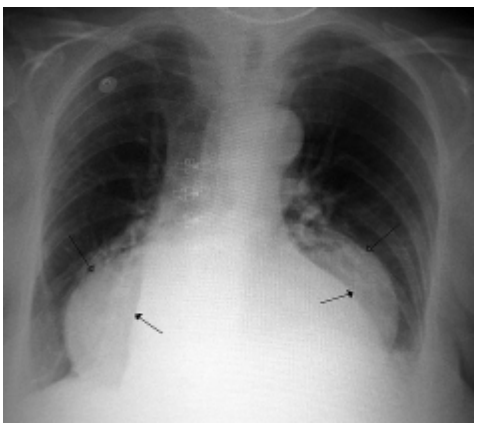


Figure 5.32. *A large hiatus hernia on X-ray marked by open arrows in contrast to the heart borders marked by closed arrows*

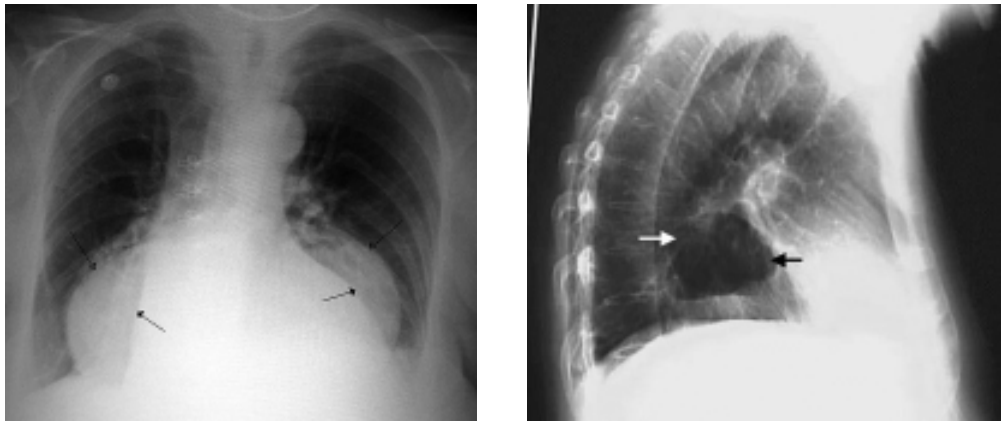


Figure 5.33. *X-ray: paraesophageal hernia (arrows)*

Diagnostic tools:

- X-ray and barium swallow (Fig. 5.32, 5.33).
- Upper endoscopy.
- Esophageal manometry.
- 24-hour ambulatory esophageal pH.
- Gastric emptying study.

Risk Factors:

- Increased intra-abdominal pressure: obesity; pregnancy; bending; coughing; weight lifting.
- Age.

Medical treatment.

- Goals: aimed at relieving symptoms and preventing complications.
- Bleeding: reduce regurgitation of stomach contents into esophagus.
- Medications: includes antacids and histamine receptor antagonists (pepcid and reglan); neutralizes stomach acidity; decrease acid production H^2 receptor blockers (ranitidine 150 mg; famotidine 20 mg and others); prokinetic agent (metoclopramide 10 mg; cimsapride 10 to 20 mg); proton pump inhibitors (omeprazole 20 mg to 40 mg and others).

Principles of operative repair: hernia reduction; hernia sac excision; crural repair; gastric fixation; fundoplication controversial.

Operation (open vs. laparoscopy) – (Fig. 5.34).

Postoperative care:

- Reduce risk for bleeding, infection and organ injury.
- Respiratory care.
- Management of nasogastric tube.
- Nutritional care.

Complications of surgery:

- Injury to an abdominal organ or to the bowel, stomach, or esophagus.
- Bleeding.
- Failure to completely relieve reflux symptoms.
- Difficulty swallowing.
- Inability to vomit.
- Diarrhea.
- Distended abdomen.

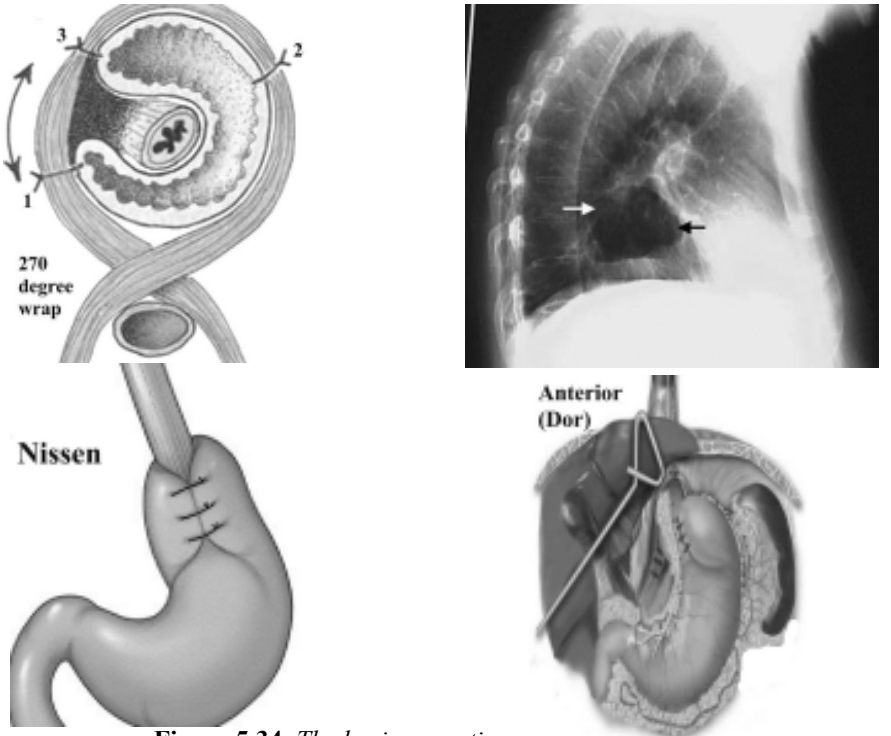


Figure 5.34. *The basic operations of hiatal hernias*

- Vagus nerve injury.
- Temporary dysphagia.
- Gas bloat syndrome (avoid carbonated beverages).
- Atelectasis, pneumonia.

5.13. TRAUMATIC DIAPHRAGMATIC HERNIA

Diaphragmatic injuries are relatively rare and result from either blunt or penetrating trauma. Regardless of mechanism, seemingly innocent penetrating injuries may be long forgotten by the patient and are the most commonly missed diaphragmatic injury. Diagnosis is often missed and high index of suspicion is vital. The clinical signs associated with a diaphragmatic hernia (Fig. 5.35) can range from no outward signs to immediately life-threatening respiratory compromise.

The diaphragm is integral to normal ventilation, and injuries can result in significant ventilatory compromise. A history of respiratory difficulty and related pulmonary symptoms may indicate diaphragmatic disruption. Penetrating injuries to the chest or abdomen also may injure the diaphragm. This specific injury is seen commonly where penetrating trauma is prevalent. This occurs most often from gunshot wounds but can result from knife wounds. Typically, the wounds are small, although occasionally a shotgun blast or impalement causes a large defect. Delay in detecting and repairing diaphragmatic injury increases both morbidity and mortality.

Diaphragmatic injuries are relatively rare and result from either blunt or penetrating trauma. These injuries were described first by Sennertus in 1541. Riolfi performed the first successful repair in 1886. Not until 1951, when Carter et al published the first case series, was this injury well understood and delineated.

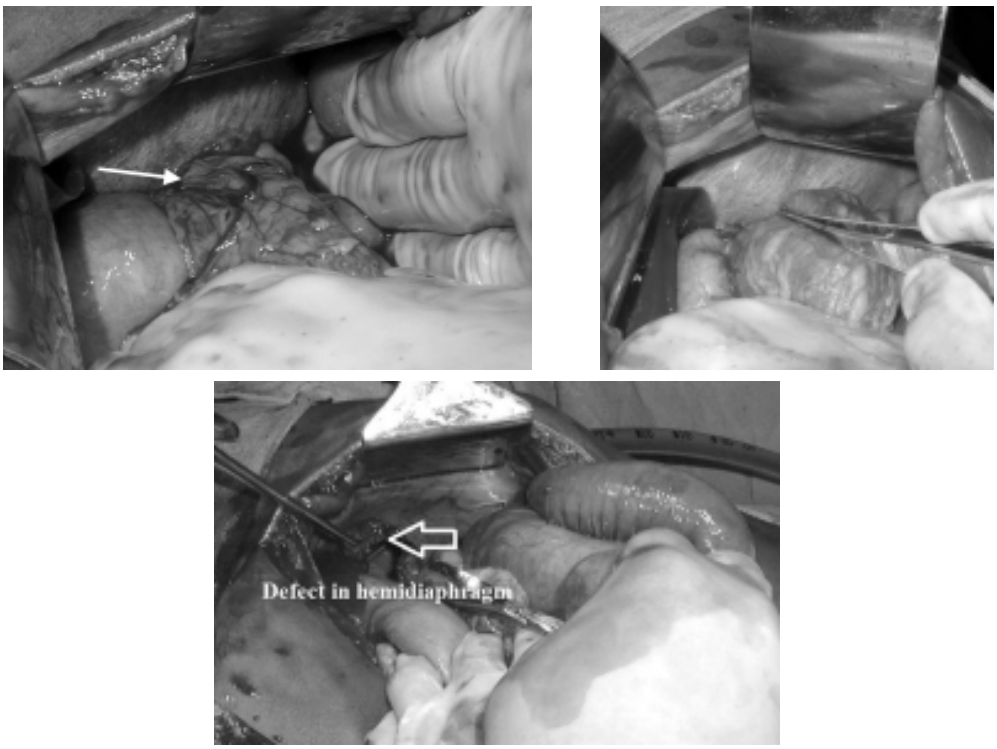


Figure 5.35. *Diaphragmatic hernia (arrow)*

Patients with delayed diaphragmatic herniation frequently present months to years after the initial injury with manifestations of visceral incarceration, obstruction, ischemia from strangulation, or perforation.

Diagnosis

The first rule leading to diagnosis of diaphragmatic hernia is to suspect the injury. Clinical presentation varies depending on the mechanism of injury (i.e., blunt vs. penetrating) and the presence of associated injuries. Penetrating trauma is the most common cause of diaphragmatic injury. The left hemidiaphragm is involved more frequently (>80%) than right. In our case too, left hemi-diaphragm was the site of injury. Visceral herniation occurs in up to 95% of left sided lacerations and stomach is the most frequent abdominal viscus to herniated.

The symptoms frequently are masked by associated injuries. A history of respiratory difficulty and related pulmonary symptoms may indicate diaphragmatic disruption. Isolated diaphragmatic injury in asymptomatic patients cannot be reliably delineated by either serial physical examination or peritoneal lavage. Clinical signs of diaphragmatic hernia vary from none to severe respiratory compromise and shock. Dyspnea is the most common clinical sign, and relates multifactorily to the presence of shock, chest wall dysfunction, the presence of air, fluid or viscera in the pleural space, decreased pulmonary compliance, edema, and cardiovascular dysfunction. The respiratory pattern is restrictive. Cardiac arrhythmias are present in 12% of small animals with diaphragmatic hernia. Other common clinical signs include muffled heart and lung sounds (may be asymmetrical), thoracic borborygmi, a strong apex beat ausculted on one side of the chest due to shifting of the apex to one side, an asymmetrical decreased caudoventral resonance when the thoracic cavity is percussed, and rarely a "tucked up" abdomen.

Chest x-ray may reveal the injury, if the abdominal contents have herniated into the chest. There may be a thickening or fuzziness of the diaphragmatic outline, an elevated hemi diaphragm, shift of mediastinum to opposite side, abnormal course of nasogastric tube or be completely normal. Haemo-pneumothoraces are a common associated finding. Overall plain chest X-ray has 50% accuracy (Fig. 5.36).

Ultrasonographic evaluation is useful to identify abdominal viscera on the thoracic side of the diaphragm especially in the presence of pleural fluid because it enhances sonographic evaluation. Ultrasound may identify abdominal organs, differentiate organs such as the spleen or liver from pleural fluid, and will sometimes identify the defect in the diaphragm. Chronic strangulation of a liver lobe results in a modified serosanguinous transsudate approximately 30% of the time. As a result people with chronic diaphragmatic hernias are often admitted with a restrictive breathing pattern.

Standard CT is limited in the diagnosis of diaphragmatic rupture due to the transaxial nature of the images-sensitivity 61–73% (Fig. 5.37).

Surgery. Surgery is the mainstay in the treatment of diaphragmatic rupture. Operative repair is technically more difficult if the surgery is delayed (Fig. 5.38).

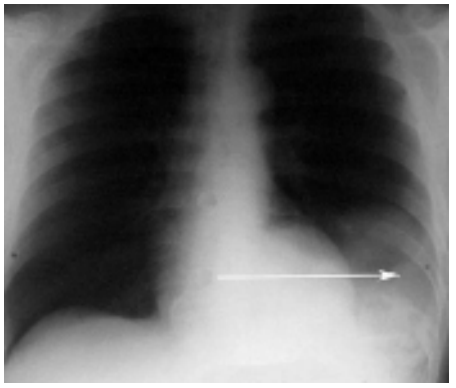


Figure 5.36. *An X-ray showing the spleen in the left lower portion of the chest cavity (arrow) after a diaphragmatic tear*

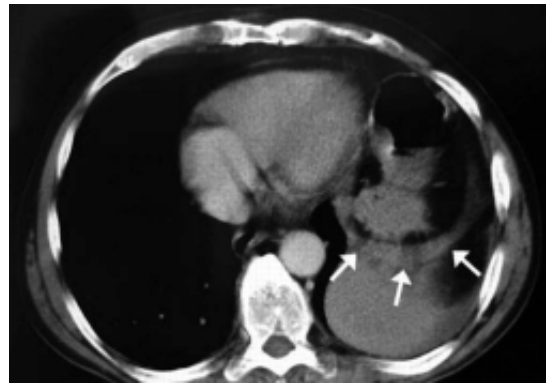


Figure 5.37. *CT image showing a replaced stomach through a defect in the left hemidiaphragm (arrows)*

The difference is based on the degree of adhesion present in the thoracic cavity and the state of the herniated organs. After reduction of the abdominal contents diaphragm can usually be repaired simply with monofilament non-absorbable suture, placed as locked stitches or horizontal mattress sutures. It is important to adequately wash out the thoracic cavity prior to closure, to remove any clot or contamination. In cases where the diaphragmatic hernia has been present for a long time simple closure may be difficult or impossible, and non-absorbable mesh may be required. In our case we did not come across any adhesions making repair difficult.

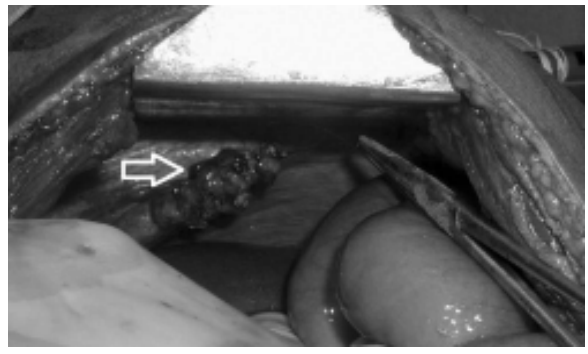


Figure 5.38. *Repair of diaphragmatic rupture (arrow)*

Chapter 6

ACUTE COMPLICATIONS OF PEPTIC ULCERS

Ulcer is a defect of gastric or duodenal mucosa which interferes with the continuity of lamina muscularis mucosae, submucosal layers or penetrates across the whole gastric or duodenal wall; occurence of ulcer is conditioned by the presence of acidic gastric content; frequent disease, men are afected 3–4 times more than women.

A peptic ulcer is an ulcer (defined as mucosal erosions equal to or greater than 0.5 cm) of an area of the gastrointestinal tract that is usually acidic and thus extremely painful. As many as 80% of ulcers are associated with *Helicobacter pylori*, a spiral-shaped bacterium that lives in the acidic environment of the stomach, however only 20% of those cases go to a doctor. Ulcers can also be caused or worsened by drugs such as aspirin and other (non steroidal anti-inflammatory drugs) NSAIDs, result of stress or intoxication condition. That's why the pathogenesis is multi factorial.

6.1. ANATOMY OF THE STOMACH

In anatomy, the stomach is a bean-shaped hollow muscular organ of the gastrointestinal tract involved in the second phase of digestion, following mastication.

The stomach lies between the esophagus and the duodenum (the first part of the small intestine). It is on the left side of the abdominal cavity. The top of the stomach lies against the diaphragm. Lying beneath the stomach is the pancreas, and the greater omentum which hangs from the *greater curvature.*

In humans, the stomach has a volume of about 50 ml when empty. After a meal, it generally expands to hold about 1 liter of food, but it can actually expand to hold as much as 4 liters. When drinking milk it can expand to just under 6 pints, or 3.4 liter.

Sections. The stomach is divided into four sections, each of which has different cells and functions. The sections are (Fig. 6.1, 6.2):

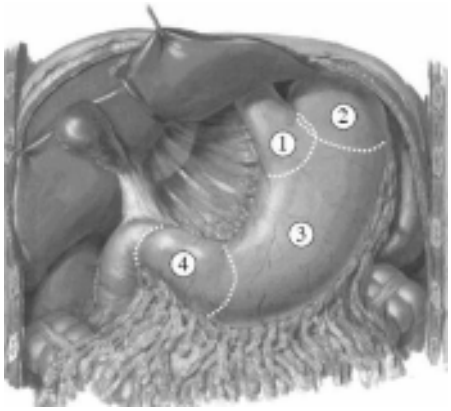


Figure 6.1.
Anatomic divisions of the stomach.

1. Cardiac region, 2. Fundus, 3. Corpus (Body), 4. Pyloric Antrum

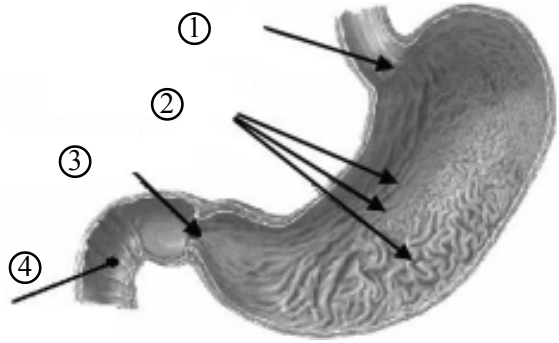


Figure 6.2.
Features of the stomach.

1. Lower esophageal sphincter, 2. Rugea, 3. Pyloric sphincter, 4. Duodenum

6.2. FUNCTIONS

1. The stomach is a highly acidic environment due to gastric acid production and secretion which produces a luminal pH range usually between 1 and 4 depending on the species, food intake, time of the day, drug use, and other factors.

2. Combined with digestive enzymes, such an environment is able to break down large molecules (such as from food) to smaller ones so that they can eventually be absorbed from the small intestine. The human stomach can produce and secrete about 2 to 3 liters of gastric acid per day with basal secretion levels being typically highest in the evening.

3. Pepsinogen is secreted by chief cells and turns into pepsin under low pH conditions and is a necessity in protein digestion.

4. Other functions include absorbing some ions, water, and some lipid soluble compounds such as alcohol, aspirin, and caffeine.

5. Another function of the stomach is simply a food storage cavity.

6. The stomach is famous for its secretion of acid, but acid is only one of four major secretory products of the gastric epithelium, all of which are important either to the digestive process or to control of gastric function.

7. Mucus: the most abundant epithelial cells are mucous cells, which cover the entire luminal surface and extend down into the glands as "mucous neck cells." These cells secrete a bicarbonate-rich mucus that coats and lubricates the gastric surface, and serves an important role in protecting the epithelium from acid and other chemical insults.

8. Acid: hydrochloric acid is secreted from parietal cells into the lumen where it establishes an extremely acidic environment. This acid is important for activation of pepsinogen and inactivation of ingested microorganisms such as bacteria.

9. Proteases: pepsinogen is secreted into gastric juice from both mucous cells and chief cells. Once secreted, pepsinogen is activated by stomach acid into the active protease pepsin, which is largely responsible for the stomach's ability to initiate digestion of proteins.

10. Hormones: the principal hormone secreted from the gastric epithelium is gastrin, a peptide that is important in control of acid secretion and gastric motility.

A number of other enzymes are secreted by gastric epithelial cells, including a lipase and gelatinase. One secretory product of considerable importance in man is intrinsic factor, secreted by parietal cell that is necessary for intestinal absorption of vitamin B_{12}.

Enzymes Secreted by the Mucosa. The mucosa is densely packed with gastric glands, which contain cells that produce digestive enzymes, hydrochloric acid, and mucus.

1. Mucous cells: secrete an alkaline mucus that protects the epithelium against sheer stress and acid.

2. Parietal cells: secrete hydrochloric acid.

3. Chief cells: secrete pepsin, a proteolytic enzyme.

4. G cells: secrete the hormone gastrin.

Whole complications of peptic ulcers

1. Bleeding (it occurs when anything erodes one of the blood vessels) – chronic (minor, cause anaemia); acute (major, form affected vessel).

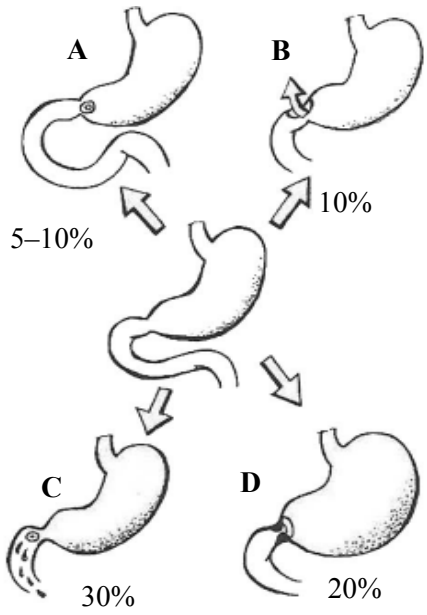


Figure 6.3.

A – penetration; B – perforation; C – bleeding; D – stenosis

2. Perforation (erosion of the gastrointestinal wall by ulcer leads to spillage of stomach or intestinal content into the abdominal cavity) – mostly bulbus duodeni, anterior gastric wall; acute violent pain; bleeding can be present.

3. Penetration – of the ulcer deeply through whole wall into neighboring organ (pancreas, liver).

4. Stenosis (scarring and swelling due to ulcers cause narrowing in the duodenum and gastric outlet obstruction) – narrowing of the lumen caused by scar, oedema or inflammatory infiltration after healing of the ulcer; arises only at pyloric localization; vomiting of huge volume of gastric content (Fig. 6.3).

ACUTE COMPLICATIONS OF PEPTIC ULCERS

6.3. BLEEDING ULCER

Peptic ulcer is the most common cause of acute hemorrhage in the upper gastrointestinal tract, accounting for about 50 percent of cases.

Bleeding from ulcers ceases spontaneously in at least 80 percent of patients, most of whom have an uneventful recovery without a specific intervention. However, a subgroup of patients with bleeding ulcers does not fare as well, accounting for an overall mortality rate that has remained around 6 to 7 percent for the past 30 years. There are at least two possible explanations for this unchanging mortality rate. First, age and the prevalence of concurrent illness, both important predictors of death, continue to rise among patients presenting with upper gastrointestinal bleeding. Patients with bleeding usually die not from exsanguination but from decompensation due to other diseases. Second, until very recently, effective nonsurgical methods for the control of bleeding from ulcers were not available.

Histologic Features

Histologic examination of a surgically resected gastric ulcer associated with bleeding reveals an artery eroded by the crater of the ulcer. In most cases the diameter of the bleeding artery is small (mean, 0.7 mm; range, 0.1 to 1.8). A larger arterial size is probably associated with increased morbidity and mortality, as well as a decreased likelihood of success with endoscopic therapy. A retrospective review noted that the arterial diameter ranged from 1.5 to 3.4 mm in approximately a quarter of patients with fatal bleeding ulcers.

The mechanism of erosion of an ulcer in presented in Fig. 6.4. The most common cause of death in patients is bleeding in those who have major medical problems or are older than 65 years. Because the duodenum has an abundant blood supply and the

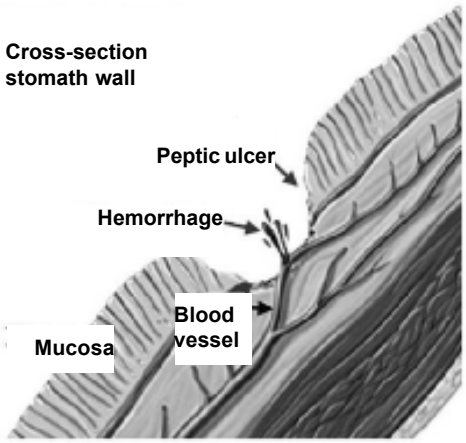


Figure 6.4. *Cross – section of stomach wall*

gastroduodenal artery lies directly posterior to the duodenal bulb, gastrointestinal (GI) bleeding from a duodenal ulcer is fairly common. Most cases of massive upper GI hemorrhage are in fact secondary to a bleeding duodenal ulcer following penetration of that ulcer into the gastroduodenal artery. Fortunately, most of the ulcers are superficial or are located on portions of the duodenum that are not adjacent to the gastroduodenal artery or its branches. Consequently, most duodenal ulcers present with only minor bleeding episodes that are detected by the presence of melanotic stool. Bleeding gastric and duodenal ulcers account for about 25% of all upper GI bleeding patients who present to the hospital.

Predisposing factors. High levels of acid secretion do not appear to account for the development of bleeding in patients with peptic ulcers. Basal and stimulated acid output, as well as the sensitivity of parietal cells to pentagastrin, is similar in patients with bleeding duodenal ulcers and in those with nonbleeding ulcers. The prevalence of Helicobacter pylori in patients with bleeding ulcers may be 15 to 20 percent lower than in patients with nonbleeding ulcers. Conversely, the use of nonsteroidal antiinflammatory drugs (NSAIDs) is reported to be an important risk factor for bleeding ulcers. A number of large case-control and cohort studies suggest that the risk of upper gastrointestinal bleeding is higher for patients who use NSAIDs than for those who do not. A recent meta-analysis found that an age over 60 years, a prior "gastrointestinal event" and use of NSAIDs for less than one month were associated with higher risks of complications.

Ingestion of NSAIDs may cause both gastric and duodenal ulcers. Although gastric ulcers are more common than duodenal ulcers, in terms of the overall incidence of ulcers, the increased rates of complications associated with use of NSAIDs are similar for the two forms of ulcer. Furthermore, complications may occur soon after the initiation of NSAID therapy and appear to be more common during the first month of therapy. Thus, it appears that NSAIDs not only induce ulcers but also increase the chance of complications such as bleeding in patients whose underlying ulcer disease is not primarily due to NSAIDs.

Although the risk of an ulcer due to use of aspirin or non-aspirin NSAIDs is dependent on the dose, the effect of the dose on the development of bleeding from an ulcer has not been studied extensively. The risk of bleeding has been carefully assessed in a large randomized controlled trial of aspirin therapy for prophylaxis against transient ischemic attacks. Patients received 300 or 1200 mg of aspirin a day or placebo. Patients receiving 300 mg of aspirin a day had a significant increase in upper gastrointestinal bleeding, as compared with those receiving placebo (relative risk, 7.7; 95 percent confidence interval, 1.7 to 33.8), whereas for the patients receiving 1200 mg of aspirin a day, the relative risk of bleeding was twice that for the patients receiving 300 mg a day. Recent evidence indicates that 10 mg of aspirin a day virtually obliterates the

synthesis of platelet thromboxane B_2 but does not significantly decrease the output of gastric prostaglandin E_2 or increase gastric injury. Careful clinical evaluation in large numbers of patients will be required to confirm the safety of treatment with very low doses of aspirin, with respect to bleeding ulcers.

Corticosteroids alone have not been demonstrated to increase the risk of ulcer development or bleeding. However, steroids are reported to double the NSAID-associated risk of serious gastrointestinal complications, and the concomitant use of steroids and NSAIDs may be associated with a 10-fold increase in the risk of upper gastrointestinal hemorrhage.

Although anticoagulation therapy would seem likely to increase the risk of bleeding in patients with peptic ulcers, placebo-controlled trials with warfarin (which generally exclude patients with known ulcer disease) have not documented a significant increase in bleeding ulcers.

6.3.1. CLINICAL FEATURES

1. Complains:

- General weakness.
- Dizziness.
- Tachycardia.
- Nausea.
- Vomiting (**hematemesis** – bright red or dark blood indicates that the source is proximal to the ligament of Treitz. It is more common from bleeding that originates in the stomach or duodenum. Coffee ground vomitus is due to vomiting of blood that has been in the stomach long enough for gastric acid to convert hemoglobin to methemoglobin).
- **Melena** – is the passage of black or tarry stools.
- **Hematochezia** – bleeding from the colon, rectum or anus can produce bright red rectal blood. However, it can also indicate a brisk bleeding in the upper intestine where the blood may be passed unchanged in the stool.

2. Objective sings of disease:

- Skin pallor.
- Tachycardia.
- Moist tongue.
- Palpation – no tension, pain or symptoms of peritoneal irritation.
- Rectal examination – presence of liquid or formed black faeces on the glove.

3. Laboratory investigations:

- Biochemical parameters of blood, blood and urine analysis.
- Coagulogramm.
- Ht (hematocrit, parity of volumes of uniform elements and plasmas of blood).
- Blood group, rhesus-factor (Rh-factor).

The classes of acute hemorrhage are presented in the table 6.2.

In our clinic we use the classification of acute hemorrhage of A.A. Shalimov et al. (1987):

Table 6.2. – Classes of acute hemorrhage

	Class I	**Class II**	**Class III**	**Class IV**
Blood loss (ml)	< 750 0–15%	750–1500 15–30%	1500–2000 30–40%	>2000 >40%
Blood loss (% BV)	<15%	15–30%	30–40%	>40%
Pulse rate (per minute)	< 100	> 100	>120	>140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure	Normal	Normal	Decreased	Negligible
Respiratory Rate (per minute)	14–20	20–30	30–40	>35
Urine Output (ml/hr)	>30	20–30	5–15	Minimal
CNS/Mental Status	Sl anxious	Anxious	Anxious, confused	Confused, lethargic

- **The first degree** – mild – is observed with blood loss to 20% of the volume of circulating blood (up to 1000 ml in a patient with a body weight of 70 kg). General condition is satisfactory or moderate severity. Skin pale, sweating appears; Pulse 90–100 per 1 min, blood pressure 100–90 / 60 mmHg. Consciousness and breath are not interruption. Expressed disorders blood circulation is not present.
- **The second degree** – of moderate severity – is observed with a loss of 20% to 30% of the volume of circulating blood (from 1000 to 1500 ml in a patient with a body weight of 70 kg). General condition of moderate severity. The patient is braked. The skin is pale, a sticky sweat appears. Pulse 120–130 per 1 min, weak filling, arterial pressure 90–80 / 50 mmHg. Breathing is quickened. Oliguria.
- **The third degree** – severe – is observed with a loss of more than 30% of the volume of circulating blood (from 1500 to 2500 ml in a patient with a body weight of 70 kg). The general condition is severe or very severe. The motor reactions are deferred. The patient responds in a whisper to questions, often loses consciousness. Skin and mucous are pale-cyanotic or spotty. The pulse is threadlike, 130–140 per 1 min, cannot be palpated. Arterial pressure from 0–60 to 50 mm Hg. Breath is superficial. Anuria.

Endoscopy. The endoscopist can visualize superficial mucosal lesions such as esophagitis, gastritis, superficial duodenal and gastric erosions, as well as Mallory-Weiss tears, varices, and ulcers. Endoscopy can usually determine which lesion is responsible for the hemorrhage (Fig. 6.5), an important consideration in patients who have multiple lesions (i.e., patients with cirrhosis and portal hypertension). The endoscopist can affect therapy in some patients who are bleeding, for example, sclerotherapy of varices and laser coagulation.

Prognostic features at endoscopy

Although clinical characteristics are important in predicting the outcome of a bleeding ulcer and in determining which patients should undergo urgent esophagogastroduodenoscopy (hereinafter referred to as endoscopy), the endoscopic appearance of an ulcer may provide the most helpful prognostic information. The ulcer may have a clean base or have one of several stigmata of hemorrhage: a flat pigmented spot (red, purple, brown, or black), an adherent clot, a visible vessel (a smooth-surfaced protuberance or plug in the base of the ulcer), or active bleeding (either oozing or spurting). The characteristic stigmata of hemorrhage are shown in Fig. 6.6.

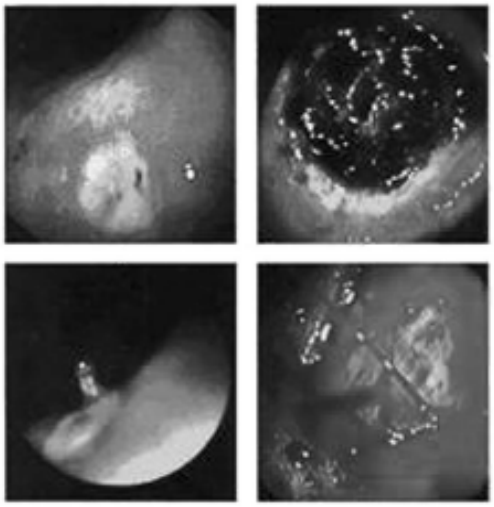


Figure 6.6. *Photographs of peptic ulcers with stigmata of hemorrhage: panel "A" shows a flat, pigmented spot; panel "B" an adherent clot; panel "C" a visible vessel; and panel "D" blood spurting*

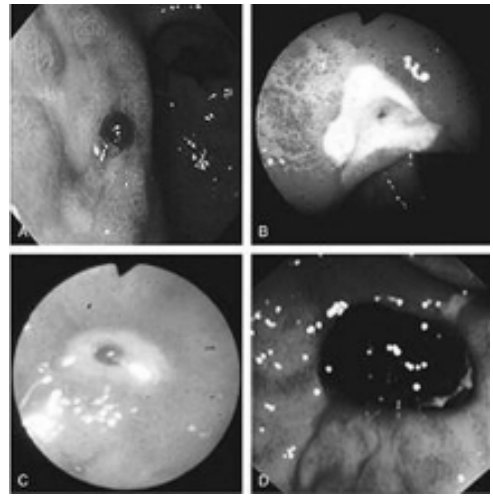


Figure 6.5. *Endoscopic views of ulcers with stigmata of recent hemorrhage: A – duodenal ulcer with a visible vessel; B – gastric ulcer with a red spot in the center of the crater; C – duodenal ulcer with a red spot in the center of the crater; D – purplish clot adherent to a gastric ulcer*

Forrest's classification of bleeding from an ulcer is presented in the table 6.1.

The size of an ulcer is also a prognostic indicator. Patients with ulcers larger than 1 or 2 cm in diameter have increased rates of rebleeding and death, even after endoscopic hemostatic therapy. Large ulcers are more frequently found to have stigmata of recent hemorrhage than are small ulcers.

Clinical investigators are exploring other methods to refine the prognostic usefulness of endoscopic features in patients with bleeding ulcers. The color of

Table 6.1 – Forrest classification

CLASSIFICATION	REBLEEDING RISK
Grade 1: active, pulsatile bleeding	High (~50–80%)
Grade Ib: active, nonpulsatile bleeding	Low
Grade IIa: nonbleeding, visible vessel	High (~50–80%)
Grade IIb: adherent clot	Low
Grade III: no signs of recent bleeding (hematemesis/melena in past 48 hours)	Low

visible vessels has been suggested as potentially helpful in predicting rebleeding, but the available data do not support this contention. One small prospective study, however, has reported that clear or translucent vessels presage a significantly higher likelihood of rebleeding than do opaque vessels.

6.3.2. HEMORRAGIC CHOCK

- Decreased perfusion to splanchnic organs precedes lower blood pressure.
- Lactic acid production.
- Base deficit.
- Normal base deficit is greater than – 2 mEq/L.
- After 1/3 of blood volume lost hypotension occurs.
- Acidemia occurs about then as patient cannot create enough respiratory compensation for the lactic acid.
- Organ injury in resuscitation.
- Release of activated neutrophils & inflammatory cytokines.
- Distorted balance of vasodilatation vs. vasoconstriction.
- May lead to acute respiratory distress syndrome (ARDS), acute tubular necrosis, & centrilobular ischemic liver damage.

Pathophysiology of hypovolemia:

- Baroreceptor → increase sympathetics, decrease parasypathetics → increase SVR, and venous tone vasoconstrict splanich vessels and skeletal muscle.
- Vasoconstrictors = vassopressin, epinephrine.
- Chemoreceptors → central and peripheral chemoreceptors respond to acidosis core perfusion and increase SVR.
- Renal → rennin angiotensin → aldosterone + ADH → increase Na+ and H_2O inc CO and arterial pressure.
- Cerebral ischemia produces very intense sympathetic discharge to protect brain (Fig. 6.8).

Heart: myocardial hypoxia → systolic and diastolic dysfunction (decrease cardiac output).

- Kidneys: "pre-renal" or renal failure occurs secondary to vasocontriction and reduced GFR; leads to acute tubular necrosis → damaged tubules fail to absorb salt.
- Lungs: decreased compliance → decreased ventilation → V/Q mismatch → hypoxemia and ARDS.
- Also release of inflammatory mediators: TNF-α, nitric oxide.
- Injury to endothelial cells allowing leakage of fluids and neutrophils into interstium.
- Gastrointestinal tract: vasoconstriction of splacnich vessels; infarction → Ileus → fluid imbalance and acidosis.
- Brain:

CPP=MAP-ICP;

MAP < 60mmHg cerebral perfusion decreases secondary to pressure below autoregulatory range.

Conclusion (Fig. 6.9):

Clinical findings of shock:

- Tachycardia.

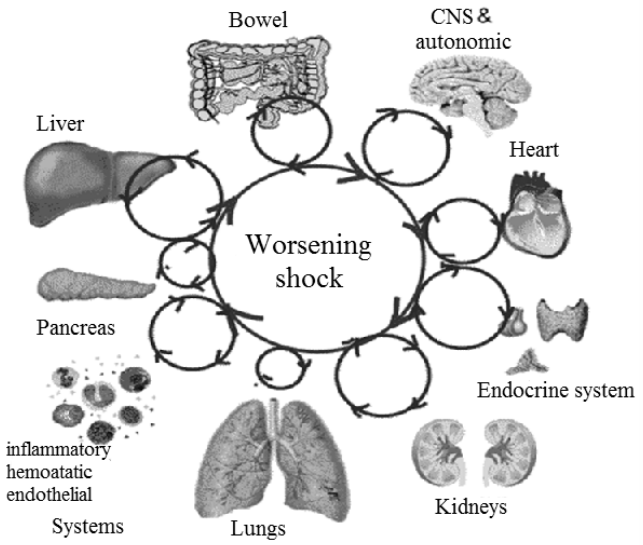


Figure 6.8. *The mechanisms decompensation*

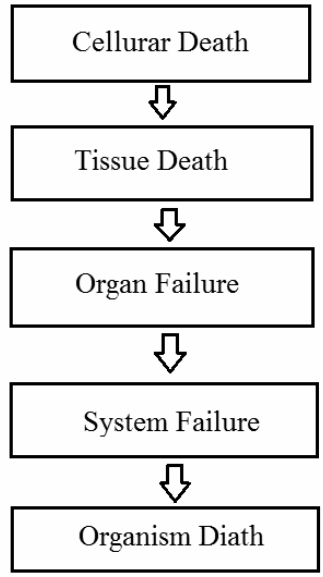


Figure 6.9. *Pathophysiology*

- Hypotension.
- Cool extremities.
- Weak peripheral pulses.
- Prolonged capillary refill (>2 seconds).
- Narrowing of the pulse pressure (<25 mmHg).
- Altered mental status.

Initial Management

In the healthy patient, melena suggests that the bleeding is slow. The patient should be admitted to the hospital and nonemergency workup is indicated. Hemodynamic assessment (blood pressure, pulse, and postural changes) and, if necessary, institution of resuscitative measures are the first steps in the management of upper gastrointestinal bleeding. Clinical prognostic features and the initial response to resuscitation are used to decide whether a patient should be hospitalized and, if so, what level of care should be provided. Patients with clinical characteristics that indicate a high risk of further bleeding or death should be admitted to an intensive care unit. Patients predicted to do well may be admitted to a regular ward or may even be kept in the emergency department until diagnostic endoscopy has been performed.

1. Assess the status of the circulatory system and replace blood loss if necessary.
2. Determine the amount and rate of bleeding.
3. Slow or stop the bleeding by ice water lavage.
4. Discover the lesion responsible for the episode.
5. Make a correct diagnosis of the cause of bleeding.
6. The patient should be questioned about NSAIDs use and any history of bleeding tendency.
7. Blood should be drawn for crossmatching, hematocrit, Hb, and liver function tests.
8. An intravenous infusion should be started and a large bore nasogastric tube inserted.
9. It is common practice to give H_2-receptor antagonist or proton pump inhibitors (e.g., omeprazole). If bleeding continues or if tachycardia and hypotension are present, the patient should be monitored and treated as for hemorrhagic shock.

All of the above tests and procedures can be performed within 1 or 2 hours after admission. Once the bleeding is under control, blood volume has been restored to normal and the patient is adequately monitored additional diagnostic tests should be performed. Once the patient is stabilized, endoscopy should be the first study, usually within 24 hours after admission. Peptic ulcer is the most common cause of massive upper gastrointestinal hemorrhage. Bleeding ulcers in the duodenum are usually located on the posterior surface. As the ulcer penetrates, the gastroduodenal artery is exposed and may become eroded. Since no major blood vessels lie on the anterior surface of the duodenum, ulceration here is not prone to bleeding.

In some patients the bleeding is sudden and massive, manifested by hematemesis and shock. In others, chronic anemia and weakness due to slow blood loss are the only findings. Twice daily Ht readings should be ordered as a check on slow continued blood loss. Rebleeding in the hospital has a mortality rate of about 30%. Patients over the age of 60, with hematemesis actively bleeding at the time of endoscopy, or with an Hb < 8 g/dl have a higher risk of rebleeding.

Endoscopic Therapy

Treatments administered through the endoscope may stop active bleeding or prevent rebleeding. Studies in the early 1980s suggested that endoscopy could not alter the care of patients with acute bleeding from a peptic ulcer. Today, there is ample evidence that the endoscopic appearance of an ulcer provides an excellent complement to clinical factors in assessing the risk of further bleeding or death. Such information dictates management in terms of therapy, level of hospital care, resumption of feeding, and length of hospitalization. Therefore, most patients with acute upper gastrointestinal bleeding should undergo endoscopic examination. Endoscopy should be performed as soon as safely possible in patients at high risk for further bleeding or death. Patients with minor bleeding who have been admitted to a medical ward can wait until the next day for endoscopy. Since the need for admission may be determined by the endoscopic findings (see below), physicians should consider placing patients at low risk in a short-stay area while they await endoscopy. As shown in figure 6.10, subsequent management of bleeding ulcers is determined by the results of endoscopy.

Effective methods include:

- Injection into the ulcer with epinephrine. Injection therapy is a nonthermal method of achieving hemostasis. One of a variety of solutions is injected into the base of the ulcer with a catheter that has a retractable needle (the type of catheter used for esophageal variceal sclerotherapy) (Fig 6.11).

Solutions documented in controlled trials to provide effective hemostasis include absolute ethanol, epinephrine (at a dilution of 1:10 000), polidocanol (a sclerosing agent usually injected immediately after the injection of epinephrine), and even normal saline. The fact that normal saline is effective suggests that at least one mechanism of hemostasis is simply local compression of the blood vessel by the injected solution. Whether the addition of a sclerosant after epinephrine or saline injection is better than either alone is unclear.

- *Cauterization using electrocautery* (Fig. 6.12). Monopolar electrocoagulation, bipolar electrocoagulation, and heater-probe therapy use thermal contact – that is, local tamponade and heat to effect hemostasis. Monopolar electrocoagulation has been

Figure 6.10. *Algorithm for the treatment of patients with bleeding peptic ulcers*

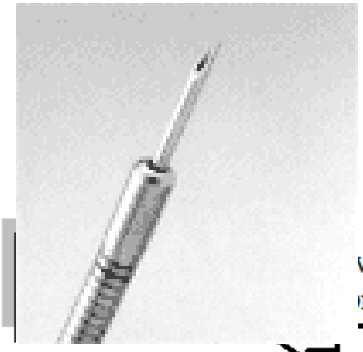


Figure 6.11. *The type of catheter*

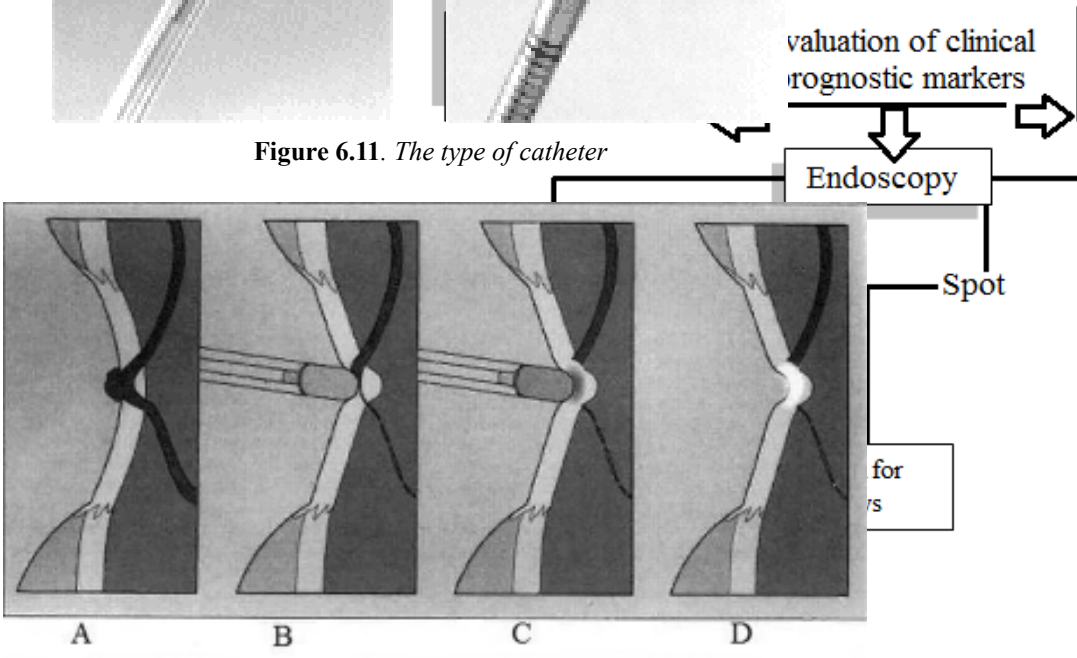


Figure 6.12. *Coagulation by a thermal-contact device:*
a vessel is visible in the base of the ulcer (panel A); the device is applied to the center of the vessel (panel B); after the device has been properly positioned, energy is applied, coagulating the compressed vessel (panel C); when the probe is removed, a white burn is visible where the energy was applied (panel D)

replaced by the other two methods primarily because of the concern that monopolar electrocoagulation causes a greater degree of tissue injury. In studies with animals, both bipolar electrocoagulation and heater probes are highly effective in stopping bleeding and coagulating mesenteric arteries up to 2 mm in diameter. As mentioned above, most ulcers bleed from vessels that are smaller than 2 mm.

- Laser coagulation (Fig. 6.13).
- Argon plasma coagulation.
- Hemostatic clips (Fig. 6.14).

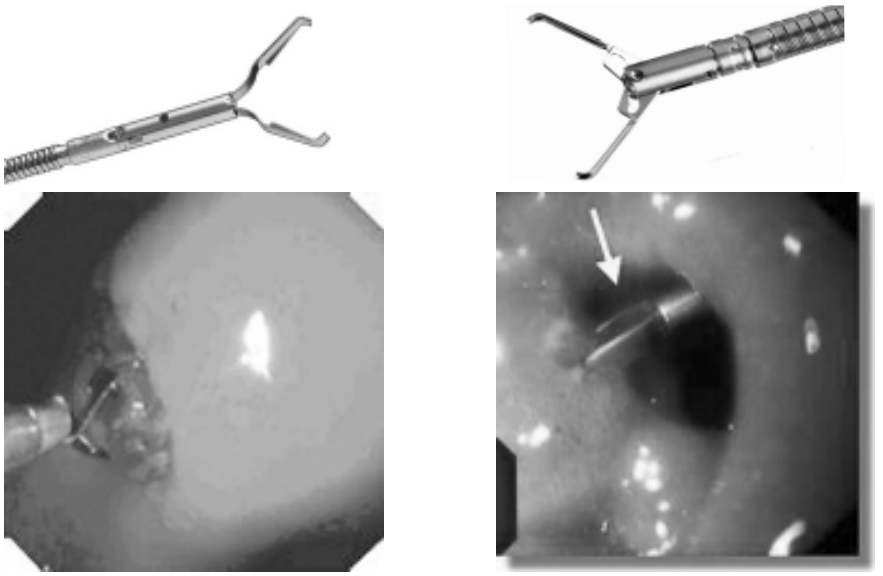


Figure 6.14. *Hemostatic clips*

Angiographic Therapy

Angiographic therapy is rarely used to treat patients with bleeding ulcers and should be considered only for severe, persistent bleeding if surgery poses an extremely high risk and endoscopic therapy has been unsuccessful or is unavailable. Ulcers may stop bleeding with an intraarterial infusion of vasopressin in up to 50 percent of cases. Uncontrolled studies suggest that arterial embolization with an absorbable gelatin sponge (Gelfoam), an autologous clot, tissue adhesives, or mechanical occlusion devices may control bleeding identified angiographically in approximately 75 to 80 percent of cases, although recurrent bleeding may occur in over half these cases (Fig. 6.15). Complications of embolization include ischemia with stenosis, infarction, perforation, or abscess formation in target and nontarget organs.

Emergency Surgery

Patients who have recurrences of bleeding from ulcers despite medical therapy are candidates for surgical therapy (about 10% of patients bleeding from a peptic ulcer require emergency surgery). The available operations include proximal gastric vagotomy, truncal vagotomy and drainage (e.g., pyloroplasty), or vagotomy and antrectomy (Fig. 6.16).

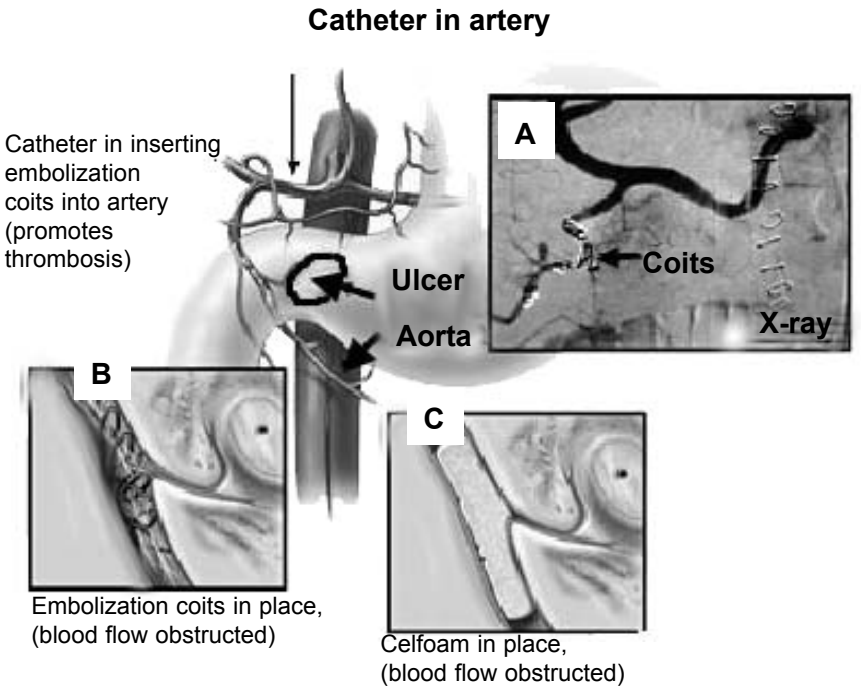


Figure 6.15. *Coil embolization of bleeding ulcer*

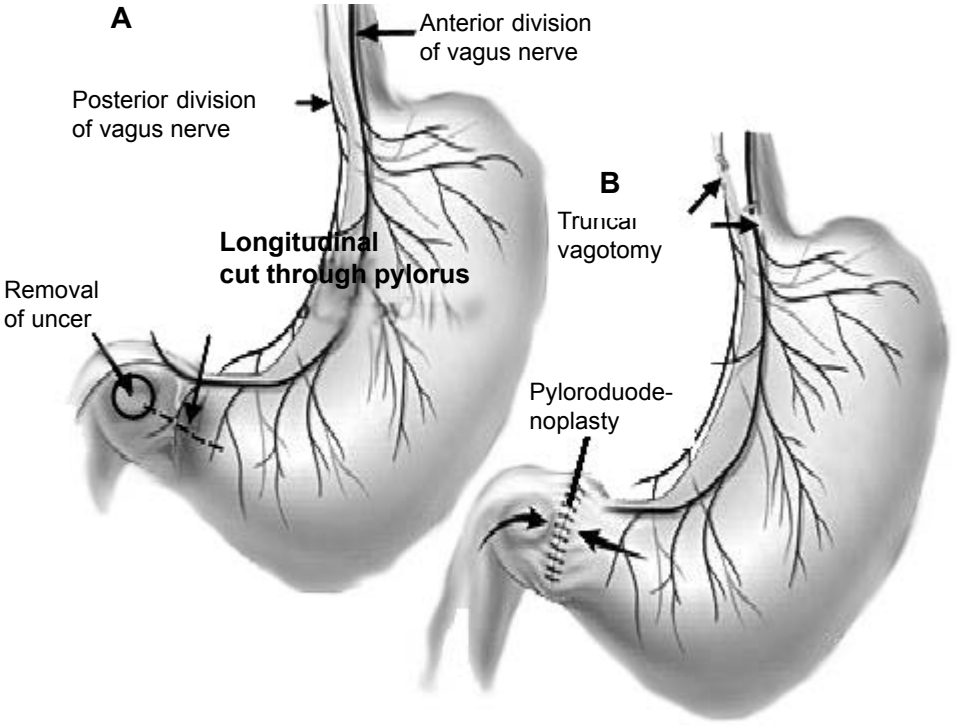


Figure 6.16. *Removal of ulcer, pyloroplasty and truncal vagotomy for bleeding ulcer*

Medical Therapy

Gastric acid and H. pylori infection are the two factors that must be considered among patients who were not using NSAIDs when the bleeding occurred. Maintenance therapy with H_2-receptor antagonists or proton pumpblocker is widely used to prevent recurrent peptic ulceration. This is best accomplished with combination therapy, such as a two-week course of bismuth subsalicylate (two tablets four times daily) plus metronidazole (250 mg four times daily) and tetracycline (500 mg four times daily).

Medical therapy of the hypovolemic shock

Regardless of the cause, the restoration of the circulating volume is priority. As soon as the airway is maintained and oxygen administered the next step is to commence replacement of fluids via the intravenous route. Immediately control the bleeding. Restore the casualty's blood volume; by giving infusions of isotonic crystalloid solutions. Blood transfusions, packed red blood cells (RBCs), Albumin (or other colloid solutions), or fresh-frozen plasma are necessary for loss of large amounts of blood (e.g. greater than 20% of blood volume.

Opinion varies on the type of fluid used in shock. The most common are: crystalloids – such as sodium chloride (0.9%), or Lactated Ringer's; colloids – for example, polysaccharide (Dextran), polygeline (Haemaccel), gelatin (Gelofusine); blood – Essential in severe hemorrhagic shock. Vasoconstrictor agents have no role in the initial treatment of hemorrhagic shock.

Prognosis. The mortality rate for an acute massive hemorrhage is 15%.

6.4. PERFORATED PEPTIC ULCER

Perforations usually occur anteriorly due to the absence of protective viscera and major blood vessels on this surface. Immediately after the perforation, the peritoneal cavity is flooded with gastric secretions and a chemical peritonitis develops (Fig. 6.17).

In a small percentage of cases, the perforation becomes sealed by adherence of the liver or omentum. In such patients the process may be self limited but a subphrenic abscess may develop.

The first report of a series of patients presenting with perforation of a peptic ulcer was made in 1817 by *Travers*. The earliest operative description was made by *Mikulicz* in 1884 but the first successful operation for a perforated duodenal ulcer was not until 1894.

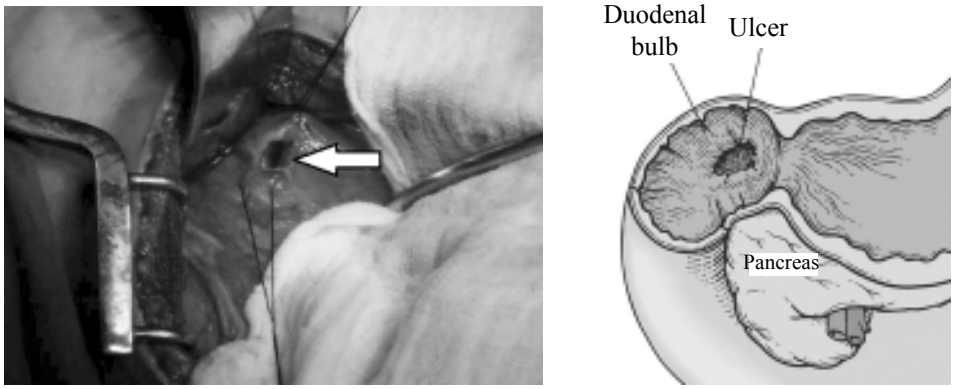


Figure 6.17. *Perforated ulcer (arrow)*

In the world the clinical picture is different with a high male: female ratio (approximately 8:1), younger age, and a strong link with cigarette smoking. In addition in the third world there is a high incidence of patients who present late and this may partly account for the high mortality (20%) reported in some studies.

Most often a peptic ulcer that perforates is situated on the anterior surface of the duodenum; much less frequently it is situated on the anterior surface of the stomach, usually near the lesser curvature or the pyloric antrum. Rarely those an ulcer on the posterior wall of the stomach perforates into the lesser sac. In 80 percent of cases, there is a history – often a long history of peptic ulceration. In 20 percent there is no such history; it is a 'silent' chronic ulcer that perforates, especially in those patients who are being treated with corticosteroids (cortisone, prednisolon) or NSAIDs.

The gastric or duodenal contents escape through the perforation into the general peritoneal cavity, resulting in peritoneal irritation. The peritoneum reacts to this chemical irritation by secreting peritoneal fluid copiously and this gives relief of pain for a short time. This *stage of reaction* lasts from 3 to 6 hours.

THE STAGES OF PERFORATED PEPTIC ULCER

1st stage – shock stage (phase of chemical peritonitis) [first 3–6 hours].

It is characterized by severe pain in abdomen (stabbing pain). This pain starts in the epigastric region and spreads quickly to the whole abdominal cavity. There is loss of abdominal wall movement due to the severe pain. The abdominal muscles become tense (hard like wood), but not in all patients. However, these symptoms are not accurate if perforation of duodenal ulcers occurs. This is because of the fact that the duodenal contents are alkali, rather than acidic in nature. During palpation severe pain in the upper part of abdomen is felt. In time, sensitivity of the nerve ends in the parietal peritoneum are disturbed, as a result pain decreases and the patient feels better, that's when the 2nd stage starts "the stage of false improvement" [6–12 hours].

2nd stage – the stage of false improvement. The condition of the patient as a whole improves, because there is a dissolution of acid contents from a digestive tract by liquid. The face has normal color; pulse, BP and temperature start to stabilize, and muscles in the epigastric region start to become rigid. But, some clinical features indicate a pathologic condition in abdominal cavity: the patient remains in forced position (movement can cause severe pain), during percussion dull sounds are not heard on the liver due to the presence of gas above it, positive signs of inflammation of peritoneum continue to be seen.

3rd stage. This is the stage **of progressive peritonitis or toxic stage** (severe abdominal sepsis) – it develops after 12 hours of perforation. First clinical features are: frequent vomiting, continuation of dehydration. The condition of the patient becomes severe again. The skin is dry, fever starts; pulse reaches 100–120 bpm, distension of abdomen increases, absence of peristalsis.

CLINICAL FEATURES

The most characteristic symptom is the suddenness of the onset of epigastric pain. The pain rapidly becomes generalized although occasionally it moves to the right lower quadrant. The abdomen is held still, moving little or not at all with

respiration. The whole abdomen is tender with board-like rigidity. Rebound tenderness (Blumberg's sign).

It is dull to percussion. Sufficient gas may have escaped to reduce liver dullness in the midaxillary line. The stage of diffuse peritonitis develops, accompanied by silent abdominal distension. Enough free fluid may have collected to be clinically detectable. The rising pulse rate marks the progressive deterioration in the patient's condition with each hour that p asses without operative treatment.

1. Clinical findings:

All clinical symptoms A. Mondor (1938) divided into two groups: the main and side.

- The main : 1) pain as a knife blow; 2) ulcerative anamnesis (however, in a number of patients perforation occurs in the full health – the so-called "mute" ulcers are noted in 8–10%); 3) the abdomen exhibitits a board-like rigidity.
- The side: functional, physical and common symptoms.
- Functional symptoms are general weakness, thirst, dry mouth, vomiting, stool and gas retention.
- Physical findings are found on examination, palpation, percussion and auscultation: 1) forced position; 2) hyperesthesia of the skin of the anterior abdominal wall and positive sing of **Blumberg:** increased pain upon removal of pressure on the abdominal wall (rebound tenderness); 3) **Spizharnogo's sing:** disappearance of hepatic dullness, high tympanitis in epigastrium; 4) **Bernstein's sing:** tightening of the testicles to the external opening of the inguinal canals in men; 5) **Kulenkampf's sing:** in digital rectal examination, acute soreness in the vesicular-rectal (Douglas) space; 6) **Gusten's triad:** the spread of heart tones to the navel; peritoneal friction at the edge of the costal arch; metallic ringing (or silvery noise) that appears when inhaling and is associated with the presence of free gas emerging through the perforation.
- Common symptoms are the pulse, breathing, temperature. Changes in these indicators correspond to a certain phase of the clinical course.

2. Laboratory findings: mild leukocytosis – 12 000/uL, after 12–24 hours may rise to 20 000/uL and more.

3. Imaging Studies

Plain X-rays of the abdomen reveal free subdiaphragmatic air in 85% of patients. Both supine and upright plates should be taken. A left lateral decubitus position may be a more practical way to demonstrate free air in the uncomfortable patient.

X-ray of the abdomen with the patient erect reveals a translucent area (free gas) beneath the right cupola of the diaphragm in 70 per cent of cases (Fig. 6.18).

Investigation include also:

- Rectal examination for the detection of the condition of the sphincter ani, the sensitivity of pelvic peritoneum, the character and color of fecal masses.
- Biochemical parameters of blood, blood and urine analysis
- Analysis of X-ray of abdomen in case of perforated ulcer, providing the assessment of pneumogastrography (**Hennelt's test:** through a probe in a stomach 1500 ml of air is introduced).
- Gastroduodenoscopy (Fig. 6.19).

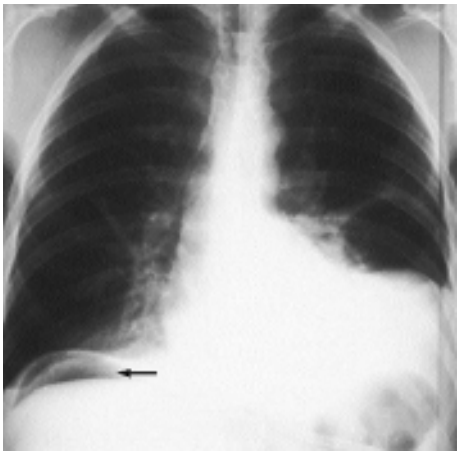


Figure 6.18. *Plain radiograph of a perforated duodenal ulcer showing gas beneath the diaphragm (arrow)*

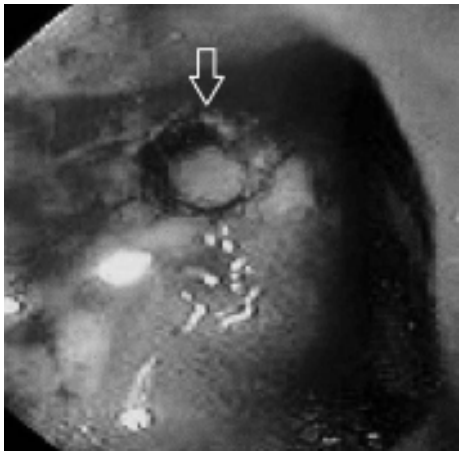


Figure 6.19. *Gastroduodenoscopy: perforated peptic ulcer (arrow)*

Differential Diagnosis:

- Acute pancreatitis: does not have such sudden onset as perforated ulcer; high serum amylase.
- Acute cholecystitis.
- Intestinal obstruction – pain more colic like.

The simultaneous onset of pain and free air in the abdomen in the absence of trauma usually means perforated peptic ulcer.

TREATMENT ARE ONLY SURGICAL

Before surgery:

- Pass a nasogastric tube and empty the stomach to reduce further contamination of the peritoneal cavity.
- The patient is kept nil per os.
- Blood for laboratory studies is taken.
- Commence with intravenous antibiotics.
- Fluid resuscitation.

Surgery

The simplest surgical treatment is laparotomy with closure by securely plugging the hole with omentum sutured in place.

Remember the ulcer still exits and medical treatment for peptic ulcer disease should continue.

The patient is placed in the supine position. A midline incision provides the most expeditious entry into the abdominal cavity. The incision can be extended to the symphysis pubis if necessary. Once the abdomen is entered, the stomach and duodenum are carefully examined to determine the site of perforation. If the anterior surface of the stomach and duodenum shows no abnormalities, the gastrocolic ligament is serially divided between clamps to allow entrance into the lesser sac and inspection of the posterior surface of the stomach. The choice of operative procedure depends on variables, such as the presence of shock, life-threatening comorbid conditions, the degree of contamination of the upper abdomen, the amount and duration of perforation,

and whether the patient has a history of or currently has intraoperative evidence of chronic peptic ulceration. In the presence of life-threatening comorbid conditions and severe intra-abdominal contamination, the safest technique for an acute anterior duodenal perforation is a simple closure with a Graham patch using omentum (Fig. 6.20). Several full-thickness simple sutures are placed across the perforation using 2–0 or 3–0 silk sutures. A segment of omentum is placed over the perforation. The silk sutures are secured. If contamination of the upper abdomen is minimal and the patient is stable, a definitive ulcer procedure can be performed.

For a perforated duodenal ulcer, this may include a highly selective vagotomy, a truncal vagotomy and pyloroplasty, or vagotomy and antrectomy (Fig. 6.21).

For a perforated gastric ulcer, the procedure performed depends on the patient's condition. If the patient is moribund, the ulcer is best excised by grasping it with multiple Allis clamps and using a GIA-60 linear stapler. Alternatively, the ulcer can be excised with electrocautery, and the defect is approximated with a 2-layer closure with inner continuous 3–0 absorbable sutures and outer interrupted Lambert sutures using 2–0 or 3–0 silk sutures. In a stable patient, the ulcer is excised and sent for frozen section analysis to exclude malignancy. For a benign gastric ulcer, a distal gastrectomy with either a Billroth I gastroduodenostomy or a Billroth II gastroduodenostomy is performed.

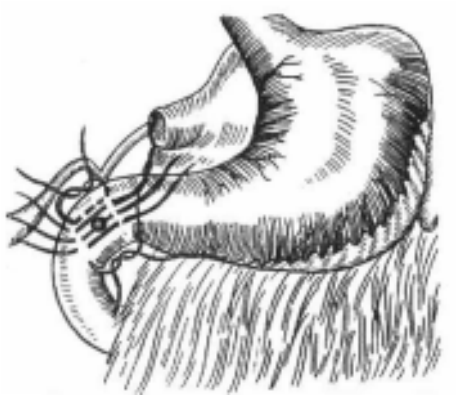


Figure 6.20. *Closure of the perforation*

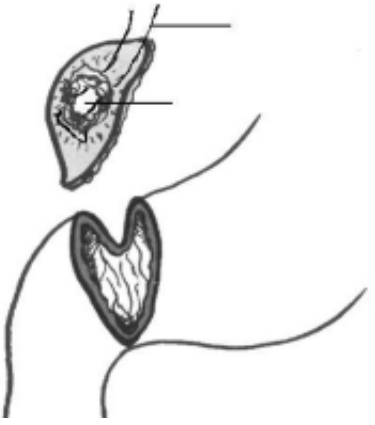


Figure 6.21. *Removal of a perforated duodenal ulcer and pyloroplast*

Laparoscopic Surgery

The traditional management of a perforated duodenal ulcer has been a Graham Omental Patch and a thorough abdominal lavage. Recently, this has been shown to be able to be performed using a laparoscope. The only proven advantage of the laparoscopic technique appears to be decreased postoperative pain. Operating times are longer compared to open techniques and hospital time appears to be similar to conventional treatment. This technique has not been subjected to any large prospective trials and at present must not be considered as standard management.

Immediate Definitive Surgery

Over the last one hundred years a number of attempts have been made to improve upon the results of simple closure and lavage. This has been in response to the large number of patients (25–5%) who continue to have symptoms attributable to their ulcer diathesis after surgery.

The incidence of ulcer symptoms, in most studies, is related to whether the ulcer is acute or chronic (history greater than 3 months) as judged by preoperative history. Patients with chronic ulcer symptoms generally have a higher incidence of subsequent recurrent ulcers. Up to 71% come to require subsequent definitive surgery although in most studies the figure is considerably less than this.

Since the 1940's the concept of immediate definitive ulcer surgery has been promulgated and debated amongst surgeons. There is good evidence that, in an emergency situation, highly selective vagotomy (proximal gastric, or parietal cell vagotomy) combined with simple omental patch closure of the perforation, in patients without the risk factors mentioned above, is just as effective as that performed in the elective setting. This is associated with a less than 1% mortality rate and a 4–11% ulcer recurrence rate. The success of this operation is surgeon-dependent. Truncal vagotomy with drainage has its advocates as an expedient operation familiar to most surgeons.

Vagotomy and pyloroplasty are adequate methods treatment

Immediate definitive ulcer surgery has not gained widespread popularity due to an unfounded feeling that it is associated with a higher mortality than simple closure. It has been argued that if blanket definitive surgical approach were adopted then 50–60% patients would be at risk of suffering from complications of surgery they did not require. In experienced hands, however, a highly selective vagotomy is associated with very low morbidity

The immediate post-operation treatment includes:

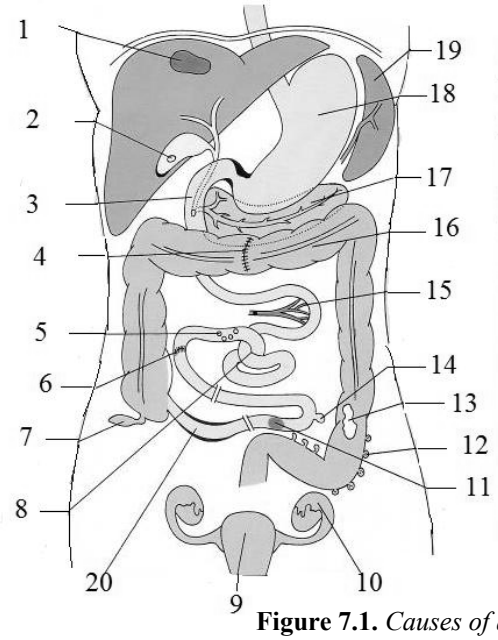
- analgesics;
- gastric aspiration;
- intravenous fluid;
- antibiotic therapy;
- therapy for H pylori (combination of Bismuth, Metronidazole and Amoxycillin;
- proton pumpblocker, antisecretory drugs (proton pump inhibitors, H_2 receptor antagonists);
- breathing exercises are important in elderly people, and in cases where operation is more than 6 hours after perforation.

Prognosis: 2–15% of patients with perforated ulcers die.

Chapter 7

ACUTE PERITONITIS

Peritonitis results from any local trigger of inflammation (Fig. 7.1). Usually infection is the trigger, although infection may not necessarily be present at the earliest stage. Forexample, sterile peritonitis may occur in the localized peritoneal space surrounding an infected but resectable intraabdominal organ, such as the appendix or gallbladder. In contrast, there may be contamination of the peritoneum from a defect in the intestinal wall, before establishment of infection or onset of an inflammatory response, e.g., immediately following penetrating abdominal trauma. Peritonitis has been categorized as primary, secondary, or (more recently) tertiary. Peritonitis complicating peritoneal dialysis can be considered as an additional category.



1. Liver abscess
2. Gallbladder perforation
3. Duodenal ulcer perforation
4. Anastomotic dehiscence in the colon
5. Small bowel perforation
6. Small bowell suture line (anastomotic dehiscence)
7. Perforated appendicitis
8. Bowell obstruction
9. Perforated uterus
10. Perforated adnexitis
11. Enteritis
12. Colon diverticulitis
13. Perforation of colon carcinoma
14. Perforation of a Meckel diverticul
15. Occlusion of mesenteric vessel
16. Ulcerative colitis
17. Pancreonecrosis
18. Perforation of gastric ulcer or carcinima of the stomath
19. Spleen infarction
20. Bowel obstuction

Figure 7.1. *Causes of abdominal infection*

7.1. ANATOMY

The peritoneum. Peritoneum is a membrane that covers the surface of both the organs that lie in the abdominal cavity and the inner surface of the abdominal cavity itself.

The peritoneal cavity is a potential space containing the abdominal viscera. The peritoneum provides a frictionless surface over which the abdominal viscera can freely move, and the mesothelial lining secretes fluid that serves to lubricate the peritoneal surfaces. The peritoneum covers the inner side of the parietes which is known as parietal peritoneum. Peritoneum also covers the outer surface of the abdominal viscera which is known as visceral peritoneum. Between these two layers lies the potential space, which is known as peritoneal cavity. Normally, there is about 100 ml of clear straw-colored fluid in the adult. The quality and quantity of this fluid may change with various pathological conditions.

Structure. The peritoneum is a single layer of flat mesothelial cells resting on a layer of elastic tissue containing macrophages, fat cells and some collagen and elastic

fibers. Beneath the peritoneum, supported by a small amount of areolar tissue, lies a network of lymphatic vessels and reach plexus of capillary blood vessels from which absorption and exudation occur. The parietal peritoneum is reinforced by the transversalis (endoabdominal) fascia which lies external to it. The visceral peritoneum is identical with the serosa or capsule of the intra-abdominal organs. It must be remembered that peritoneal cavity is a completely closed sac except the ends of the fallopian tubes, in case of females through which it communicates with the exterior. The peritoneal cavity is divided into the greater sac and the lesser sac. The lesser sac lies behind the stomach and lesser omentum, whereas the greater sac covers the whole of the abdomen including the pelvic cavity.

7.2. PATHOPHYSIOLOGY

Peritonitis may be either localized or generalized. The factors responsible for localization of the inflammation are both anatomic and pathologic.

Anatomic factors. The peritoneal cavity is divided into a greater and a lesser sac; these communicate with each other through the foramen of Winslow. The greater sac is further divided into a supracolic and an infracolic compartment by the mesentery of the transverse colon. The mesentery of the small bowel, which extends from the left upper quadrant to the right lower quadrant, divides the infracolic compartment into a right and left half. These peritoneal folds subdivide the peritoneal cavity into compartments and deter the spread of infection from one compartment to another. The parietal peritoneum is innervated by both somatic and visceral afferent nerves and so it is quite as sensitive as the skin. The anterior parietal peritoneum is the most sensitive, while the pelvic peritoneum is the least sensitive. Local injury or inflammation of parietal peritoneum leads to protective voluntary muscular guarding and later on to reflex muscular spasm, the signs which are indicative of such insult. Perception of pain is indicated as localized tenderness and rebound tenderness.

In contrast to the parietal peritoneum, the visceral peritoneum receives afferent innervations only from the autonomic nervous system and hence is insensitive. However visceral afferent nerves respond well to traction or distension and less to pressure, but no receptors for pain or temperature.

Pathologic factors. When the inflammation responsible for acute abdomen progresses slowly, there will be sufficient time for adhesions to form between the inflamed organ and adjacent structures, thus confining the inflammation. In contrast, when the inflammation is sudden in onset and is associated with massive contamination of the peritoneal cavity (as in a perforated ulcer) there is not enough time for adhesions to form and confine the inflammation, and diffuse peritonitis ensues. The greater omentum plays an important role in confining inflammation by enveloping and adhering to the inflamed structure. In children this structures is not well developed, and the barrier formed against the spread of infection is less effective. Therefore, children are more prone to develop generalized peritonitis and at a much earlier stage of the disease than are adults. With the onset of peritonitis, peristaltic activity in the adjacent coils of the intestine ceases – this again helps form an effective barrier the spread of infection. Stimulation of peristalsis, either by ingested food or by ill-advised administration of cathartics, would interfere with nature's attempt to confine the inflammation.

The surface area of the peritoneum is about 2 m^2, which is approximately identical to the area of the skin. The peritoneum acts as a semipermeable membrane and permits transport of water, electrolytes and peptides both directions according to the osmotic concentration. Absorption from the peritoneal cavity of particulate matter takes place when the size is the same or less than bacteria. Such particulate matters may include bacteria, formed blood elements, proteins, etc. Such absorption takes place through stomata to the lymphatic channels. These lymphatic channels are also responsible for the appearance of the so-called sympathetic pleural effusions whenever transabdominal inflammatory process takes place as invariably subdiaphragmatic lymphatic plexus is involved early.

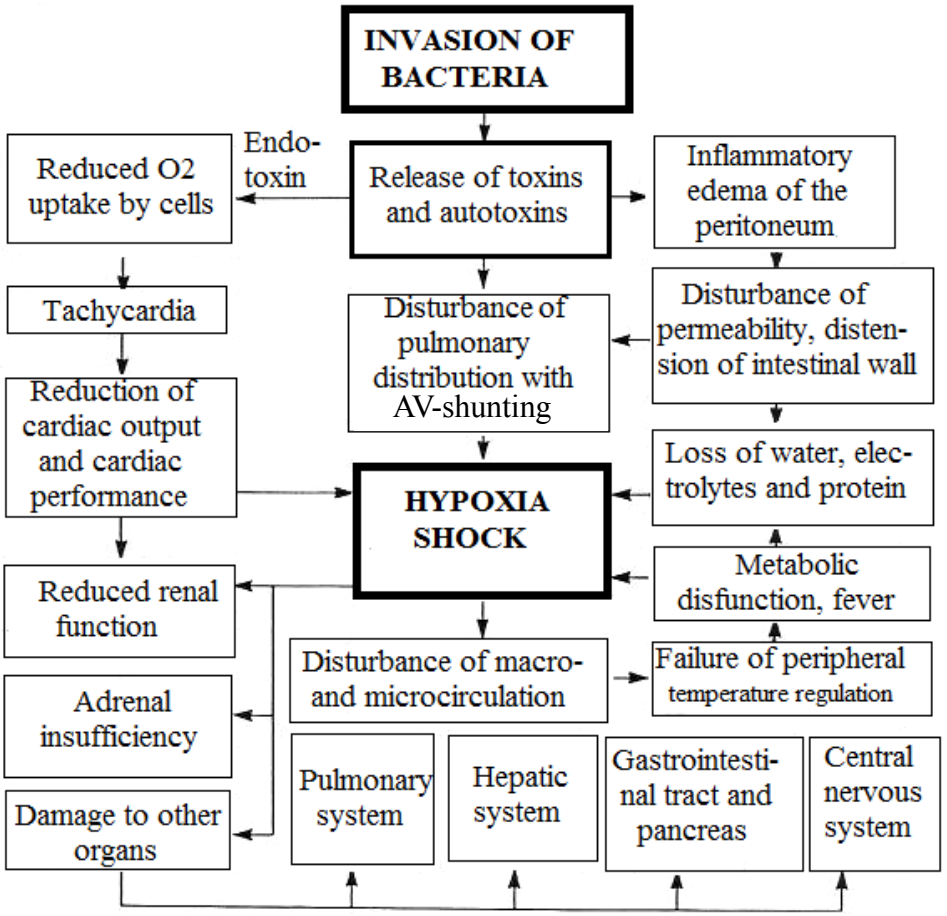


Figure 7.2. *Pathophysiology of peritonitis*

Peritonitis is a complex disease state that results in many pathophysiologic alterations that affect multiple organs (Fig. 7.2). The clinician must possess a thorough understanding of the pathophysiology and pathogenesis of the disease so that effective treatment can be instituted. Treatment should be directed at eliminating the cause of peritonitis and preventing or reversing the resulting pathophysiologic alterations.

The peritoneum heals very rapidly following injury. Even large defects can be restored in a matter of hours. The majority of cells involved in healing of the peritoneal wound are derived by differentiation of stem cells present within the subperitoneal

tissues in the surrounding area. Growth of mesothelial cells occur from margins of the defect. If normal peritoneal healing is delayed or incomplete, adhesions form. The adhesions may be transient, eventually resolving as delayed healing becomes complete or they may be permanent in nature. Fibrins elaborated from the inflamed peritoneal mesothelium are the scaffold upon which adhesions are built. Formation of adhesions is a protective response helping to localize the peritoneal insult and is also an adaptive healing response which helps to bring additional blood supply to the ischemic injured areas of the peritoneum. The beneficial effects of adhesions should always be remembered.

Toxemia and septicemia, shock, hemorrhage, abdominal pain, paralytic ileus, fluid accumulation, and adhesions all contribute to the clinical signs and progression of peritonitis. Toxins produced by bacteria and tissue breakdown are readily absorbed through the peritoneum. Bacterial or chemical irritants increase serosal capillary permeability resulting in leakage of plasma proteins, solutes, and water into the peritoneal cavity. Exudation of protein-rich fluid can result in hypoproteinemia and bacterial proliferation. Endotoxins absorbed from the peritoneal cavity have systemic effects leading to hypotension, shock, and systemic inflammatory response syndrome (SIRS) and disseminated intravascular coagulation (DIC). Endotoxins, myocardial depressant factor, acid-base, and electrolyte disturbances directly affect the cardiac function, leading to reduced cardiac output. The combined effect of large fluid losses into the peritoneal cavity and vasodilatory effects of absorbed toxins can produce profound hypotension and hypovolemia. Rupture of the gastrointestinal tract, with spillage of large volumes of intestinal contents, leads to acute peritonitis. Death due to endotoxic shock may occur suddenly with limited clinical signs or lesions. Shock and hemorrhage associated with rupture of the gut or uterus often lead to death in animals with infection of the gastrointestinal or reproductive tracts; however, shock and hemorrhage may be minor following uterine rupture in cows. Peritonitis may not develop if the uterine contents are not contaminated, but it may follow if the uterus is not repaired or healed within a few days. Paralytic ileus is a frequent result of acute peritonitis and may also follow intestinal obstruction or surgery, leading to functional obstruction and increased mortality rate if it persists. Large volumes of inflammatory exudates may be secreted into the peritoneal cavity during peritonitis and may lead to impaired respiration by impinging on the diaphragm. Peritoneal trauma leads to secretion of fibrinogen and formation of fibrinous adhesions. Such adhesions help localize the inflammation but may cause mechanical or functional obstruction of the gastrointestinal tract (Fig. 7.2).

The cytokine response in peritonitis has been the subject of a recent excellent review. Undoubtedly, many of the systemic as well as abdominal manifestations of peritonitis are mediated by cytokines, such as TNF, IL-1, IL-6, IFN-γ, and others. Cytokines appear in the systemic circulation of patients with peritonitis and to a much greater extent in the peritoneal exudates. These cytokines are produced by macrophages and other host cells in response to bacteria or bacterial products, such as endotoxin, or by tissues traumatized during the operative procedure. Another potential source is direct translocation of cytokines through the intestinal barrier.

Cytokine responses have been studied in the peritoneal exudate in experimental animal models of peritonitis, in patients with spontaneous bacterial peritonitis, in patients undergoing continuous ambulatory peritoneal dialysis, and in patients, with

severe secondary bacterial peritonitis who were undergoing planned relaparotomy. Antibodies to TNF have been found to fail to protect against death and failed to reduce serum levels of IL-1 and IL-6 in an experimental model of peritonitis. In contrast, antibodies to endotoxin were found to prevent death in this model, as well as to reduce bacterial numbers in the peritoneal exudate. Antibodies to IFN-γ also afforded a protective effect both in this model of experimental peritonitis and following intravenous injection of endotoxin (Fig. 7.3).

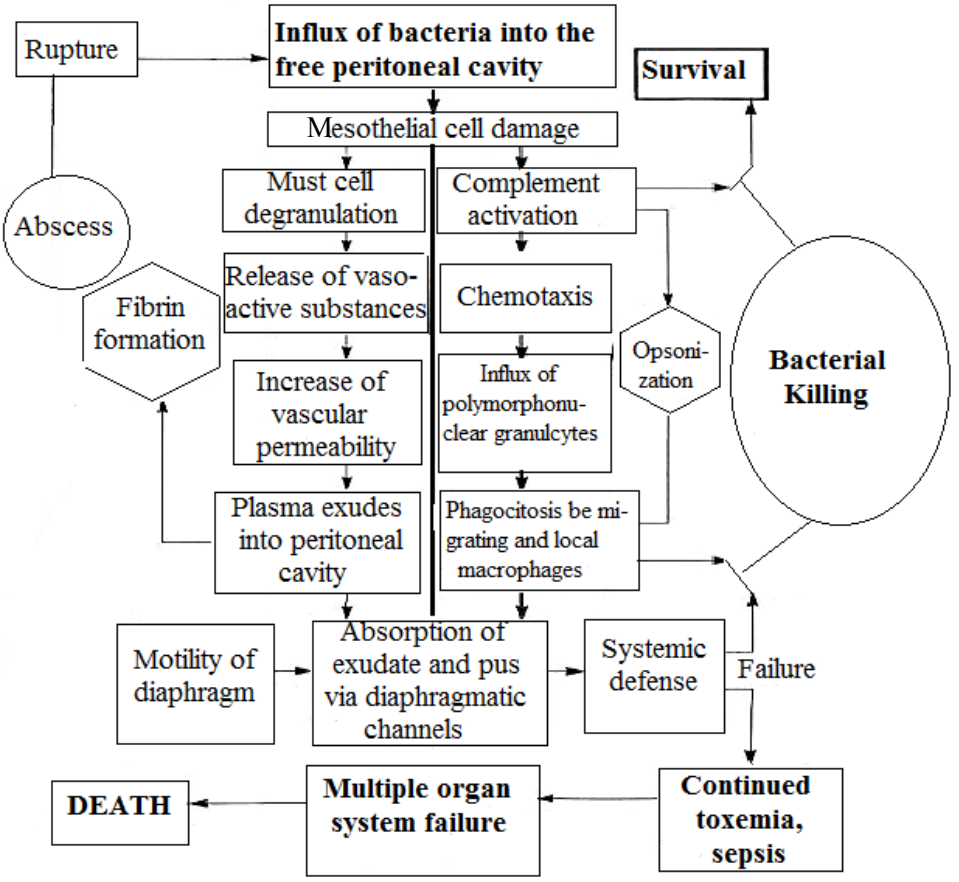


Figure 7.3. *Pathogenesis of multiple organ system failure*

7.3. CLASSIFICATION

I. Three Major Types: primary, secondary and tertiary peritonitis.

Primary: primary peritonitis refers to inflammation of the peritoneal cavity without a documented source of contamination.

The vast majority of cases are due to bacterial infection: it is also commonly known as spontaneous bacterial peritonitis. Usually it occurs in the presence of ascites from any of a variety of underlying conditions. Although primary peritonitis may occur in children without predisposing disease, it is especially associated with postnecrotic cirrhosis and nephrotic syndrome. In adults, primary peritonitis develops in up to 25% of patients with alcoholic cirrhosis but has also been reported to occur in adults with postnecrotic cirrhosis, chronic active hepatitis, acute viral hepathis

congestive heart failure, metastatic malignant disease, systemic lupus erythematosus, and lymphedema and, rarely, in adults with no underlying disease. The presence of ascites appears to be the common link among these various conditions.

The route of infection in primary peritonitis is usually not apparent but is thought to be hematogenous, lymphogenous, via transmural migration through an intact gut wall from the intestinal lumen, or, in women, from the vagina via the fallopian tubes. In cirrhotic patients the hematogenous route is most likely. Organisms removed from circulation by the liver may contaminate hepatic lymph and pass through the permeable lymphatic walls into the ascitic fluid. In addition, portosystemic shunting greatly diminishes hepatic clearance of bacteremia, which would tend to perpetuate bacteremia and increase the opportunity to cause metastatic infection at susceptible sites such as the ascitic collection.

Enteric bacteria may also gain access to the peritoneal cavity by directly traversing the intact intestinal wall. In an animal model, *Escherichia* coli passes from the bowel into the peritoneal cavity after the introduction of hypertonic solutions into the peritoneum. The infrequent occurrence of bacteremia and the multiplicity of species in peritoneal fluid when anaerobic bacteria arc involved suggest that transmural migration of bacteria is the probable route of infection of ascitic fluid in most of these patients.

Transfallopian spread is also suggested by the development of primary peritonitis in women with intrauterine devices The route of spread in women with gonococcal or chlamydial perihepatitis ("Fitz-Curtis syndrome") is presumably via the fallopian tubes and paracolic gutters to the subphrenic space, but it may also be hematogenous.

Although tubcrculous peritonitis may result from direct entry into the peritoneal cavity of tubercle bacilli (from the lymph nodes, intestine, or genital tract in patients with active disease of these organs), it is more likely to be disseminated hematogenously from remote foci of tuberculosis, most commonly in the lung. Tuberculous peritonitis may become clinically evident after the initial focus has completely healed.

Secondary: most surgical peritonitis is secondary to bacterial contamination from the gastrointestinal tract or biliary tract.

Common Causes of Secondary Peritonitis (see Table 7.1, 7.2).

Secondary intraabdominal infection usually is due to spillage of gastrointestinal or genitourinary microorganisms into the peritoneal space as a result of loss of integrity of the mucosal barrier. Examples include appendicitis, diverticulitis, cholecystitis penetrating wound of the bowel, and perforation of a gastric or duodenal ulcer. Secondary infection is relatively common, taking the form of either generalized peritonitis or localized abscesses. Abscesses may be restricted to the immediate peritoneal space around a diseased intraabdominal organ, such as pericholecystic, periappendiceal, or peridiverticular abscesses, or to certain peritoneal recesses, such as ing cytomegalovirus enterocolitis has been described as a common cause of acute abdomen in patients with AIDS.

Tertiary peritonitis has been conceived as a later stage in the disease, when clinical peritonitis and systemic signs of sepsis (e.g.. fever, tachycardia, tachypnea, hypotension. elevated cardiac index, low systemic vascular resistance, leukopenia or leukocytosis, and multiorgan failure) persist after treatment for secondary peritonitis

Table 7.1 – *Common causes of secondary peritonitis*

Source Regions	Causes
Esophagus	Borhaave syndrome Malignancy Trauma (mostly penetrating) Iatrogenic
Stomach	Peptic Ulcer Perforation Malignancy (e.g., adenocarcinoma, lymphoma, gastrointestinal stromal tumor) Trauma (mostly penetrating) Iatrogenic
Duodenum	Peptic Ulcer perforation Trauma (blunt and penetrating) Iatrogenic
Biliary tract	Cholecystitus Stone perforation from gallbladder (i.e., gallstones ileus) or common duct Malignancy Choledochal cyst (rare) Trauma (mostly penetrating) Iatrogenic

Table 7.2 – Common causes of secondary peritonitis

Source Regions	Causes
Pancreas	Pancreatitis Trauma (blunt and penetrating) Iatrogenic
Small bowel	Ischemic bowel Incarcerated hernia (internal and external) Closed loop obstruction, Crohn disease Malignancy (rare) Meckel diverticulum Trauma (mostly penetrating)
Large bowel and appendix	Ischemic bowel Diverticulitis, Malignancy Ulcerative colitis and Crohn disease Appendicitis, Colonic volvulus Trauma (mostly penetrating) Iatrogenic
Uterus, salpinx, and ovaries	Pelvic inflammatory disease (e.g., salpingoophoritis, tuboovarian abscess, ovarian cyst) Malignancy (rare) Trauma (uncommon)

and either no organisms or low-virulence pathogens, such as enterococci and fungi, are isolated from the peritoneal exudate. These organisms may gain access to the peritoneal cavity by contamination during operative interventions, by selection from

the initial polymicrobial peritoneal inoculum by antibiotic therapy, or by translocation of bowel flora. Translocation may be promoted by intestinal ischemia, endotoxemia, malnutrition, or proliferation of resistant bowel flora by antibiotic pressure.

Tertiary Peritonitis:

- Tertiary peritonitis represents the persistence or recurrence of peritoneal infection following apparently adequate therapy, often without the original visceral organ pathology.
- Tertiary peritonitis develops more frequently in patients with significant preexisting co morbid conditions.
- Patients who are immunocompromised.
- Resistant and unusual organisms (e.g. Enterococcus, Candida, Staphylococcus, Enterobacter, and Psuedomonas species) are found in a significant proportion of cases of tertiary peritonitis.

II. LOCAL AND DIFFUSE PERITONITIS

Local peritonitis is bound up intimately with the causative lesion, and the initial symptoms and signs are those of that lesion. When the peritoneum becomes inflamed the temperature, and especially the pulse rate, rise. The pain increases and usually there is associated vomiting.

The most important sign is guarding and rigidity of the abdominal wall over the area of the abdomen which is involved, with a positive "release" sign. If inflammation arises under the diaphragm, shoulder tip ("phrenic") pain may be felt. In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight, deep tenderness of one or both lower quadrants alone being present, but a rectal or vaginal examination reveals tenderness, often exquisite, of the pelvic peritoneum. With appropriate treatment, localized peritonitis usually resolves. In about 20% of cases an abscess follows. Infrequently, local peritonitis becomes diffuse or localized (abscess). Peritoneal abscess describes the formation of an infected fluid collection encapsulated by fibrinous exudate, omentum, and/or adjacent visceral organs. The overwhelming majority of abscesses occurs subsequent to SP. Approximately half of patients develop a simple abscess without loculation, whereas the other half of patients develop complex abscesses secondary to fibrinous septation and organization of the abscess material. Abscess formation occurs most frequently in the subhepatic area, the pelvis, and the paracolic gutters, but it may also occur in the perisplenic area, the lesser sac, and between small bowel loops and their mesentery.

The incidence of abscess formation after abdominal surgery is less than 1–2%, even when the operation is performed for an acute inflammatory process. The risk of abscess increases to 10–30% in cases of preoperative perforation of the hollow viscus.

Diffuse (syn. generalized) peritonitis has 3 phases (Simonyan's classification, 1971):

Reactive (Initial phase). There is severe pain which is made worse by moving or breathing. It is first experienced at the site of the original lesion, and spreads outwards from this point. Vomiting may occur. The patient usually lies still. Tenderness and rigidity on palpation are typically found when the peritonitis affects the anterior abdominal wall. Abdominal tenderness and rigidity are diminished or absent if the anterior wall is unaffected, as in pelvic peritonitis or, rarely, peritonitis in the lesser sac. Patients with pelvic peritonitis may complain of urinary symptoms; they are tender

on rectal or vaginal examination. Infrequent bowel sounds may still be heard for a short time, but they cease with the onset of paralytic ileus. The pulse rises progressively, but if the peritoneum is deluged with irritant fluid, there is a sudden rise. The temperature changes are variable and can be subnormal.

Toxic (Intermediate phase). Peritonitis may resolve, so that the pulse slows, the pain and tenderness diminish, leaving a silent, soft abdomen. (These are features that can easily mislead the observer.) The condition may localize, producing one or more abscesses, with overlying swelling and tenderness.

Terminal phase. If resolution or localization has not occurred, the abdomen remains silent, and increasingly distends. Circulatory failure ensues, with cold, clammy extremities, sunken eyes, dry tongue, thready (irregular) pulse, drawn and anxious face (facies Hippocratica). The patient finally lapses into unconsciousness. With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice.

III. MODERN CLASSIFICATION: peritonitis without and with abdominal sepsis.

Sepsis represents the systemic response to infection, characterized by an exaggerated inflammatory response (SIRS) and widespread tissue injury involving otherwise normal tissue that is unregulated and self-sustaining. In 1992 sepsis was defined as "the host response to infection".

The principles of classification (Consensus Conference ACCP/SCCM (1991):

- Consistent definition for sepsis.
- Tools for improving detection of sepsis.
- Standardization of nomenclature to advance sepsis research

Systemic Inflammatory Response Syndrome (SIRS):

Clinical syndrome defined by the presence of two or more of the following:

- **Fever** > 380 °C or hypothermia < 360 °C.
- **Leukocytosis** > 12 000 or leucopenia < 4 000 or > 10% bands.
- **Tachycardia** > 90 beats per minute.
- **Tachypnea** RR > 20 breaths per minute

SEPSIS – SIRS DUE TO A SUSPECTED INFECTION.

SEVERE SEPSIS – SEPSIS WITH ACUTE ORGAN DYSFUNCTION OR MULTIPLE ORGAN DYSFUNCTIONS

Organ dysfunction:

- Acute cardiovascular failure.
- Acute respiratory failure.
- Acute renal failure.
- Hepatic failure
- Disseminated intravascular coagulopathy.
- Encephalopathy.

SEPTIC SHOCK – SEVERE SEPSIS WITH HYPOTENSION (SYSTOLIC BLOOD PRESSURE < 90 mmhg) UNRESPONSIVE TO FLUIDS.

Pathophysiology of Sepsis

- Sepsis can lead to widespread inflammation and blood clotting.
- Inflammation may result in redness, heat, swelling, pain, and organ failure.
- Blood clotting during sepsis causes reduced blood flow to limbs and vital organs, and can lead to organ failure or tissue damage.

In simple terms sepsis can be viewed as an imbalance of inflammation, coagulation, and fibrinolysis. In normal patients homeostasis is maintained when these are balanced. During a normal response to bacteria in the blood the immune system releases inflammatory mediators to promote recovery of the tissue.

These mediators are known as:

- Tumor necrosis factor (TNF).
- Interleukins (IL).
- Prostaglandins.
- Platelet Activating Factor (PAF) and other.

The release of the inflammatory mediators starts the coagulation cascade leading to the development of a clot. To maintain this clot, inhibitors are released to suppress fibrinolysis or breakdown. This is necessary to have time for the body to destroy the bacteria before the clot is gone.

Once the bacteria or antigen is isolated, the pro-inflammatory mediators attract neutrophils or WBCs which attack the antigen and try to engulf it. To prevent the response from damaging normal tissue, anti-inflammatory mediators are released including transforming growth factors and interleukins (IL-4). This balance of inflammatory and anti-inflammatory mediators restricts the inflammation response to the local site of infection. When the body is unable to maintain the appropriate balance, the immune response is no longer local but becomes systemic. Inflammation and altered clotting quickly spread through the body. The person with the infection which was once localized could become critically ill if this process is not corrected.

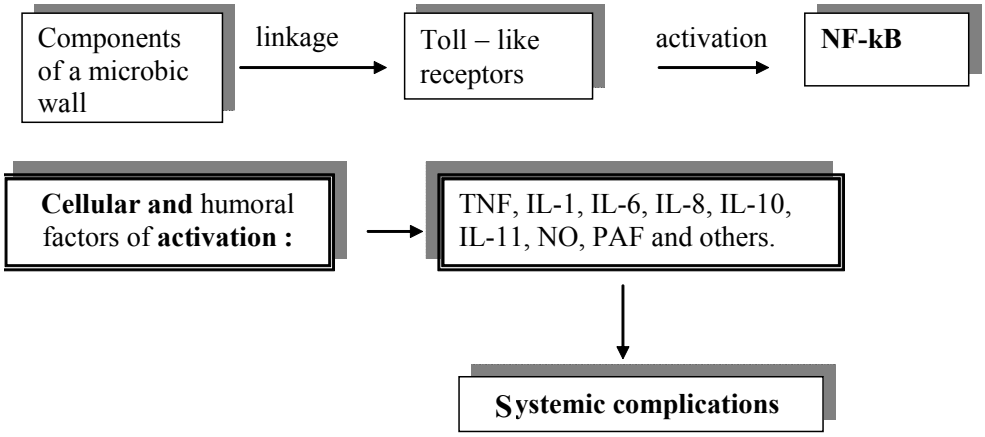


Figure 7.4. *Pathophysiology of systemic complications due to sepsis*

A number of researches testifies (Doughty L.A. et al., 2006; Foulds S. et al., 2001; Ueno C., et al., 2005; Veres B. et al., 2003) that Toll-like receptors are the key structures, distinguishing various substances of the microbic origin, starting an expression of factors of nonspecific resistance through activation of the nuclear factor kappa- (NF-kB), occurrence liberated of various stages. Pro-and anti-inflammatory mediators, and also various humoral factors which, along with process of neutralization of the alien agent, damage own factors with systemic disease consequences. The basic stages of these processes look like as follows: **a gene → activation → systemic complications** (Fig. 7.4):

Bacterial translocation. In recent years it has been increasingly recognized that the gastrointestinal tract has functions other than simply the digestion and excretion of foodstuffs. The gut is also a metabolic and immunological organ that serves as a barrier against living organisms and antigens within its lumen. This role is termed "gut barrier function". The fact that luminal contents in the caecum have a bacterial concentration of the order of 10^{12} organisms per millilitre of faeces,1 whilst portal blood and mesenteric lymph nodes are usually sterile, dramatically illustrates the efficacy of this barrier function.

The idea that the alimentary tract, teeming with its own bacterial flora, could represent a source of sepsis under certain conditions has interested clinicians for many years. This theory, usually referred to as the "gut origin of sepsis" hypothesis, is not new. In the late nineteenth century, the idea developed that peritonitis could result from the passage of bacteria from organs adjacent to the peritoneal cavity. In Germany this was referred to as *durchwanderungs-peritonitis*, literally translated as "wandering through peritonitis".

The term "bacterial translocation" is used to describe the passage of viable resident bacteria from the gastrointestinal tract to normally sterile tissues such as the mesenteric lymph nodes and other internal organs (Berg R.D., Garlington A.W., 1979). The term also applies to the passage of inert particles and other macromolecules, such as lipopolysaccharide endotoxin, across the intestinal mucosal barrier.

There is no doubt that bacterial translocation occurs in humans. It has a prevalence of about 15% in elective surgical patients and occurs more frequently in patients with intestinal obstruction and those who are immunocompromised. There is good evidence to show that translocation is associated with an increased incidence of septic complications, but not with mortality. Many studies have established an association between gastrointestinal microflora and nosocomial infection, supporting the concept of the gut as a reservoir of bacteria and endotoxin. However, the evidence that the mechanism of this association between enteric organisms and subsequent sepsis is bacterial translocation remains, at least in humans, largely circumstantial. This may reflect the methodological and ethical difficulties involved in obtaining samples of portal venous blood or mesenteric lymph nodes, which are necessary for unequivocal confirmation of the occurrence of translocation. To date there has been only one clinical study providing compelling evidence for a mechanistic link between translocation and the development of late sepsis. This study demonstrated that septic complications in 448 surgical patients were significantly more prevalent in those who showed bacterial translocation when compared with patients with no organisms in their mesenteric lymph nodes collected at laparotomy (O'Boyle C.J. et al., 1998). Furthermore, the spectrum of organisms responsible for septic morbidity was similar to those observed in the mesenteric lymph nodes, many being from enteric strains. These data strongly support the gut origin of sepsis hypothesis.

Whilst it is tempting to assume that any bacteria or endotoxin passing through the intestinal barrier might cause septic complications in the host, there is growing evidence to suggest that translocation may in fact be a normal phenomenon. It is possible that translocation occurs so that the alimentary tract can be exposed to and sample antigens within the lumen such that the gut can mount a *controlled* local immune response helping to keep these antigens away from the internal milieu; this process is known as "oral tolerance". It is only when the host's immune defences are overwhelmed

that septic complications arise. Other authors go further and depict translocation not as the initiator of septic complications, but as a result of other insults which have triggered off a systemic inflammatory response, one of the effects of which is manifested as deterioration or breakdown of gut barrier function.

Bacterial translocation has been shown to occur in various patient populations. As already stated, it occurs in patients undergoing elective abdominal surgery, organ donors and those with intestinal obstruction, colorectal cancer, ischaemia-reperfusion injury shock, pancreatitis and peritonitis. Many authors suggest an increased prevalence in patients with obstructive jaundice, those receiving parenteral nutrition and the malnourished, but the evidence for this is limited. Interestingly, translocation, assessed by endotoxin or bacterial culture of portal or systemic blood, has only rarely been demonstrated after trauma.

Host defence mechanisms directed against invasion by microbes comprise many factors, such as gastric acid, pancreatic enzymes, bile, mucus, bowel motility, the antigen-specific local immune system known as gut-associated lymphoid tissue (GALT) and, arguably most important, the epithelial cell barrier. The intestinal epithelium is a polarized monolayer of enterocytes covered with mucus, a glycocalyx brush border and secretory immunoglobulin A (sIgA). It represents a specialized anatomical barrier separating the luminal contents of the bowel from the internal milieu. Physical breaches in this barrier, which can occur following ulceration, may predispose to translocation. A rapid rate of cellular turnover, specialization and migration helps the intestinal mucosa to maintain physical integrity. Translocation may occur via transcellular or paracellular routes, or a combination of the two. Transcellular translocation is under the control of specific membrane pumps and channels, whereas paracellular translocation is permitted (at least theoretically) by breakdown of tight junctions.

Transcellular migration has been shown to occur in rats where various organisms, including *Escherichia coli* and *Proteus mirabilis*, were visualized within intact enterocytes. There is in vitro evidence that opening up the gaps between enterocytes by loosening the intercellular tight junctions may increase bacterial translocation. Tight junctions can adjust their degree of 'leakiness' to meet physiological needs, and are also susceptible to numerous pathological stimuli that may change the intestinal permeability. However, it is important to emphasize that there are, as yet, no data from animal or human studies that directly confirm a causative relationship between changes in permeability and bacterial translocation. Similarly, there is no evidence to confirm that gross changes in villus morphology are causally related to increased rates of translocation.

The human gastrointestinal tract contains a wide variety of aerobic and anaerobic bacteria. Absolute bacterial counts vary along the length of the bowel, increasing from a concentration of 10^8 organisms/ml in the region of the distal ileum to ~ 10^{12} organisms/ml beyond the ileocaecal valve. The upper gut and stomach are usually sterile or are sparsely populated with relatively avirulent bacteria. It is a remarkable testimony to the efficiency of the intestinal barrier that large populations of indigenous bacteria reside in the healthy lower gut without causing harm. This indigenous microflora exerts an important influence in preventing colonization with exogenous pathogens, so-called colonization resistance. Many factors have been implicated in the regulation of the different populations of microflora. These include the creation of 'microclimates' whereby facultative bacteria, for example, utilize oxygen, thereby ensuring a suitable environment for obligate anaerobes. This has prompted suggestions

that obligate anaerobic bacteria may be the principal inhibitors of translocation of *E.coli* and other potentially pathogenic bacteria. Disruption of the normal flora may predispose to a breakdown in colonization resistance. This may occur, for example, with antibiotic therapy and there is evidence that substrate utilization by different organisms will also influence other bacterial concentrations.

Critical illness is often associated with significant proximal gut overgrowth of enteric organisms, which may contribute to nosocomial infection. The similarity in the spectrum of organisms identified in septic foci and those cultured from gastric aspirates suggests that the infecting organisms are of gut origin. This infers, but does not prove, a role for bacterial translocation. J.C. Marshall et al. (1993) showed that >90% of their intensive care unit (ICU) patients with infection had at least one episode of infection with an organism that was simultaneously present in the upper gastrointestinal tract. In our own studies we have consistently found organisms in mesenteric lymph nodes that are similar to those identified in septic foci as overgrowth in the upper gut. All these data point to the importance of the gastrointestinal microflora in maintaining health and that disruption is associated with illness. However, it should be emphasized that the influence of enteric bacteria is not simply related to population density of specific bacteria. Gram-negative facultative anaerobes, particularly *E.coli*, are consistently shown to be the most common organisms identified in mesenteric lymph nodes. Obligate anaerobes are known to translocate but rarely do so despite their luminal concentrations. Thus it appears that absolute intraluminal population density is not a major determinant of translocation and therefore it is unlikely that rates of translocation will be influenced by alterations in gut permeability.

Under normal circumstances, translocating bacteria should be phagocytosed before reaching mesenteric lymph nodes or lymphatic vessels. The presumption is that if the host is immunocompromised the normal defence mechanisms fail, permitting egress and survival of these bacteria at distant extra-intestinal sites. The inference is that with increasing severity of illness, bacterial translocation occurs because of the inability of the host to deal adequately with the numbers of bacteria present. Much experimental evidence exists to support this concept. Bacterial translocation is more common in athymic mice than in control animals, suggesting a specific role for T-cell-mediated immunity in inhibiting translocation. Immunosuppressant agents and leukaemia have been associated with increased translocation to mesenteric lymph nodes in rodents, and leukaemia has been associated with increased translocation to blood in humans. The possibility that bacterial translocation may not be an all-or-none phenomenon has been previously suggested. There is experimental evidence in human studies attesting to the invariable presence of *E.coli* β-galactosidase in the cytoplasm of mesenteric lymph nodes but positive cultures in only 5% of the same patients. This suggests translocation and subsequent control in the mesenteric nodes. The association between multi-organism colonies and an increasing incidence of postoperative sepsis, as shown in our own studies, is compatible with this speculative mechanism of translocation and is probably a manifestation of immunosuppression. Furthermore, it is of some interest to note that our results show that the incidence of bacterial translocation was greatest in patients aged >70 years, those undergoing urgent surgery and those with intestinal obstruction, all of whom might be expected to have a degree of impaired immunity. Bacterial translocation may thus serve as a promoter of septic morbidity, but not actually be the initiator.

There is now much circumstantial evidence to support the view that translocation is associated with an increase in septic morbidity. To date, however, there has been only one study attempting to prove a mechanistic link between translocation and the development of late sepsis. *Escherichia coli* was the most common organism to be isolated from the lymph node specimens (48%) and septic foci (53%). Fungal translocation did not occur. An identical genus was identified in the nasogastric aspirate and septic focus in 30% of patients, in the nasogastric aspirate and the lymph node in 31%, and in the lymph node and a postoperative septic focus in 45%. The authors concluded that proximal gut colonization is associated with both increased bacterial translocation and septic morbidity. The commonality of organisms identified was considered as compelling evidence in support of the gut origin of sepsis hypothesis and confirms that bacterial translocation is associated with an increase in postoperative septic morbidity.

The potential importance of the gut origin of sepsis theory in the causation or perpetuation of multiple organ dysfunction (MOD) has been reviewed elsewhere. The data in support of MOD infections originating from the gut are strong. Epidemiological studies have convincingly demonstrated that the gut is the reservoir for pathogens in late MOD-related infections. In addition, there is much circumstantial evidence. Critically ill patients are often maintained on parenteral nutrition and receive no enteral stimulation for long periods. It has been suggested, although not confirmed, that this may be associated with increased rates of septic morbidity related to bacterial translocation. These patients also frequently receive narcotics (which depress gut motility) together with antacids and broad-spectrum antibiotics, all of which can lead to alterations in gastrointestinal microflora which may in turn predispose to translocation. Gut-specific interventions have been shown to reduce ICU-acquired infection. Thus sucralfate, which maintains gastric acidity, may reduce nosocomial pneumonias. Recent work has shown that preoperative optimization of oxygen delivery with fluid and the splanchnic vasodilator dopexamine in surgical patients can also significantly reduce postoperative mortality, which may be a reflection of improved gut function.

Although these data support the view that the gut origin of sepsis hypothesis is valid as an explanation for some episodes of sepsis in the critically ill, are they sufficient to account for the development of systemic inflammatory response syndrome (SIRS) or MOD? There is increasing support for the concept of a "two-hit model" to explain the clinical and metabolic changes that occur in multiple organ failure (MOF). In this first hit, direct cell trauma as a consequence of mechanical, thermal or cytotoxic injury, or cell ischaemia from shock, results in an inflammatory response that is appropriate and potentially advantageous. It would seem unlikely that bacterial translocation has any role in the pathogenesis of these early responses. In some patients, a second hit occurs as a result of repeated surgery, transient bacteraemia or persistent cell damage related to the initial injury. The two-hit theory suggests that the ongoing inflammatory response results in either an excessive compensatory anti-inflammatory response or an imbalance between pro- and anti-inflammatory responses. In other words, an autodestructive inflammation occurs which is the manifestation of severe and sometimes irreversible cell damage induced by cytokine mediators. Bacterial translocation has been suggested as inducing or perpetuating this continuous inflammatory state as endotoxin is known to induce the release of both pro- and anti-inflammatory cytokines.

There is substantial evidence from animal studies to support this theoretical basis of SIRS and MOD. In particular, there is good evidence to suggest that the mechanism behind continued inflammation of the gut is an ischaemia-reperfusion injury. In humans, the counter-current oxygen tension at the tip of the villus is lower than that in arterial blood. As a consequence epithelial cell injury may develop in any situation in which tissue oxygenation is diminished. This injury may permit bacterial translocation, which acts as a perpetuator of the inflammatory response. Evidence for this in human experiments is scant. A. Cabie et al. (1993) reported significantly higher levels of tumour necrosis factor in the portal vein than in the systemic circulation in patients undergoing aortic surgery. R. Shenkar and E. Abraham (1993) described the first induction of cytokine mRNA after haemorrhage into the gut. These data are in accord with the possibility that, on reaching the lamina propria, endotoxin or bacteria encounter macrophages which locally produce cytokines without it being necessary to first transport endotoxin or bacteria via the portal vein to the liver or mesenteric lymph nodes. This would also account for the failure of clinical studies to consistently identify endotoxin or bacteria in patients with SIRS or MOD.

Oxygenation to the villi in humans is dependent on a counter-current exchange mechanism such that oxygen saturation at the tip of the villi is lower than that of arterial blood. Consequently, any reduction in splanchnic blood flow is likely to result in ischaemia-reperfusion damage. Diminished splanchnic blood flow, as seen in hypovolaemic shock and bowel ischaemia, is associated with translocation and septic complications in animal models. The potential importance of splanchnic flow as a determinant of outcome in humans with critical illness is supported by the findings of a recent study which demonstrated that the use of the splanchnic vasodilator dopexamine was associated with a significant reduction in post-operative mortality. Whether this was a result of reduced translocation is unclear. It has been suggested that splanchnic hypoperfusion results in the gut becoming a major site of proinflammatory factor production. The possibility exists that splanchnic hypoperfusion can lead to an ischaemia-reperfusion injury to the gut, with a resultant loss of gut barrier function and an ensuing gut inflammatory response without the need for translocation of microbes as far as the mesenteric lymph nodes or beyond. Once endotoxin or bacteria cross the mucosal barrier, even if trapped within the gut wall, they can trigger an immune response such that the gut becomes a proinflammatory organ, releasing chemokines, cytokines and other proinflammatory intermediates which affect both the local and the systemic immune systems, finally giving rise to SIRS and MOF. E. A. Deitch (2002) has termed this "the three-hit model" of gut inflammation. It comprises hypoperfusion, ischaemia-reperfusion injury and the loss of gut barrier function in association with a markedly increased proinflammatory response. In this model it is not considered that the gut barrier is simply a barrier to the passage of intraluminal bacteria to distant sites, but rather that bacteria or gut ischaemia, or both, cause the gut-associated lymphoid tissue to generate immuno-inflammatory factors that contribute to distant organ injury.

Conclusion. The gut origin of sepsis hypothesis is an attractive and simple concept that presupposes that bacteria cross the intestinal barrier and cause sepsis at distant sites. There is now good evidence from human subjects to support this theory, which confirms that translocation predisposes to an increase in septic morbidity. Bacterial and endotoxin translocation probably also occurs to a limited extent on a

regular basis in healthy individuals. In this situation translocation serves to provide an antigenic stimulus, but normal barrier function is preserved and morbidity does not ensue. Only if normal mechanisms of defence are overwhelmed does translocation occur. Therefore there seems little doubt that the phenomenon of bacterial translocation is associated with septic morbidity. However, it is very important to place this in the context of the surgical or non-surgical patient. Septic morbidity is multifactorial. Bacterial translocation is likely to play an aetiological part in only a minority of patients. Other factors are equally important in the causation of postoperative septic morbidity. These may relate to colonization of the upper gut, aspiration or the presence of indwelling foreign material such as catheters and lines, and cannot be directly attributed to the phenomenon of translocation. Similarly, whilst it is almost certainly true that translocation is a factor in the causation of SIRS and/or MOF in those patients who survive the first insult of their trauma, it is difficult in the present state of our knowledge to define the relative importance of this compared with other factors. In particular, it is disappointing to record that the attractive simplicity of the gut origin of sepsis hypothesis, whilst confirmed in certain patients has not yet been associated with any proven beneficial therapeutic measures in patient care. In particular, attempts at selective gut decontamination, the use of pre- or probiotics, alterations in preoperative antibiotic prophylaxis and mechanical bowel preparation have not translated into benefits to patient care with particular regard to septic morbidity. Bacterial translocation remains a fascinating epiphenomenon to those with an interest in the metabolic care of the critically ill surgical patient. It is not, as yet, a reason to change clinical practice.

7.4 CLINICAL FEATURES

There are several diseases of the abdominal cavity unified under the common designation of the patient's death. The picture of acute abdomen is determined by the following signs which occur in aggregate, as well as separately.

Initially, the pain may be dull and poorly localized (visceral peritoneum) and often progresses to steady, severe, and more localized pain (parietal peritoneum). If the underlying process is not contained, the pain becomes diffuse. In certain disease entities (gastric perforation, severe acute pancreatitis, intestinal ischemia), the abdominal pain may be generalized from the beginning. Anorexia and nausea are frequent symptoms and may precede the development of abdominal pain. Vomiting may be due to underlying visceral organ pathology (obstruction) or be secondary to peritoneal irritation.

On physical examination, patients with peritonitis generally appear unwell and in acute distress. Many of them have a temperature that exceeds 38° C, although patients with severe sepsis may become hypothermic. Tachycardia is caused by the release of inflammatory mediators, intravascular hypovolemia from anorexia vomiting and fever, and third-space losses into the peritoneal cavity. With progressive dehydration, patients may become hypotensive, as well as oliguric or anuric; with severe peritonitis, they may present in overt septic shock.

On abdominal examination, almost all patients demonstrate tenderness to palpation. (When examining the abdomen of a patient with peritonitis, the patient should be supine. A roll or pillows underneath the patient's knees may allow for better relaxation of the abdominal wall). In most patients (even with generalized peritonitis and severe diffuse

abdominal pain), the point of maximal tenderness or referred rebound tenderness roughly overlies the pathologic process (i.e., the site of maximal peritoneal irritation).

Most patients demonstrate increased abdominal wall rigidity. The increase in abdominal wall muscular tone may be voluntary in response to or in anticipation of the abdominal examination or involuntary because of the peritoneal irritation. Patients with severe peritonitis often avoid all motion and keep their hips flexed to relieve the abdominal wall tension. The abdomen is often distended, with hypoactive-to-absent bowel sounds. This finding reflects a generalized ileus and may not be present if the infection is well localized. Occasionally, the abdominal examination reveals an inflammatory mass.

Rectal examination often elicits increased abdominal pain, particularly with inflammation of the pelvic organs, but rarely indicates a specific diagnosis. A tender inflammatory mass toward the right may indicate appendicitis, and anterior fullness and fluctuation may indicate a cul de sac abscess. In female patients, vaginal and bimanual examination findings may be consistent with pelvic inflammatory disease (endometritis, salpingooophoritis, tuboovarian abscess), but exam findings are often difficult to interpret in severe peritonitis.

7.5. EXAMINATIONS

Radiographs. Plain films of the abdomen (e.g., supine, upright, and lateral decubitus positions) are often the first imaging studies obtained in patients presenting with peritonitis. Their value in reaching a specific diagnosis is limited.

Free air is present in most cases of anterior gastric and duodenal perforation but is much less frequent with perforations of the small bowel and colon and is unusual with appendiceal perforation. Upright films are useful for identifying free air under the diaphragm (most often on the right) as an indication of a perforated viscus. Remember that the presence of free air is not mandatory with visceral perforation and that small amounts of free air are missed easily on plain films (Fig. 7.5–7.7).

Ultrasonography. Abdominal ultrasonography may be helpful in the evaluation of right upper quadrant (e.g., perihepatic abscess, cholecystitis, biloma, pancreatitis, pancreatic pseudocyst), right lower quadrant, and pelvic pathology (e.g., appendicitis, tubo-ovarian abscess, Douglas pouch abscess), but the examination is sometimes limited because of patient's discomfort, abdominal distension, and bowel gas interference.

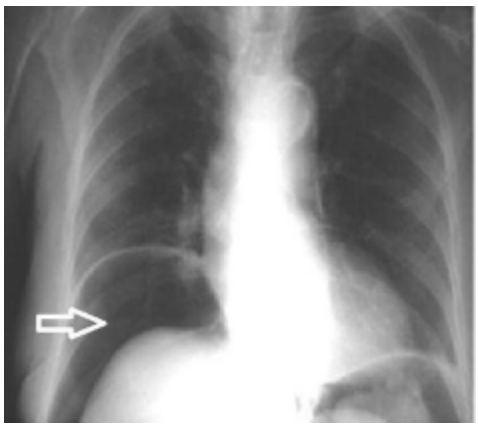


Figure 7.5. *Gas under diaphragm (arrow)*

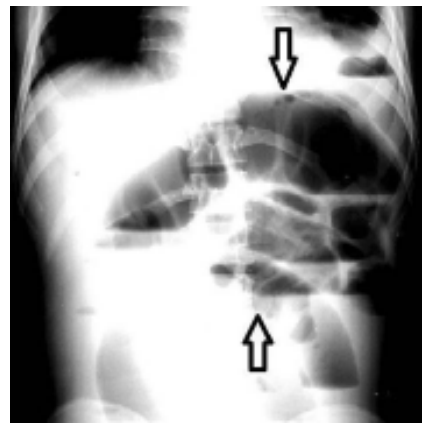


Figure 7.6. *Multiple air fluid levels (arrows)*

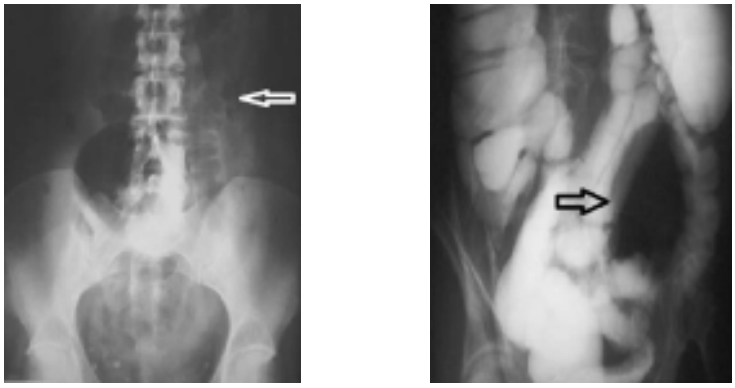


Figure 7.7. *Sigmoid diverticulitis: gas filled diverticulum (arrow)*

Ultrasonography may detect increased amounts of peritoneal fluid (ascites), but its ability to detect quantities of less than 100 mL is limited. The central (perimesenteric) peritoneal cavity is not visualized well with transabdominal ultrasonography. Examination from the flank or back may improve the diagnostic yield, and providing the ultrasonographer with specific information of the patient's condition and the suspected diagnosis before the examination is important. With an experienced ultrasonographer, a diagnostic accuracy of greater than 85% has been reported in several series.

Ultrasonographically guided aspiration and placement of drains has evolved into a valuable tool in the diagnosis and treatment of abdominal fluid collections **CT scanning** (Fig. 7.8).

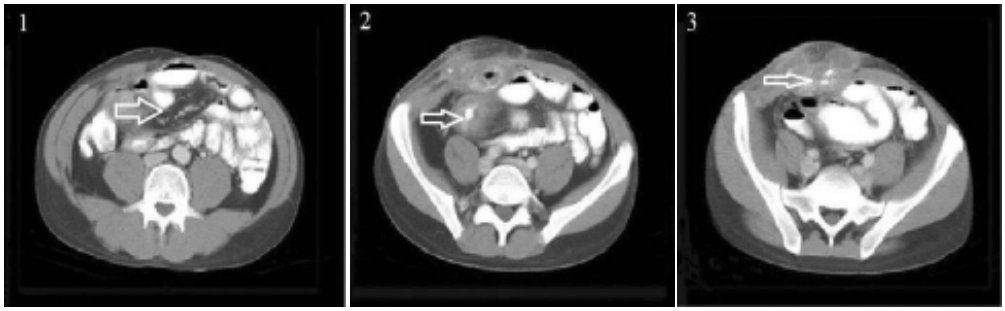


Figure 7.8. *A 35-year-old man with a history of Crohn disease presented with pain and swelling in the right abdomen. In figure 1, a thickened loop of terminal ileum is evident adherent to the right anterior abdominal wall (arrow). In figure 2, the right anterior abdominal wall is markedly thickened and edematous, with adjacent inflamed terminal ileum (arrow). In figure 3, a right lower quadrant abdominal wall abscess and enteric fistula are observed and confirmed by the presence of enteral contrast in the abdominal wall (arrow)*

If the diagnosis of peritonitis is made clinically, a CT scan is not necessary and generally delays surgical intervention without offering clinical advantage.

CT scans of the abdomen and pelvis remain the diagnostic study of choice for peritoneal abscess and related visceral pathology. CT scanning is indicated in all cases in which the diagnosis cannot be established on clinical grounds and findings on abdominal plain films. Whenever possible, the CT scan should be performed with enteral and intravenous contrast. CT scans can detect small quantities of fluid, areas of inflammation, and other GI tract pathology, with sensitivities that approach 100%.

CT has become invaluable in evaluating patients suspected of having an intraabdominal infection CT- or ultrasonography-guided aspiration of suspected intraabdominal abscesses has also become standard in evaluating and managing selected patients. Intraabdominal infection encompasses many different pathological and clinical entities that are associated with widely varying mortality rates that rangt from zero to 60%.

Magnetic resonance imaging (MRI). MRI is an emerging imaging modality for the diagnosis of suspected intra-abdominal abscesses. Abdominal abscesses demonstrate decreased signal intensity on T1-weighted images and homogeneous or heterogeneous increased signal intensity on T2-weighted images; abscesses are observed best on gadolinium-enhanced, T1-weighted, fat-suppressed images as well-defined fluid collections with rim enhancement.

Limited availability and high cost, as well as the need for MRI-compatible patient support equipment and the length of the examination currently limit its usefulness as a diagnostic tool in acute peritoneal infections, particularly for patients who are critically ill.

Laboratory Studies. CBC (complete blood count) with differentials – Most patients will have leukocytosis (>11 000 cells/μL), with a shift to the immature forms on the differential cell count. Patients with severe sepsis, who are immunocompromised, or who have certain types of infections (e.g., fungal, cytomegaloviral) may demonstrate absence of leukocytosis or leucopenia. In cases of suspected SBP, hypersplenism may reduce the polymorphonuclear leukocyte count.

Examinations:

- Blood chemistry may reveal dehydration and acidosis.
- PT (prothrombin time), PTT (partial thromboplastin time).
- Liver function tests if clinically indicated.
- Amylase and lipase if pancreatitis is suspected.
- Urinalysis (UA) to rule out urinary tract diseases (e.g., pyelonephritis, renal stone disease); however, patients with lower abdominal and pelvic infections often demonstrate WBCs in the urine and microhematuria.
- Stool sample in patients with diarrhea, evaluate a stool sample – employing a *Clostridium difficile* toxin assay, a WBC count, and a specific culture (i.*e., Salmonella, Shigella,* cytomegalovirus [CMV]) – if the patient's history suggests infectious enterocolitis.
- Aerobic and anaerobic blood cultures.
- Peritoneal fluid (i.e., paracentesis, aspiration of abdominal fluid collections, intraoperative peritoneal fluid cultures).

Diagnostic peritoneal lavage (DPL) may be helpful in patients who do not have conclusive signs on physical examination or who cannot provide an adequate history. A DPL with more than 500 leukocytes/mL is considered positive and suggests peritonitis.

- Evaluate the sample for pH, glucose, protein, lactate dehydrogenase (LDH), cell count, Gram stain, and aerobic and anaerobic cultures.
- Include analysis if pancreatitis or pancreatic leak is suspected.
- Test for bilirubin when a biliary leak is suspected and for fluid creatinine level when a urinary leak is suspected.
- Compare the peritoneal levels to the respective serum levels.

Scoring

The prognosis of peritonitis and intra-abdominal sepsis, particularly when multiorgan dysfunction develops-remains poor despite improvements in diagnosis and surgical and medical management of this condition. Early and objective classification of the severity of peritonitis may help in selecting patients for aggressive surgical approach. Several scoring systems have been developed for this purpose, such as the Acute Physiology and Chronic Health Evaluation (APACHE) II score that considers 12 physiological variables, Simplified Acute Physiology Score (SAPS), Sepsis Severity Score (SSS), and Mannheim Peritonitis Index (MPI) (Table 7.3).

Table 7.3 – Mannheim Peritonitis Index

Risk factor	Weightage, if any
Age >50 years	5
Female gender	5
Organ failure*	7
Malignancy	4
Preoperative duration of peritonitis >24 h	4
Origin of sepsis not colonic	4
Exudates	
• Clear	0
• Cloudy, purulent	6
• Fecal	12
Maximum	47

*Definitions of organ failure: *Kidney:* creatinine >177 mol/L, urea >167 mol/L, oliguria <20 mL/h; *Lung*: pO_2<50 mmHg, pCO^2 >50 mmHg; *Shock*: hypodynamic or hyperdynamic; *Intestinal* obstruction (only if profound); *Paralysis* >24 h or complete mechanical ileus

Accuracy of the MPI was comparable or slightly superior to that of other sepsis classification systems, including APACHE II. In previous studies, patients with a score less than 21 had mortality rate ranging from 0%–2,3%, and patients with MPI more than 29 had the highest mortality rate, up to 100% in some studies; prognosis with MPI between 21 and 29 was approximately 65%. A score of 26 had sensitivity of 86% and specificity of 74% in predicting death (Fugger R. et. al., 1988).

7.6. TREATMENT

The general principles guiding the treatment of intra-abdominal infections are 4-fold:

- to control the infectious or irritate source (to eliminate the cause);
- to eliminate bacteria and toxins;
- to maintain organ system function;
- to control the inflammatory process.

Management and prevention of primary peritonitis. The management of primary peritonitis, an essentially "nonsurgical", antibiotic-treated disease is referred to elsewhere. The *sine qua non* of success is timely surgical intervention to stop delivery of bacteria and adjuvants into the peritoneal cavity. All other measures are of little use if the operation does not successfully abort the infective source and quantitatively reduce the inoculum of micro-organisms and adjuvants of infection so that they can be effectively handled by the patient's defenses, supported by antibiotic

therapy. As yet, effective "antimediator" therapy to modulate the local and systemic inflammatory repercussions of peritonitis is not available. Consequently, intensive measures to support tissue oxygenation and maintain organ function are necessary while awaiting recovery brought on trough antibiotic and surgical therapy.

Because the gram sum is frequently negative in primary bacterial peritonitis, the initial choice of antimicrobial drag is often empirical, based on the most likely pathogens. Some of the third-generation cephalosporin antibiotics have been demonstrated to be as efficacious as the combination of ampicillm plus an aminoglycoside in primary bacterial peritonitis They also eliminate the risk of nephrotoxicity, which is sufficiently frequent in this group of patients to warrant avoidance of aminoglycosides if an equally effective alternative antimicrobial regimen can be used.

Other antimicrobial agents such as the broad-spectrum penicillins (e.g. mezlocillin, tikarcillin, and piperacillin), carbapenems (e.g. imipenem) and β-lactam antibiotic β-lactamase-inhibitor combinations (e.g. ticarcilin-clavulanat and ampicillm-sulbactam) are potential alternatives. If peritonitis develops during hospitalization, the therapeutic regimen, such as administration of an aminoglycoside antibiotic and an antipseudomonal penicillin or cephalosporin in combination, should also be active against.

For those situations in which the pram stain is suggestive of a *Bacteroides* species or polymicrobial peritonitis is evident, examination for additional pathological conditions. Antimicrobial therapy is usually continued for 10–14 days if improvement is noted, but short-course therapy for 5 days has been shown to be as efficacious as the longer course in some patients. Administration of intraperitoneal antimicrobials is not пecesary.

Treatment of primary peritonitis is successful in more than one-half of cirrhotic patients, but because of the underlying liver condition, the overall mortality has been reported as high as 95% in some series. Those patients with the poorest prognosis were found to have renal insufficiency, hypothermia, hyperbilirubinemia, and hypoalbuminemia.

Treatment of peritonitis caused by gram-positive organisms, as well a of early infections, has been more frequently successful than treatment of gram-negative or late infections. In nephrotic patients with gram-positive infections or in patients who do not have a preterminal underlying illness, the survival rate is >90%.

Given the common occurrence and high mortality of primary peritonitis in the setting of cirrhosis with ascites, prevention is a desirable strategy. This is particularly true for patients who are awaiting liver transplantation. Selective decontimination of the gut with oral norfloxacin (400 mg daily) has been shown to reduce the incidence of spontaneous bacterial peritonitis. Norfloxacin has the disadvantage of selecting for gram-positive organisms, including *S. aureus*, and quinolone-resistant gram-negative organisms. More recently, trimethopim-sulfamcthoxazole (double-strength, given once daily for 5 days each week) has been shown to reduce the incidence of peritonitis and be well tolerated. A survival-rate advantage has not been demonstrated for any of these preventive regimens.

Antibiotics that are known to be effective against the full spectrum of aerobic and anaerobic gastrointestinal bacteria should be started as soon as possible, as the actual organism may not be known. Antibiotics that are effective against *E. coli* are mostly used. These are cephalosporins, aminoglycosides, cefamycin and

chloramphenicol, tetracycline and ampicillin are also active against many coliforms. The drugs effective against bacteroids are clindamycin, chloramphenicol, metronidazole and the newer cephalosporins.

Medical management of secondary peritonitis includes use of antimicrobial therapy and supportive measures to maintain vital functions, e.g. to improve or maintain circulation, nutrition, and oxygenation to vital organs. Antimicrobial agents must penetrate to the site of infection in concentrations that are sufficient to overcome the effects of high bacterial density, metabolic inactivity and slow growth rate of the bacterial inoculum, low pH, low redox potential, necrotic tissue, and bacterial products that may lower the drugs' activity. For example, aminoglycosides and clindamycin are less active at acid pH values, aminoglycosides are less active at low redox potentials, and β-lactam drugs are less active against high bacterial densities.

Early clinical trials have established the therapeutic regimen of clindamycin plus aminoglycoside as the "gold standard". Aminoglycosides are frequently underdosed because of fear of nephrotoxicity or because of underestimation of the expanded volume of distribution in critically ill patients with intraabdominal sepsis. Once-daily aminoglycoside therapy – in which the total daily dose is given to patients with normal renal function as a single dose every 24 hours rather than as multiple smaller, divided doses – obviates these dosing problems, because both the bactericidal activity and postantibiotic effect of aminoglycosides are concentration-dependent, while their nephrotoxicity is lime-dependent. However, too few severely ill patients with intraabdominal sepsis have been studied to allow recommendation of the general use of single daily doses of these drugs.

Regimens in which a third-generation cephalosporin is substituted for the aminoglycoside or metronidazole is substituted for clindamycin compare favorably to the "gold standard". Resistance, however, emerges readily under selective pressure of antimicrobial therapy with third-generation cephalosporins among certain gram-negalive bacilli that produce inducible β-lactamases, such as *Enlerobacter, Serratia, Ciirobacler, Morgonella*, and *Acinctohacter* species and *Ps. aeruginosa*. These organisms have a high spontaneous mutation rate for constitutive production of large amounts of these β-lactamases that confer resistance to all lactam drugs, except the carbapenems (imipenem and meropenem) and cefepime, and are poorly antagonized by sulbactam, clavulanic acid, and tazobactam.

Palients likely to be infected with these organisms – e.g., those having prolonged hospital stays, prior antibiotic treatment, postoperative peritonitis, or tertiary peritonitis – are best treated with a regimen that includes imipenem, meropenem, cefepime, a fluoroquinolone, or an aminoglycoside. Resistance among isolates of *B. fragilis* to metronidazole is rare, while resistance to clindamycin is now unacceptably high in many centers. Furthermore, the relative incidence of *Clostridium difficile* associated diarrhea and colonization was found to be less following use of metronidazole than thai following use of clindamycin in a retrospective study.

Microaerophilic gram-positive cocci, which are frequent co-palhogens in polymicrobial anaerobic infection, are resistant to metronidazole, unlike clindamycin, so if metronidazole is used it should be combined with an agent active against these pathogens, such as a β-lactam antibiotic other than aztreonam. Aztreonam, fluoroquinolones, and aminoglycosides also lack activity against microaerophilic gram-positive cocci and theoretically may not be optimal agents to combine with

metronidazole, although clinical trials of these combinations have been found to be efficacious in intraabdominal infection.

Treatment against *Enterococcus* or *Candida* species involved in polymicrobial infections is controversial. Identification of either microorganism in blood cultures, as the sole organism within residual or recurrent intraabdominal infection, or as the predominant pathogen on a gram stain of peritoneal exudate is an indication for specific antimicrobial therapy plus adequate surgical debridement. Enterococci have emerged as leading nosocomial pathogens, undoubtedly as a result of their inherent resistance to many commonly used antimicrobial agents and the recent acquisition of resistance to previous standard therapy, i.e. with ampicillin, aminoglycosides, and vancomycin. Only clinically unproven agents to which the strains are susceptible in vitro, such as doxycycline, novobiocin, or quinupristin/ dalfopristin, are available as potential therapeutic agents for infection caused by these multiresistant strains of enterococci.

Administration of amphotericin B is standard therapy for invasive candidal infection, although amphotericin lipid complex is an equally efficacious, less nephrotoxic, but more expensive alternative. Azoles such as fluconazole and itraconozole are also less toxic, although no comparative trials for candidal peritonitis have been performed. *Candida krusei*, *Candida tropicalis,* and *Torulopsis glabrata* are inherently more resistant to azoles, and resistance to azoles among previously susceptible *Candida* species is increasing.

Management of secondary peritonitis

Preoperative critical care:

- Fluid resuscitation.
- Administration of antibiotics and oxygen.
- Nasogastric intubation.
- Urinary catheterization.
- Monitoring of vital sign and biochemical and hemodynamic data.

Operative management. Operation is mainly aimed at to correct the underlying cause (eliminate the cause). Every attempt should be made to perform the operation as soon as possible.

The incision of choice is midline vertical incision, though some surgeons prefer right paramedian incision or even transverse incision for better healing of the wound. Transverse incisions are particularly better in children below 2 years of age due to more susceptibility of these patients towards postoperative wound dehiscence. In case of localized peritonitis however incision is made over the region involved.

As mentioned above the aim behind surgery is to correct the underlying cause. In case of perforated appendicitis, appendicectomy should be made, only in rare cases however appendicular abscess may be drained only. In case of acute perforated diverticulitis with generalized peritonitis, resection of the involved segment of sigmoid colon should be performed followed by end-to-end suturing or temporary end colostomy. In case of ulcerative colitis, total proctocolectomy with ileostomy is the treatment of choice, though in occasional grave cases only terminal ileostomy may be made. In case of perforated peptic ulcer, either suturing of the peptic ulcer or definitive operation of the peptic ulcer is performed if the patient is fit for that. In case of gangrenous cholecystitis, cholecystectomy should be the treatment of choice, though in occasional grave cases only cholecystostomy may be performed. Only in case of acute pancreatitis, conservative treatment is the treatment of choice.

As soon as the abdomen is opened, odor of the peritoneal fluid is of significance. In case of lower small intestinal perforation the odor is faeculous, whereas in case of upper GI tract perforation above the duodenojejunal flexure, the peritoneal fluid is almost odorless. So a preliminary diagnosis can be made just after opening the abdomen. Odor also depends on the type of infecting organism. In case of *E. coli*, usually fetid odor is detected, whereas in case of infection by anaerobic streptococci or bacteroids or clostridium a penetrating foul odor can be detected. The general rule is that aerobic infections do not produce marked odor, whereas anaerobes do. Surgeon should take bacterial inoculation because it is obligatory.

Although a comprehensive discussion of surgical management of secondary peritonitis is beyond the scope of this review, certain recent trends will be mentioned. Optimal management includes the following: 1) bowel decompression, e.g. by proximal colostomy for perforation, diverticulitis, or colonic carcinoma; 2) closing of traumatic perforations and resection of a diseased, perforated viscus to stop continued peritoneal contamination; and 3) drainage of any purulent collections to reduce the bacterial inoculum and to remove excessive levels of proinflammatory cytokines and other adjuvants, such as fecal matter, food, blood, bile, bullets, or barium.

Intra-abdominal abscesses that present as localised peritonitis. An intra-abdominal abscess may arise following: localisation of peritonitis; gastrointestinal perforation; anastomotic leak; haematogenous spread. They develop in sites of gravitational drainage: pelvis; subhepatic spaces; subphrenic spaces; paracolic gutters.

Clinical features:

- Postoperative abscesses usually present at between 5 and 10 days after surgery.
- Suspect if unexplained persistent or swinging pyrexia.
- May also cause abdominal pain and diarrhea.
- A mass may be present with overlying erythema and tenderness.
- A pelvic abscess may be palpable only on rectal examination.

Management:

- Ultrasound scanning may reveal the diagnosis.
- Contrast-enhanced CT is probably the investigation of choice.
- May delineate a gastrointestinal or anastomotic leak.
- Identifies collection and often allows percutaneous drainage.

1. Operative drainage may be required if:

- Multi-locular abscess.
- No safe route for per cutaneous drainage.
- Recollection after percutaneous drainage.

2. Patients should receive antibiotic therapy guided by organism sensitivities.

Peritonitis caused by colonic perforation due to diverticulitis or colon cancer has been treated with one of three procedures, depending on the particular clinical situation: 1) a three-stage procedure, which involves an initial proximal colostomy to decompress the bowel and divert the fecal stream, followed by resection of the diseased bowel, and finally anastomosis to restore bowel continuity several months later; 2) a two-stage procedure, which involves initial resection and temporary proximal colostomy, with cither a mucous fistula or a blind pouch (Hartmann's procedure), followed by anastomosis; or 3) a one-stage procedure of primary resection and immediate anastomosis.

The three-stage procedure has traditionally been reserved for critically ill, high-risk patients however; the two-stge procedure may be preferred for these patients because it eliminates the source of peritoneal contamination early. The one-stage procedure in the patient with colonic perforation has been questioned because the lumen of the bowel is not cleansed preoperatively and because of the assumed risk of breakdown of the anastomosis in the presence of peritonitis. However, the one-stage primary resection and immediate anastomosis eliminate need for further hospitalization and shorten disability due to a colostomy. Indeed, the one-stage procedure has been shown to be efficacious for the moderate-risk patient with colonic perforation (APACHE II score of ≤15), even in the case of emergency when the bowel has not been cleansed or peritonitis is present.

Intraoperative peritoneal lavage with saline, following drainage of purulent peritoneal exudates, fecal matter, food, and other foreign debris, is standard procedure during laparotomy for peritonitis. However, radical peritoneal debridement of all fibrinous deposits on peritoneal surfaces is no longer thought to be effective. Continuous peritoneal lavage for 41–72 hours postoperatively or until the fluid is clear has been studied, but at present the evidence is insufficient to recommend this procedure. Addition of antibiotics or antiseptics to intraperitoneal lavage fluid has also been shown to have no added benefit.

Peritoneal lavage:

- Peritoneal lavage often used but benefit is unproven.
- Simple swabbing of pus from peritoneal cavity may be of same value.
- Has been suggested that lavage may spread infection or damage peritoneal surface.
- No benefit of adding antibiotics to lavage fluid.
- No benefit of adding Chlorhexidine or Betadine to lavage fluid.
- If used, lavage with large volume of crystalloid solution probably has best outcome.

The ability to predict outcome and stratify severity of disease is important for clinical decision-making as well as ensuring comparability in trials that evaluate different management strategies with surgical protocols or antimicrobial agents. Outcome has been found to be related mainly lo host factor (e.g., preoperative nutritional status, organ impairment, the severity of the patient's systemic response, and the premorbid physiological reserves, as predicted by the APACHE II scoring system) rather than to type and source of the infection. Death from intraabdominal infection, especially when the tertiary phase is reached, is thought to result from an exaggerated uncontrolled cytokine release that is unresponsive to all therapeutic attempts. This has led to investigation of endotoxin and cytokine levels in circulation to predict outcome. However, the magnitude of levels of endotoxin, TNF, and IL-6, in circulation in patients with peritonitis, has not been invariably related to prognosis. It has been suggested that cytokine levels m the peritoneal exudate, rather than the blood, belter reflect the severity of the compartmentalized peritoneal infection and predict outcome. A skeptical attitude is necessary when reviewing reports on clinical trials antimicrobial therapy for intraabdominal sepsis. Surgical therapy atone may be sufficient to cure many otherwise healthy, young patients with intraabdominal infection who have no signs of severe sepsis: even antimicrobial agents to which the pathogens are resistant may be associated with a good outcome for these patients.

To establish a specific role for antimicrobial regimens requires clinical trials with sufficient numbers of high-risk patients whose seventy of illness has been stratified by APACHE II scoring. Clinical trials are frequently diluted by low-risk patients, since the sickest patients who would test the efficacy of the antimicrobial agent arc often excluded by strict enrollment requirements

Intestinal intubation. The type of the tube which will be employed during a certain type of small intestine intubation is to be determined by the patient's health care prescriber or by the surgeon who is to perform the operation. There are several types of tubes, some of them are relatively stiff and preformed, while others are very thin and very flexible. Most tubes employed in the process of intubation are made of silicone or of flexible plastic. Surgeon performs intestinal intubation for complete evacuation of gas and contents from small intestine during paralytic ileus.

Drainage. In case of diffuse peritonitis the surgeon performs drainages of all parts of abdominal cavity: both subdiaphragmatic spaces, right and left flanks, pelvic region. However drains are undoubtedly of great help in cases of localized peritoneal fluid collections.

Closure. In case of midline sutures the linea alba are sutured as a single layer with polypropylene or nylon taking generous bites of tissues on either side. In case of right paramedian or transverse incision, the closure is performed in the usual fashion. The skin and subcutaneous layers of the wound usually should be left open.

The 1980s witnessed the evolution of two therapeutic concepts: open management (laparostomy) and "planned" relaparotomies. Planned relaparotomy addresses principle 4: multiple operative interventions are planned before or during (but not after) the first procedure for peritonitis. The commitment is made to return to the abdominal cavity to re-explore, evacuate, debride, or resect, until those disease processes indicating the need for serial relaparotomies are resolved. Moreover, planned relaparotomy allows verification of the integrity of anastomoses, fashioned at the first operation, or their eventual construction during subsequent relaparotomy, as opposed to the routine avoidance of suture-lines advocated by the single operation proponents.

Open management addresses principle 3 and facilitatis frequent re-exploration. It serves to decompress the high intra-abdominal pressure caused by peritoneal edema associated with inflammation and fluid resuscitation, thus obviating the deleterious systemic consequences of the abdominal compartment syndrome. Early results of these methods were promising, particularly in the management of infected pancreatic necrosis, but were less favorable in cases of postoperative peritonitis. Simple open management was plagued by intestinal fistulas and abdominal-wall defects, problems that were almost eliminated by introduction of temporary abdominal closure devices such as the artificial burr or mesh-zipper techniques. Staged abdominal repair as a conceptual operative approach combines the advantages of planned relaparotomy and of open management with a minimal rate of complications.

Second-look operations (predictable laparotomy) may be used in a damage control fashion. In these cases, the patient at initial operation is severely ill and unstable from septic shock or coagulopathy (mediator liberation, disseminated intravascular coagulation). The goal of the initial operation is to provide preliminary drainage and to remove obviously necrotic tissue. Relaparotomies were performed every 36 to 48 hours after the index laparotomy to inspect, drain, lavage, and perform other necessary abdominal interventions for residual peritonitis or new infectious focus. The sequence of planned relaparotomies was terminated when a macroscopically clean abdomen

was found at relaparotomy, indicating negative findings. That decision was made by the operating surgeon.In conditions related to bowel ischemia, the initial operation aims to remove all frankly devitalized bowel. The second-look operation serves to reevaluate for further demarcation and decision-making regarding re-anastomosis or diversion. Relaparotomy was only performed in patients with clinical deterioration or lack of clinical improvement with a likely intra-abdominal cause. Other (intercurrent) infectious foci (eg, pneumonia) were ruled out using laboratory tests, imaging modalities, or both. The decision to perform an on-demand relaparotomy was made by the multidisciplinary medical team. To guide the decision for reoperation, the following definitions of *deterioration* and *lack of improvement* were specified in the protocol. Deterioration after the previous operation was considered if there was an increase of more than 4 points in the Multiple Organ Dysfunction Score or prespecified surgical emergencies (ie, abdominal compartment syndrome; intra-abdominal bleeding with persistent decrease in hemoglobin despite replacement, and hemodynamic instability; burst abdomen; perforation of visceral organ; anastomotic leakage; intra-abdominal abscess that cannot be drained percutaneously; ischemia/necrosis of a visceral organ. Lack of improvement of clinical signs of persistent sepsis was considered if the Multiple Organ Dysfunction Score was unchanged (±2 points) for at least 48 hours following the index laparotomy or the previous relaparotomy. Abscess detected at CT imaging with positive fine-needle aspiration results (Gram stain with evidence of bacterial involvement) that could not be drained percutaneously was another reason for relaparotomy.

Table 7.4 – Disease-related morbidity needing nonsurgical treatment or surgical intervention

Disease-Related Major Morbidity Needing Readmission and Conservative Treatment but Not Surgery
1. Fistula: nonanatomical connection between hollow organ and cutis or between 2 hollow organs
2. Wound dehiscence/incisional hernia with obstruction: full-thickness discontinuity in abdominal wall with bulging of abdominal content
3. Abscess needing percutaneous drainage: pus-containing non-preexisting cavity confirmed by positive Gram stain or culture
4. Renal failure: urine production <500 mL/24 h with rising level of blood urea nitrogen and creatinine combined with dehydration (decreased circulating volume with elevated hematocrit needing intravenous rehydration) based on inadequate oral intake, nausea/vomiting, or both (only when needing readmission)
5. Myocardial infarction (electrocardiogram and enzyme changes suggestive of myocardial infarction or needing admission to coronary care unit), pulmonary embolus (ventilation-perfusion mismatch on lung scintigraphy), or cerebrovascular accident (ischemic or nonischemic with persistent paresis or paralysis without previous history)
6. Gastric or duodenal bleeding: needing endoscopic treatment or embolization therapy
7. Respiratory failure due to pneumonia, pleural effusion, or pulmonary edema and needing oxygen therapy or mechanical ventilation
8. Urosepsis: urinary tract infection with positive urine and blood cultures and circulatory shock

All participating surgeons and institutions had experience with both strategies. It was not required by protocol to keep patients in the planned relaparotomy group mechanically ventilated between operations. The decision to perform additional procedures during the relaparotomy was left to the discretion of the operating surgeon. Standardized cointerventions included direct postoperative care, intensive care unit (ICU) care, use of corticosteroids, postoperative feeding, and antibiotic treatment – all directed by physicians unrelated to the study.

The primary end point was a combination of all-cause mortality and major disease-related morbidity in surviving patients within 12-month follow-up after index laparotomy. A major morbidity end point in survivors was counted only if a prespecified major disease-related morbidity led to a surgical reintervention during index admission or readmission during the 12-month follow-up (with or without the need of surgical intervention) (Table 7.4, 7.5).

Table 7.5 – Disease-related morbidity needing nonsurgical treatment or surgical intervention

Disease-Related Major Morbidity Needing Surgical Intervention During First Admission or Readmission
1. Incisional hernia: full-thickness discontinuity in abdominal wall with bulging of abdominal contents with or without obstruction with disabling concerns interfering with daily activities
2. Bowel obstruction or herniation due to intra-abdominal adhesions: diagnosis must be confirmed during surgery
3. Burst abdomen: complete midline or transverse discontinuity in abdominal wall
4. Abdominal compartment syndrome: intra-abdominal hypertension >25 mm Hg with tense abdomen and with increasing respiratory failure, renal failure, or both; measured by the urinary bladder pressure method (modified Burch criteria)
5. Fistula: nonanatomical connection between intestine and cutis or between 2 hollow organs
6. Intra-abdominal bleeding: only when septic bleeding after index laparotomy or relaparotomy or surgical bleeding after relaparotomy but not after index laparotomy
7. Intra-abdominal hematoma needing surgical evacuation
8. Perforation of visceral organ confirmed at surgery
9. Anastomotic leakage: anastomotic leak on contrast imaging needing surgery or contrast-enhanced computed tomography scan, confirmed at relaparotomy
10. Ischemia or necrosis of a visceral organ: critically reduced blood flow to an intra-abdominal organ causing tissue loss, confirmed at pathological examination
11. Enterostomy dysfunction due to prolapse, stenosis, or retraction
12. Gastric or duodenal ulcer bleeding needing intervention of any type

Additional outcomes included health care utilization and direct medical costs during 12-month follow-up.

Laparostomy. In severe peritonitis, particularly with extensive retroperitoneal involvement (necrotizing pancreatitis), open treatment with repeat re-exploration, debridement, and intraperitoneal lavage has been shown to be effective. In these cases, the abdominal wall is temporarily closed, and a laparostomy is created to facilitate re-exploration or to prevent abdominal compartment syndrome (Fig. 7.9, 7.10).

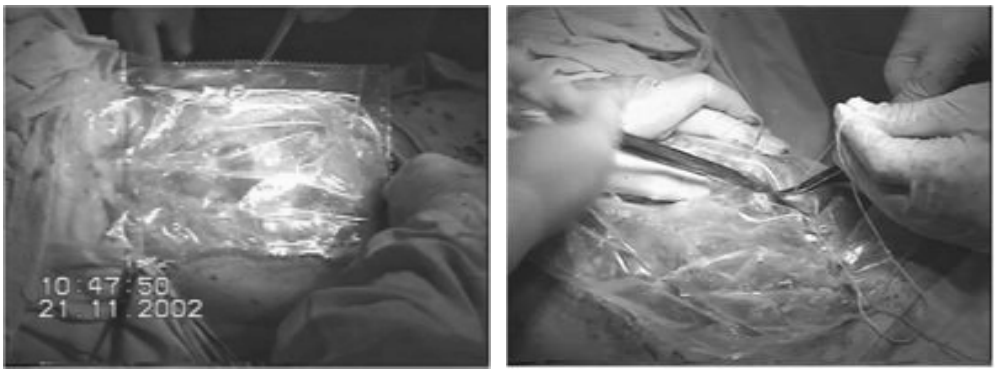


Figure 7.9. *Technique of "bogota bag" at a laparostomy*

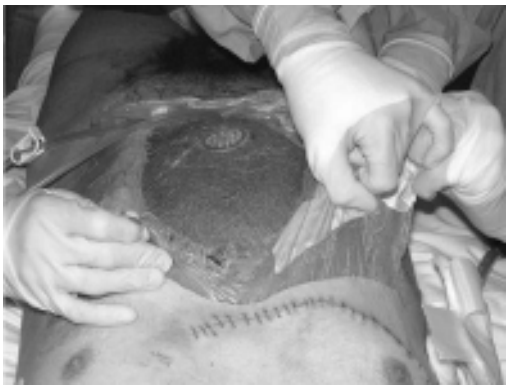


Figure 7.10. *Vacuum device (L. C. Argenta et al., 1997)*

Regarding the technique and material used for the temporary closure, no prospective randomized data exists, but mesh materials are commonly used.

They provide drainage of infectious material, permit visual control of the underlying viscera, facilitate access to the abdominal wall, preserve the fascial margin, enable healing by secondary intention, and allow mobilization of the patient. In the case of decreasing intra-abdominal pressure, meshes can be trimmed to centralize the rectus muscle and to facilitate definitive closure. Non-absorbable meshes have been frequently reported to cause enteric fistulae and persistent infection necessitating mesh explantation. Temporary closure of the abdomen to prevent herniation and contamination from outside the abdominal contents can be achieved using gauze and large, impermeable, self-adhesive membrane dressings, mesh (Vicryl, Dexon).

Some studies have described that the planned strategy increases the risk of multiple organ failure due to amplifying the systemic inflammatory response by multiple surgical lavages, leading to increased mortality, ICU stays, and hospital stays. We also observed that patients treated with the planned strategy had longer ICU stays and had a longer overall hospital stay. The duration of mechanical ventilation was significantly longer for the planned treatment patients. However, this difference in ventilation time may in part be related to the short period between scheduled relaparotomies in the planned strategy, inherent to the planned nature of the procedures, as a result of which some patients could not be weaned off ventilation before the next operation. The number of (minimally invasive) percutaneous interventions was also significantly lower in the on-demand group. Possibly, free abdominal fluids and abscesses were more frequent after multiple surgical interventions due to the reinterventions or a modified

inflammatory response. Other potential drawbacks of the planned relaparotomy strategy are the observed strong adherence of microbes residing in the peritoneum making them resistant to peritoneal lavage. This reduces the effectiveness of the procedure and the damaging effects of lavage to the mesothelial layer may even reduce the innate resistance to infection.

In our trial, the on-demand and planned relaparotomy strategies were equally apt to identify patients with remaining or new intra-abdominal infection after the index laparotomy. This also confirms that patients in the planned group were not more frequently determined to show positive findings due to differential verification. In other words, surgeons were not more inclined to determine a planned reoperation as positive, for example, to justify this scheduled intervention.

Although the on-demand strategy reduced the number of relaparotomies, there was still a 31% chance that macroscopic findings were negative in patients selected for relaparotomy. The key challenge in the on-demand strategy is to adequately select patients for relaparotomy and to prevent potentially harmful delay in reintervention by adequate and frequent patient monitoring. A more rigorous use of CT scanning as part of the procedure of selecting patients with abdominal sepsis for relaparotomy may well reduce the proportion of patients with negative findings at relaparotomy even further.

Multiple independent variables and combinations of variables have been described to predict outcome of peritonitis. However, results in the literature are inconclusive and the majority of studies predict disease outcome (mortality) of sepsis rather than positive findings at relaparotomy in secondary peritonitis.

In attempt to guide decision making for relaparotomy and enhance timing of relaparotomy in the on-demand group within this trial, we prespecified the main criteria for necessity of relaparotomy as lack of clinical improvement or clinical deterioration using a quantified method (the Multiple Organ Dysfunction Score). The European Sequential Organ Failure Assessment score may have been an alternative prognostic score for predicting presence of persisting peritonitis. However, although scoring systems were used, the final decision to perform a reoperation on a patient in the on-demand setting was always made within a multidisciplinary team. Therefore, considerations for relaparotomy concerning clinical, laboratory, and imaging parameters were less explicit, but in line with current clinical practice. Future research should focus on optimizing adequate and timely selection of patients for relaparotomy by identification of predictive variables for positive findings at relaparotomy and evaluating the added diagnostic value of diagnostic imaging and potential biomarkers.

One of the difficulties in research on secondary peritonitis is heterogeneity of the study population regarding, eg, severity of disease, etiology, and localization of the infectious focus,1which often make it difficult to extrapolate study results to individual patients in clinical practice. For this reason, we have excluded disease entities with substantially different prognosis and requiring different treatment strategy, like pancreatitis, perforation due to endoscopy operated within 24 hours, and catheter-associated peritonitis. We also excluded patients with an APACHE-II score of 10 or lower, because the on-demand strategy is already the treatment of choice in mild peritonitis.Within the trial, we examined whether the treatment effect with respect to the combined end point differed with the severity of disease at index laparotomy. We found no indication that relative treatment effects differed between patients with moderate to severe disease (APACHE-II score 11–20) and those with severe disease

(APACHE-II score > 20). Treatment effects were also comparable across including hospitals, translating to the on-demand strategy being a feasible and valid option in both moderately and severe secondary peritonitis in every hospital setting.

Postoperative management

Fluid resuscitation. The patients are usually hypovolaemic with disturbed electrolytes. Large volumes of fluid may be needed very rapidly till blood volume and urine output are restored. Till the central venous pressure (CVP) reaches the level of 10cm of water, fluid administration should be at a rapid rate. The most frequent mistake made is that the rate of initial fluid administration is too slow. The slow rate is undertaken for fear of precipitating congestive cardiac failure. If CVP is monitored, there is no chance of occurrence of congestive cardiac failure.

Central venous pressure (CVP) describes the pressure of blood in the thoracic vena cava, near the right atrium of the heart. CVP can be measured by connecting the patient's central venous catheter to a special infusion set which is connected to a small diameter water column. If the water column is calibrated properly the height of the column indicates the CVP.

In most progressive intensive care units, specialized monitors are available to continuously measure CVP as well as other hemodynamic values.

Normal values are 2–8 mmHg. CVP reflects the amount of blood returning to the heart and the ability of the heart to pump the blood into the arterial system.

It is a good approximation of right atrial pressure, which is a major determinant of right ventricular end diastolic volume. (However, there can be exceptions in some cases.) It is an important diagnostic information for various serious diseases of the heart and lungs.

Factors which increase CVP include:

- Hypervolemia.
- Forced exhalation.
- Tension pneumothorax.
- Heart failure.
- Pleural effusion.
- Decreased cardiac output.
- Cardiac tamponade.
- Factors which decrease CVP include: hypovolemia, deep inhalation, distributive shock.

Fluid administered must include crystalloid to replace water and electrolytes. Colloids (albumin or plasma) should also be given to restore an effective volume quickly. Plasma protein depletion needs correction as inflamed peritoneum leaks protein continuously. Whole blood or packed red blood cells are administered, if needed, to correct anemia and to maintain an adequate red cell mass.

Antibiotics

Antibiotic therapy appears less effective compared to all other forms of peritonitis. The microbiology of peritonitis is well characterized. Because it is mainly a community-acquired disease, advanced antimicrobial resistance does not usually occur, and effective antibiotic strategies have been derived from the knowledge of the bacteria involved .

The micro-organisms associated with primary, secondary and tertian peritonitis are summarized in Table 7.6, 7.7, 7.8.

Table 7.6 – Microbiology of primary peritonitis

Peritonitis (type)	Etiologic class	Organisms type of organism
Primary	Gram-negative	E.coli (40%) K. pneumoniae (7%) Pseudomonas species (5%) Proteus species (15%) Streptococcus species (15%) Staphylococcus species (3%) Anaerobic species (<5%)

Table 7.7 – Microbiology of secondary peritonitis

Peritonitis (type)	Etiologic class	Organisms type of organism
Secondary	Gram-negative	E. coli, Enterobacter species Klebsiella species Proteus species
Secondary	Gram-positive	Streptococcus species Enterococcus species
Secondary	Anaerobic	Bacteroide fragilis Other: Bacteroides species Eubacterium species Clostridium species Anaerobic Streptococcus species

Table 7.8 – Microbiology of tertiary peritonitis

Peritonitis (type)	Etiologic class	Organisms type of organism
Tertiary	Gram-negative	Enterobacter species; Pseudomonas species
Tertiary	Gram-positive	Staphylococcus species
Tertiary	Fungal	Candida species

Ventilatory support. Oxygen is administered to help the response to the increased metabolic demands of peritonitis which are so often associated with impairment of pulmonary ventilatory function and mild hypoxia. A nasal catheter supplying oxygen at about 5 litres per minute is sufficient prior to induction of anesthesia. Assessment of respiratory function should be made clinically noting the work of breathing and apparent tidal volume. If impairment is suspected, measurement of ventilatory volume and arterial blood gases are indicated. When the patient's arterial oxygen tension falls below 70 mm Hg, ventilatory support with inspired gas concentration of 40% oxygen should be given.

Vasoactive drugs. Administration of these drugs has not been very effective. Only when *Staphylococcus aureus*, an exotoxin-producing organism is the causative agent, combination of phentolamine and propranolol significantly improves the

condition of the patients. Drugs with alpha-adrenergic effects are of limited value as this artificial attempt to maintain blood pressure by inducing vasoconstriction is potentially harmful.

Analgesics should not be administered to patients until diagnosis is made. Analgesia may obscure abdominal findings and gives rise to difficulty in establishing a firm diagnosis. Once a decision on diagnosis and operation has been made, pain may be relieved with potent narcotics e.g. morphine intravenously.

Tertiary peritonitis

Tertiary peritonitis refers to recurrent intra-abdominal infection after initial surgical and antimicrobial therapy of secondary bacterial peritonitis. The entity has a variable definition, in part because of the different populations of patients in whom it has been described. Earlier reports have focused on patients in the intensive care unit. In this setting, it has been referred to as a "syndrome" characterized by the presence of multiple organ failure, poorly localized infections typically not amenable to percutaneous drainage and a microbial flora different from that of secondary bacterial peritonitis with a preponderance of Enterococci, Candida spp., Coagulase negative staphylococcus and Enterobacter spp. In this population, mortality is 64%, twice that of comparable patients who do not go on to develop tertiary peritonitis. It is not clear precisely why outcomes are so poor in patients with tertiary peritonitis. The most important predictors of who will go on to develop tertiary peritonitis are patients with a high APACHE II score, pancreatic or small bowel source, gram-positive, fungal, or other resistant pathogens, malnutrition, and poor surgical source control. In spite of the identification of specific risk factors for tertiary peritonitis, the data supporting a change in empiric antimicrobial therapy or a different surgical approach (e.g., scheduled relaparotomy) to prevent the development of tertiary peritonitis are limited. These findings again suggest that the syndrome might be more a reflection than a cause of adverse outcome. Aggressive supportive and operative measures allow the salvage of patients who previously would have died of uncontrolled peritonitis.

However, delays in management and iatrogenic factors during the 1980s produced a "new" subgroup of patients who died of "sepsis" and multiple-organ failure despite having a "clean" abdomen. However, occasionally at surgery, some thin, cloudy, microorganism – containing peritoneal fluid was found. The term tertiary peritonitis was coined to describe this situation, which develops late in the postoperative phase, presents clinically as sepsis, and is associated with a sterile peritoneal cavity or peculiar microbiology (Table 8.8). Two or three planned reoperations, supplemented with a short course of antibiotics, are sufficient to sterilize the peritoneum in the most severe peritonitis. This phase, when infection is cured but severe peritoneal and systemic inflammation persists, represents tertiary peritonitis. Further antimicrobial administration and operative interventions are futile and may contribute to the peritoneal superinfection with yeasts and other commensuals. The low virulence of these organisms, which represent a marker of tertiary peritonitis and not its cause, reflects the global immunodepression of the affected patients. The usually fatal outcome of tertiary peritonitis, which conceptually falls within the systemic inflammatory response syndrome-multiorgan failure complex, indicates that current antibiotic-assisted, mechanical answers to severe peritonitis have about reached their limits. To further improve results, both the initiator and the biologic consequences of the peritoneal infective-inflammatory process should be addressed. Current therapy deals adequately

with the initiator. The search for a magic bullet (or bullets) to abort its systemic consequences continues.

7.7. COMPLICATIONS OF PERITONITIS

- Acute intestinal obstruction due to peritoneal adhesions.
- Abscesses of abdomen cavity.
- Eventration.
- Intestinal fistula.
- Persistent abdominal sepsis.
- Bleeding.

Навчальне видання

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