**Surgical infections**

*Physiology*
Wound infection results from bacterial contamination of the wound.
Infection rate is proportionate to:
• Number of bacteria;
• Type of bacteria;
• Incisions involving mucus surfaces;
• Sites of existing infection in the body;
• The use of prosthetic implants.

An appreciation of the sources of bacteria is important and in abdominal surgery these may be summarised as:
1. Endogenous from the patient’s viscera (98 per cent);
2. Endogenous from the patient’s skin;
3. Contamination from the air in the operating theatre — rare (by comparison, in 98 per cent of orthopaedic infections the contaminating organisms are ultimately derived from the air.
4. Direct contamination, such as punctured gloves — very uncommon.

- Nosocomial infections are acquired in hospital
- Community-acquired infections are acquired outside hospital

Three-quarters of nosocomial infections occur in surgical patients, who account for 40% of hospital inpatients. Sources of infection could include the following:

- **Patient’s own body flora:**
  - Failure of correct aseptic technique;
  - Contaminated surgery.

- **Indirect contact:**
  - Contact from hands of doctors, nursing staff, patients, visitors;
  - Contaminated surfaces, e.g. door handles, cups.

- **Direct inoculation:**
  - Surgeon or environmental flora through failure of aseptic technique;
  - Contaminated instruments or dressings;
  - Colonization of indwelling drains, catheters, IV lines.

- **Airborne contamination:**
  - Skin and clothing of staff, patients, and visitors;
  - Air flow in operating theatre or ward.

- **Haematogenous spread:**
  - IV and intraarterial lines;
  - Contaminated infusions;
  - Sepsis at other anatomical sites.

- **Food- and water-borne.**
- **Faecal-oral.**
- **Insect-borne.**
Bacteria are normally prevented from causing infection in tissues by intact epithelial surfaces, but these are broken down by surgery. Host response is weakened by malnutrition which may present as obesity as well as recent rapid weight loss. Metabolic diseases, diabetes mellitus, uraemia and jaundice may weaken defences, and disseminated cancer may also be included together with immunosuppression caused by radiotherapy, chemotherapy, steroids and acquired immunodeficiency syndrome. When enteral feeding is suspended in the perioperative period, the gut rapidly becomes colonised and bacteria, particularly Gram-negative bacilli, translocate to mesenteric nodes. Release of endotoxin may follow, which further increases susceptibility to infection.

The pathogenicity and size of bacterial inoculum also relates to the chance of developing an established wound infection after surgery. Poor surgical technique that leaves devitalised tissue, excessive dead space or haematoma may increase this risk. Foreign materials of any kind, including sutures and drains, promote infection.

Types of infection

Wound abscess

A wound abscess presents all the clinical features of acute inflammation: heat, redness, pain and swelling, to which can be added loss of function. Pyogenic organisms, predominantly Staphylococcus aureus, cause tissue necrosis and suppuration. Pus is also composed of dead and dying white blood cells which release damaging cytokines, oxygen-free radicals and other molecules. An abscess is surrounded by an acute inflammatory response, and a pyogenic membrane composed of fibrinous exudate and oedema, and the cells of acute inflammation. Granulation tissue (macrophages, angiogenesis and fibroblasts) forms later around the suppuration and beads to collagen deposition. If excessive or partly sterilised by antibiotics (antibio), a chronic abscess may result. Abscesses may need debridement and curettage with an exploration to break down all loculi before resolution can occur. Persistent chronic abscesses may lead to sinus or fistula formation. In a chronic abscess, lymphocytes and plasma cells are seen with sequestration and later calcification. Certain organisms are related to chronicity, sinus and fistula formation, e.g. mycobacteria and actinomyces, and should not be forgotten.

The role of antibiotics in the treatment of wound abscesses is controversial unless there are signs of spreading infection (ceblulitis or lymphangitis).

Cellulitis

This is the nonsuppurative invasive infection of tissues. In addition to the cardinal signs of inflammation, there is poor localisation. Spreading infection is typical of organisms such as B-haemolytic streptococci, staphylococci and C. perfringens. Tissue destruction and ulceration may follow, caused by release of streptokinase, hyaburonidase and other proteases. Systemic signs (toxaemia) are common: SIRS, chills, fever and rigors. These follow release of exotoxins and cytokines but blood cultures are often negative.

Lymphangitis

is caused by similar processes but presents as painful red streaks in affected lymphatics. Cellulitis is usually located at the point of injury.
and subsequent tissue infection. Lymphangitis is often accompanied by painful lymph node groups in the rebated drainage area.

**Bacteraemia and septicaemia**
These are unusual in superficial wound infections but common after anastomotic breakdown. They are usually transient and follow procedures undertaken through infected tissues (particularly instrumentation in infected bile or urine). Bacteraemia is important when prosthetics have been implanted, particularly cardiac valves. Septicaemia commonly relates to colonisation and translocation in the gastrointestinal tract and may follow anastomotic breakdown accompanied by MSOF. Aerobic Gram-negative bacilli are mainly responsible but S. aureus and fungi may be involved, particularly after the use of broad-spectrum antibiotics.

**Specific wound infections**

**Gas gangrene**
Gas gangrene is caused by C. perfringens. The Gram-positive, spore-bearing bacilli are widely found in nature, particularly soil and faeces, which is relevant to military and traumatic surgery, and colorectal operations. Patients who are immunocompromised, diabetic or have malignant disease are at risk, particularly when anaerobic wound conditions are present with necrotic or foreign material. Wound infections are associated with severe local wound pain and crepitus (gas in the tissues which may also be noted on plain radiographs). Systemic complications with circulatory collapse and MSOF supervene without appropriate intervention. Prophylaxis in patients at risk should always be considered, particularly amputation for peripheral vascular disease. Once established, large doses of intravenous penicillin and aggressive debridement of affected tissues are required.

**Synergistic spreading gangrene (necrotising fasciitis)**
It is not caused by clostridia. A mixed pattern of organisms is responsible — coliforms, staphylococci, Bacteroides spp., anaerobic streptococci and peptostreptococci have been implicated. Synonyms have been associated with abdominal wall infections (Meleney’s synergistic hospital gangrene) and scrotal infection (Fournier’s gangrene). Patients are almost always immunocompromised (such as diabetes mellitus).

**Treatment**
Debridement and antibiotic cover (Penicillin and clindamycin)

**Clostridia Tetanus**
The causal organism, is a Gram-positive anaerobic rod with terminal spores (drumstick appearance). Found in manure and soil, it will invade any wound. It multiplies and produces a powerful toxin in any deep, contused wound in the presence of dead tissue, foreign bodies and other bacteria. Penetrating injury from the hoof of an animal can be associated with this infection, while the prick from a rose thorn in a well-manured rose garden can be the sting of death to an elderly assiduous horticulturalist. The exotoxin produced in the inoculation site inhibits the cholinesterase at the motor endplates, resulting in an excess of acetylcholine locally and, therefore, a sustained state of tonic muscle spasm. The exotoxin also travels along the nerves to the central nervous system and causes extreme hyperexcitability of motor neurons in the anterior horn cells, thereby evoking explosive and
widespread reflex spasms of muscle in response to sensory stimuli. Once fixed in the nerve tissue, the toxin can no longer be neutralised by antitoxin.

**Symptoms and signs**  
Dysphagia, jaw stiffness and severe pains in the neck, back and abdomen precede the tonic muscle spasms. The sardonic smile of tetanus (risus sardonicus) is evidence of the onset of tonic muscle spasm. Respiration and swallowing become progressively more difficult, and reflex convulsions occur affecting all muscles and causing great pain, opisthotonus (spasm of the extensors of the neck, back and legs to form a backward curvature) and even muscle rupture. The spasms are spontaneous, but can be induced by trivial stimuli such as noise or movement and, when severe, will prevent respiration and produce cyanosis. The temperature is elevated, the pulse is rapid, and respiratory failure and death during a cyanotic attack will usually follow if treatment is not initiated.

**Treatment**  
Isolation, quietness and comfort, drainage of pus and wound toilet will be needed. Human anti-tetanus globulin is given intramuscularly (i.m.) to limit the effects of free toxins and should be used in doses of 25—500 units to give cover throughout the period of establishing active immunity by giving toxoid (tetanus vaccine, adsorbed) i.m. The patient may need ventilatory support or general anesthesia for the convolution.

**Salmonella (typhi, paratyphi)**  
These are enteric pathogens which cause enteric fevers with bacteraemia, osteomyelitis and sometimes perforation of ileal ulcers. Persistence of the bacteria in the gallbladder may lead to the carrier state and subsequently person-to-person spread in the community. The use of ciprofloxacin 500 mg twice daily for 10 days is recommended for S. Typhi infections. For salmonella paratyphi give ampicillin.

**Mycobacterium Tuberculosis**  
There are three routes of primary infection:  
• Direct spread to lungs;  
• From tonsils to the lymph nodes of the neck where an abscess may form and track round the edge of the sternomastoid muscle, producing a collar-stud abscess;  
• From lower ileal infection to the lymph nodes of the ileocaecal angle.  

**Guidelines for treatment**  
Nutrition and hygienic living conditions are still crucially important in preventing the spread of this infection.  
Treatment with triple therapy consisting of rifampcin 600 mg, isoniazid 300 mg and pyrazinamide 1500—2000 mg per day given orally for at least 2—3 months is the standard chemotherapy at present, followed by 6 months of double therapy (rifampicin plus isoniazid).

**Actinomycosis**  
This disease is caused by Actinomyces israeli, an anaerobic, Gram-positive, branching, filamentous organism Trauma and the presence of carious teeth are important predisposing factors in the development of lesions in the mouth.
Diagnosis depends on finding the organism in pus or in tissue section. Pus should be collected in a sterile tube (a swab is usually insufficient) and inspected in a good light for the presence of pinhead-sized ‘sulphur granules’. On microscopy, the granules are seen to consist of Gram-positive branching bacilli. The peripheral filaments radiate from the central part of the granule and may be surrounded by Gram-negative tissue clubs.

There are four main clinical forms of actinomycosis.

- **Faciocervical is the commonest.** The lower jaw is more frequently affected, often adjacent to a carious tooth.
- **Thorax.** The lungs and pleura are infected, either by aspiration of the bacillus or by direct spread from the pharynx or neck, or even upwards through the diaphragm. The chest wall, in the late stages, becomes riddled with sinuses.
- **Right iliac fossa.**
- **Liver.**

**Treatment.**

Actinomycosis is usually sensitive to penicillin, tetracycline and some other antibiotics, e.g. lincomycin, but the sensitivity should be checked in the laboratory. A prolonged intensive course of penicillin (10 megaunits reducing to 4 megaunits daily) is usually the best treatment until all signs of the disease have disappeared.