Peritonitis

**Definition**
Peritonitis is an inflammation (irritation) of the peritoneum, the thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs.

**Etiology**
Bacteria from the alimentary canal:
- Usually the infection is caused by two or more strains.
- The commonest invaders are E. coli, aerobic and anaerobic strept, and the Bacteroides.
- Less frequently C. welchi, or Klebsiella.

Bacteria not from the alimentary canal:
- Gonococc, B-haemolytic strept, pneumococci, and Mycobacterium tuberculosis.
- In girls and women, pelvic infection via the F. tubes ‘non-alimentary’ infections, e.g. gonococcus and streptococcus, & Bacteroides.

**Pathway of bacterial invasion of peritoneal space**
1. Direct infection
   - a) Perforation of some part or Site gastrointestinal canal;
   - b) Penetrating wound of the abdominal wall;
   - c) Operative, e.g. drains, dialysis tubes, foreign material.
2. Local extension
   - a) Inflamed organ, e.g. appendicitis, cholecystitis;
   - b) migration through gut wall, e.g. strangulated hernia;
   - c) from or via the Fallopian tubes.
3. Blood-stream
   - a) Part of general septicemia.

**Mortality risk**
1. The degree and duration of peritoneal contamination
2. The age of the patient.
3. The general health of the patient.
4. The nature of the underlying cause

**Natural factors favor localization of peritonitis**
- Excluding the subphrenic spaces, the greater sac of the peritoneum is divided into the pelvis, and the peritoneal cavity proper.
- The latter is re-divided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon, which deter the spread of infection from one to the other.
- When the supracolic compartment overflows (ulcer perforation) into the infracolic compartment, or, by way of the right paracolic gutter to the right iliac fossa, and thence to the pelvis.
- Posture can assist in directing collections into the pelvis, as in the Sherren regime for perforated appendicitis.
- The clinical course is largely governed by the manner in which adhesions form around the affected organ.
- Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety.
- Flakes of fibrin appear and cause coils of intestine to become adherent to one another and to the parietes.
- There is an outpouring of serous fluid rich in leukocytes and antibodies that soon becomes turbid; if localization occurs, the turbid fluid becomes frank pus.
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- Peristalsis is retarded in affected coils, and this helps in preventing distribution of the infection.
- The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.
- Drains are used postoperatively to assist localization (and exit) of intra-abdominal collections: their value is disputed.
- Certain types of drain, e.g. red rubber, are known to be both a cause of increased peritoneal exudate and a route for bacterial invasion.

**Pathological**
- It is bound up intimately with the causative lesion.
- Initial symptoms and signs are those of that lesion.
- When the peritoneum becomes inflamed the temperature, and the pulse rate, rise.
- The pain increases and usually there is associated vomiting.

**Surgical**
1. Whether peritonitis develops rapid or slow, inflamed appendix perforates early before protective mechanisms have mobilized.
2. Perforation proximal to an obstruction, or from sudden anastomotic separation, is associated with severe generalized peritonitis and a high mortality.
3. Ingestion of food or water, by stimulating peristaltic action, hinders localization. Violent peristalsis (purgative or enema), promotes a widespread distribution of an infection.
4. High virulence of the infecting organism
5. In children (small omentum)
6. Injudicious and rough handling of localized collections, e.g. appendicular mass or pericolic abscess;
7. Deficient natural resistance (‘immune deficiency’):

**Local extension**
- It is bound up intimately with the causative lesion.
- Initial symptoms and signs are those of that lesion.
- When the peritoneum becomes inflamed the temperature, and the pulse rate, rise.
- The pain increases and usually there is associated vomiting.

The most important sign is
1. Guarding and rigidity over the area of the abdomen which is involved, with a positive ‘release’ sign.
2. If inflammation arises under the diaphragm, shoulder tip (‘phrenic’) pain may be felt.
3. 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.

**Intermediate phase**
- Peritonitis may resolve, so that the pulse slows, the pain and tenderness diminish, leaving a silent, soft abdomen. (These features can be easily mislead the observer.) The condition may localize, producing one or more abscesses, with overlying swelling and tenderness.

**Terminal phase**
- If resolution or localization have 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 (Hippocratic facies).
- The patient finally lapses into unconsciousness.

(With early diagnosis and adequate treatment, this condition is rarely seen in modern surgical practice)
## Investigations

1. **Blood picture**: Peritonitis usually produces leucocytosis, often delayed for many hours.
2. **Peritoneal diagnostic aspiration** may be helpful but is usually unnecessary.
3. A radiograph of the abdomen may reveal free air, or confirm the presence of dilated gas-filled loops of bowel with multiple fluid levels. If the patient is too ill for an ‘erect’ film to demonstrate free air collecting under the diaphragm, a lateral decubitus film is just as useful, showing gas beneath the abdominal wall.
4. Serum amylase estimation may uphold the diagnosis of pancreatitis, provided it is remembered that raised values are frequently found following other abdominal catastrophes, and operations, e.g. perforated duodenal ulcer.
5. Ultrasonography is a most valuable investigation for demonstrating the presence of intraperitoneal collections of fluid or pus. It can be combined with guided aspiration of any demonstrated collection.

## Treatment

### General care of the patient

1. **Intravenous fluids**
   - Correct hypovolaemia and electrolyte disturbances
   - Central venous line: for monitoring accurate fluid replacement.
   - Plasma protein depletion need correction as inflamed peritoneum leaks protein continuously.
   - If the patient’s recovery is delayed for more than 7—10 days, intravenous feeding (‘hyperalimentation’) is required.

2. **Nasogastric tube**:
   - Stomach is aspirated.
   - Intermittent aspiration is maintained until paralytic ileus resolved.
   - Measured volumes of water are allowed by mouth when only small amounts are being aspirated.
   - If the abdomen is soft and not tender, and bowel sounds return, oral feeding may be progressively introduced.

3. **Antibiotics**.
   - Administration of antibiotics prevents the multiplication of bacteria, and the release of endotoxins.
   - As the infection is usually a mixed one, initially parenteral broad-spectrum antibiotics active against aerobic and anaerobic organisms and metronidazole may be given.

4. **A fluid balance chart**
   - Must be started at once so that daily output by gastric aspiration and urine are known, and losses from the lungs, skin, and in faeces are estimated, so that the intake requirements can be calculated and administered.

5. **Analgesia**.
   - The patient is nursed in the sitting-up position, must be relieved of pain before and after operation.
   - Once the diagnosis has been made, morphine may be given, and small doses continued for 48 hours.
   - Freedom from pain allows early mobilisation and adequate physiotherapy in the postoperative period, to prevent atelectasis, deep-vein thrombosis and pulmonary embolism.

6. **Oxygen**:
   - Especially if septic shock is present.
   - Special measures may be needed for cardiac, pulmonary and renal support.
   - Administration of oxygen postoperatively can help to prevent the effects of septic shock, especially adult respiratory distress syndrome (ARDS).
   - If oligaemia persists despite adequate fluid replacement, both dopamine and diuretics may be needed.

### Neutralization of the local source

- If the cause is amenable to surgery (appendicitis, diverticulitis, peptic ulcer, gangrenous cholecystitis, perforation of the small bowel), operation must be carried out as soon as the patient is fit for anesthesia. This is usually within a few hours.
- In peritonitis due to pancreatitis, salpingitis, or in cases of primary peritonitis of streptococcal or pneumococcal organisms, conservative treatment is the procedure of choice (if the diagnosis can be made with certainty).

### Peritoneal lavage

- After the cause has been dealt with, the whole peritoneal cavity should be explored with the sucker and mopped dry, if necessary until all seropurulent exudate is removed.
- The use of a large volume of antibiotic saturated saline (1—2 litres) has been shown to be very effective for ‘cleaning’ the peritoneum (Matheson).

## Prognosis

With modern treatment diffuse peritonitis carries a mortality of about 10 %. The lethal factors are:

- a) bacterial toxemia;
- b) paralytic ileus;
- c) bronchopneumonia;
- d) electrolyte imbalance;
- e) renal failure;
- f) undrained collections;
- g) bone-marrow suppression;
- h) ‘multisystem breakdown’.