Infection impacts upon surgical practice in two main ways. First, many of the conditions treated by the surgeon are caused by infection, commonly bacterial but occasionally with other organisms. Second, safe surgical treatment is only possible if peri-operative infection is eliminated or controlled. It is nowadays taken for granted that operations are carried out in a clean environment rendered so by sterilization. However, as many infections come from organisms carried by the patient, infection remains a constant risk.

Antibiotics are used frequently in the treatment and prevention of infections, but their very wide use and often abuse has resulted in the emergence of resistant strains of bacteria that cause major problems, particularly in the hospital environment.

This chapter discusses the general principles of the investigation of infection in the surgical patient, particularly in the per-operative period, and the principles of management of those infections. Other specific infections are dealt with later in the appropriate chapters.

Virus infections rarely require surgical treatment, but patients suffering from conditions such as viral hepatitis and human immunodeficiency virus (HIV) present special problems, which are discussed below.

**ANTISEPSIS AND STERILIZATION**

Surgeons aim to carry out surgical operations in an environment free of bacteria. This may be achieved by:

- **Asepsis**: the concept of eliminating all bacteria from instruments and everything that might enter the operative area
- **Antisepsis**: the concept of reducing the number of bacteria, hopefully to zero by the use of antibacterial chemicals.

Lister carried out the first clean operations at the end of the eighteenth century. Although bacteria were known to exist at that time, following the work of Pasteur some years earlier, Lister did not believe that bacteria caused infection and used phenol, an antiseptic, on purely empirical grounds.

In practice, the distinction between asepsis and antisepsis is not important, and it is usual to combine both approaches.

Although it is always desirable to eliminate any bacteria before and during surgery, the intensity of the measures employed depends upon the type of operation contemplated, e.g. any infection after a heart valve or joint replacement can be a disaster, whereas the level of asepsis/antisepsis required for drainage of an abscess is less exacting. In the emergency setting the patient may already be infected with endogenous organisms.

**PRECAUTIONS BEFORE AN OPERATION**

Many wound infections are caused by the patient’s own skin flora and many methods are used to reduce this source of contamination before operation. Evidence of their efficacy is largely lacking, but it is reasonable to allow the patient a bath or shower before surgery to achieve at least social cleanliness. Any obvious infection, such as infected acne, or any dirty areas such as material accumulated in the umbilicus, should be eliminated. If these simple methods fail, an operation may have to be postponed.

Bathing or showering with colourful antiseptic solutions before surgery is sometimes used, but there is no evidence that it has any effect on the rate of wound infection.

Shaving body hair is traditional. It is certainly neater if the area to be incised is cleared of hair. It also makes the removal of adhesive dressings less
There are three main methods for sterilizing instruments and endoscopes:

- **heat**
- **chemicals**
- **radiation.**

The aim is to eliminate bacterial spores as well as bacteria.

**Heat** is applied in an autoclave filled with high-pressure steam. Usually the process is carried out at a central site, away from the theatre suite.

The most effective of the chemical methods is prolonged soaking in gluteraldehyde, but the solution is toxic and requires special facilities for its use. It is often the preferred method for delicate endoscopes.

**Radiation** with gamma-rays is highly effective but is not available in most hospitals. Its main use is to sterilize pre-packed mass-produced disposable items such as meshes for hernia repair and artificial joints.

There are many other methods of sterilization but a full discussion of their merits is beyond the scope of this book.

**WOUND INFECTION**

Wound infection almost invariably occurs during the operation when tissues are exposed and tissue planes opened and separated. In most cases the organisms come from the patient, either from the organ being operated upon or the skin. Other contaminating bacteria come from the theatre environment or personnel. Bacteria usually enter a wound directly, but it is possible for a wound to become infected by haematogenous spread with organisms coming from infected invasive monitoring and intravenous lines.

A wide variety of organisms may be involved. It is often possible to deduce the source of infection from the type of bacteria isolated, e.g. a coliform bacillus in a wound will suggest the gut as the likely source, particularly after abdominal surgery.

The precise definition of a wound infection is surprisingly difficult as wounds may become inflamed without infection. For most purposes, including the measurement of wound infection rates, a wound is declared infected if, after an operation, it discharges pus or bacteria are isolated from an exudate.
Antibiotic prophylaxis against wound infection

Risk factors that influence the incidence of wound infection after surgery

Contamination
The risks of wound infection after an operation depend upon the field through which the operation is being performed.

The site of the incision may be:

- **Clean.** Elective surgery through a clean field, e.g. the repair of a groin hernia, carries an infection risk of the order of 1 per cent. Nevertheless, the consequences may be significant if implanted synthetic materials become infected and have to be removed.

- **Clean then contaminated.** A clean wound may become contaminated by an intra-operative procedure that opens a contaminated area, e.g. opening a deep abscess or opening a viscus. A good example is cholecystectomy. The gall bladder is part of the gut and in 40 per cent of the gall bladders that contain gall stones the bile contains bacteria. The risk of wound infection after operating through a clean wound that becomes contaminated is 5–10 per cent.

- **Contaminated.** If the operative field contains bacteria at the time of surgery, as in an operation for appendicitis, the chance of infection is 10–20 per cent.

- **Dirty.** If the operative field contains pus or bowel content at the time of surgery the risk of postoperative infection is at least 50 per cent.

Obesity
Fat is a good culture medium for bacteria.

Diabetes

Immunosuppression caused by disease or chemotherapy

Heavy blood loss during the operation

Foreign bodies in the wound

Postoperative haematoma

Blood clot is a good culture medium. This risk can be reduced by the use of closed suction wound drainage.

**ANTIBIOTIC PROPHYLAXIS AGAINST WOUND INFECTION**

Prophylaxis with antibiotics is used when the risk of wound infection is high or when the consequences of infection are very serious. The basic principles guiding the use of prophylactic antibiotics are set out in Table 3.1.

The most effective way to give a prophylactic antibiotic is intravenously at the induction of the anaesthetic. Only in long procedures will it be necessary to give more than one dose. There is no advantage in continuing prophylactic antibiotics after the operation.

The illogical and indiscriminate use of antibiotics is a major factor in the induction of resistant strains of bacteria. The choice of antibiotic depends on the surgery and the likely infecting organisms. For example, with bowel surgery it is vital to include an agent such as metronidazole, which is active against anaerobic organisms.

The penetration of the antibiotic into the tissues must also be considered.

There is good evidence for the use of antibiotic prophylaxis against wound infection. Its incidence can be reduced to one-tenth. It is however important to appreciate that this is prophylaxis not treatment and it must be confined to the operative period.

---

**Table 3.1**
**Principles for antibiotic prophylaxis against wound infection**

<table>
<thead>
<tr>
<th>Principle</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>The antibiotic prophylaxis must be given peri-operatively because infection occurs during the operation</td>
<td></td>
</tr>
<tr>
<td>There is no advantage (and potential disadvantages) in continuing antibiotic cover after the operation</td>
<td></td>
</tr>
<tr>
<td>The antibiotic chosen must be effective against the expected contaminating organism</td>
<td></td>
</tr>
</tbody>
</table>
**MANAGEMENT OF WOUND INFECTION**

- **Suspect the diagnosis** if there is an otherwise unexplained postoperative fever or leucocytosis.
- **Inspect the wound regularly** for the signs of inflammation described by Celsus in AD 30, namely heat, redness, swelling and pain.
- Whenever possible **collect any pus or exudate** and take a wound swab for bacterial culture and sensitivity.
- Only if the patient is systemically ill, or the consequences of infection serious, should antibiotics be started blindly. Otherwise await identification of the organism and then give an antibiotic to which it is sensitive.
- An exception to this is rapidly spreading erythema and tenderness around a wound, signs which indicate a streptococcal infection. In this event there will be no exudate or pus formation and usually a negative wound swab, so treatment with **penicillin** in adequate doses should be started at once.
- **Drain** small abscesses by removing one or more of the skin sutures.

With major wound infection a drastic but effective measure is to return the patient to the operating theatre and **lay the wound open** down to the deep fascia. The wound may subsequently be allowed to granulate or may be re-sutured once it is clean.

Synthetic material in the wound may act as a nidus of infection which will not drain fully and will not be penetrated by antibiotics. It may be necessary to **remove all synthetic material** before the infection will resolve, and this may result in failure of the operation, e.g. a hernia repair.

**ABSCESES**

An abscess is defined as a discrete collection of pus usually surrounded by granulation tissue. To occur it is necessary to have an infection with an organism, such as *Staphylococcus aureus*, which produces toxins that cause local tissue necrosis which then liquefies – the pus. Pus contains bacteria, dead tissue and protein-rich inflammatory exudate. The wall of an abscess is not a definite layer but consists of inflamed compressed surrounding tissue. As the compression increases there is more ischaemic necrosis, which makes the abscess bigger. If the abscess is superficial, it will enlarge until it meets the skin. The skin is more resistant than subcutaneous fat but if it slowly undergoes necrosis, a dark area appears through which the abscess will eventually burst. This process is known as ‘pointing’ (see Chapter 5).

**Natural history of an abscess**

An abscess may:

- **burst**, either externally, or internally into a hollow viscus or a serous cavity
- **become chronic**
- **resolve** spontaneously.

The likelihood of each event depends upon the infecting organism and the situation of the abscess. A superficial abscess such as one in an infected sebaceous cyst is likely to ‘point’ and burst through the skin. An intra-abdominal abscess such as an appendix abscess may burst into the adjacent right colon or through the overlying abdominal wall. Rapid resolution of the symptoms and signs follows rupture.

A small abscess in the abdomen may be observed to resolve on serial scanning.

The correct management of an abscess is to drain it (Table 3.2).

---

**Table 3.2**

**General principles of management of an abscess**

<table>
<thead>
<tr>
<th>Principle</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remember</td>
<td>the core of an abscess is dead and not perfused with blood; therefore, antibiotics will not penetrate into it</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>may be needed if there is septicaemic toxicity</td>
</tr>
<tr>
<td>An external abscess</td>
<td>may be allowed to point before drainage</td>
</tr>
<tr>
<td>An internal abscess</td>
<td>needs to be localized with ultrasound or computed tomography scanning</td>
</tr>
<tr>
<td>There are three definitive treatments:</td>
<td></td>
</tr>
<tr>
<td>- surgical drainage</td>
<td>externally but sometimes into a hollow viscus</td>
</tr>
<tr>
<td>- serial aspiration</td>
<td>often with image guidance</td>
</tr>
<tr>
<td>- image-guided continuous drainage</td>
<td>by inserting a catheter for continued drainage and aspiration until the cavity obliterates</td>
</tr>
</tbody>
</table>
The management of the many specific varieties of abscess is discussed in the appropriate chapters. With the advent of safe image guidance and ultrasound scanning, abdominal and other internal abscesses are now more often drained by an interventional radiologist than a surgeon.

SEPTIC SHOCK

Septic shock is a state of profound tissue hypoperfusion and failure of oxygen delivery brought about by severe infection and sepsis. It has a mortality of around 50 per cent and is more likely to occur in elderly and immunocompromised patients.

Investigation

Clinical diagnostic indicators

For the diagnosis of septic shock there must be:

- evidence of circulating bacteria, usually confirmed by a positive blood culture (virtually any human pathogen may be responsible)
- persistent and refractory hypotension.

There will also be tachycardia and hyperventilation.

A feature of septic shock is arterial vasodilatation. The patient may feel warm and have bounding pulses yet be hypotensive.

The clinical end result is often multi-organ failure, i.e.

- acute renal failure caused by tubular necrosis
- acute lung injury leading to adult respiratory distress syndrome (ARDS)
- deteriorating liver function
- gastrointestinal tract damage, allowing translocation of bacteria into the blood stream which adds to the septic burden
- encephalopathy, manifest as confusion.

Blood tests

Paradoxically although there may be fever and a raised leucocyte count, both of these may be low because the sepsis overwhelms the immune system.

The pathophysiology of septic shock is complex. There is an exaggerated inflammatory response brought about by inflammatory mediators released by bacteria. These include cytokines such as tumour necrosis factor (TNF), interleukins and many others. The complement system is activated. The inflammatory cytokines usually induce disseminated intravascular coagulopathy.

Management

The treatment of septic shock is set out in Table 3.3.

Table 3.3

<table>
<thead>
<tr>
<th>Treatment of septic shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early recognition based on clinical suspicion</td>
</tr>
<tr>
<td>Adequate and immediate intravenous antibiotics</td>
</tr>
<tr>
<td>Vigorous resuscitation with replacement of circulating volume</td>
</tr>
<tr>
<td>Identification of the source of sepsis, usually clinically</td>
</tr>
<tr>
<td>Definitive treatment of the source of sepsis, which must be carried out as soon as resuscitation has been achieved</td>
</tr>
<tr>
<td>Support for failing organs</td>
</tr>
</tbody>
</table>

TETANUS

Tetanus is caused by Clostridium tetani, a Gram-positive, spore-forming, anaerobic organism found everywhere, particularly in soil. It produces a neurotoxin that prevents the release of inhibitory neurotransmitters that cause muscle spasm.

Tetanus is a rare disease in the UK, but not in the developing world. It has been virtually eradicated by immunization of the population with tetanus toxoid.

Investigation

Clinical diagnostic indicators

The clinical features of facial muscle spasm producing the ‘risus sardonicus’ and back spasms producing opisthotonos are diagnostic. Death is caused by respiratory failure.

The neurological complications appear several weeks after the patient sustains a wound. Diagnosis is clinical and difficult, as few doctors have seen a case. The original contaminated entry wound, the clue to suspecting the diagnosis, has usually been dealt with satisfactorily weeks before.
Management
The treatment of tetanus includes:

- **Drug-induced muscle paralysis to allow long-term ventilation** if the respiratory muscles are paralysed, until the toxin is eliminated. This may take a few of months, and has its own complications, particularly in older people. The mortality is around 40 per cent.

- **Wound excision.** Any dead tissue in the wound should be excised followed by delayed closure.

- **Penicillin.** Although *Clostridium tetani* is sensitive to penicillin, it has no effect on the neurological complications that are caused by neurotoxins.

- **Passive immunization.** Any un-immunized patient who sustains a high-risk wound should be passively immunized by giving, classically, antitetanus serum raised in horses, but nowadays immunoglobulin.

**GAS GANGRENE**

The pathogens causing gas gangrene are *Clostridium welchii* from soil or *Clostridium perfringens* found in normal gut flora. These anaerobic organisms produce toxins that cause necrosis and liquefaction of any ischaemic or poorly perfused tissues and a distinctive foul-smelling gas consisting mostly of nitrogen, with a small amount of hydrogen.

**Investigation**

**Clinical diagnostic indicators**

A clostridial infection occurs where there is dead tissue and often some degree of immunocompromise, e.g. the stump of a diabetic after an amputation.

There are the usual signs of wound infection but the patient is disproportionately ill. Septic shock is common. There may be evidence of gas in the tissues, manifest as a cracking sensation in the affected tissues when the area is palpated (surgical emphysema).

**Microbiology**

*Clostridium welchii* or *Clostridium perfringens* may be cultured from excised tissue or wound exudates.

These organisms may also be seen on a fresh microscopic specimen of the dead tissues.

**Imaging**

Gas may be seen in the soft tissues as radiolucent areas on X-rays (Fig 3.1).

**Management**

- **Penicillin** Clostridial organisms are sensitive to penicillin, which should be given as prophylaxis in any situation where anaerobic infection is possible. In established cases the principles of treatment are to give high doses of **intravenous benzypenicillin**.

  - **All dead tissue should be removed**, at whatever cost. This may require a major limb amputation or massive debridement.

  - **Hyperbaric oxygen**, given in a national centre in an appropriate chamber, to increase tissue oxygen perfusion and palliate the effect of the clostridial toxins, may have a role.

  Mortality is high. Most patients with gas gangrene die, whatever is done. This emphasizes the importance of prophylaxis (Table 3.4).

![Figure 3.1 CT scan showing gas in the tissues of gas gangrene (dark area)](image-url)

**Table 3.4 Management of gas gangrene**

Give prophylactic penicillin where there is any risk. If it does occur:

- give high-dose penicillin
- cut out all dead tissue
- consider hyperbaric oxygen
INTERSTITIAL INFECTIONS

There are a number of infections of the skin and subcutaneous tissues that have in common the involvement of a streptococcus and the ability to spread diffusely without becoming walled off to form an abscess.

Streptococcal cellulitis (erysipelas)

Investigation

Clinical diagnostic indicators

This pure streptococcal infection of the skin and subcutaneous tissues is characterized by redness, heat and tenderness (see Chapter 5 and Symptoms and Signs). A raised edge of the inflamed area caused by oedema of the subcutis is a reliable diagnostic feature. The entry site is rarely seen.

Erysipelas is the old name for streptococcal cellulitis.

Microbiology

There is rarely any exudate to culture but blood should be taken for culture. There may be no growth as the organisms may not have reached the circulation.

Management

Intravenous benzylpenicillin, to which streptococci are sensitive, should be commenced as soon as possible. Extension or regression of the infection may be monitored by marking the edge of the inflamed area.

Streptococcal cellulitis usually responds rapidly to penicillin.

SYNERGISTIC GANGRENE

This is a serious condition with a high mortality. There is infection with a streptococcus combined with an anaerobic organism such as a bacteroides.

The incidence is higher in those immunocompromised by conditions such as cancer, HIV infection, diabetes or immunosuppressive drugs. The combined infection produces toxins that cause tissue necrosis, which in turn acts as a culture medium for the responsible bacteria. The streptococci then flourish and spread, leading to a rapid advance of the process along tissue planes. A characteristic feature is necrosis of the fascia, usually with sparing of the underlying muscle (Fig 3.2). There is sometimes an obvious cause, such as local trauma or an infected wound, but sometimes the aetiology is obscure.

The condition has numerous synonyms:

- Fournier’s gangrene, when the condition arises in the scrotum
- Meleney’s gangrene
- necrotizing fasciitis
- hospital gangrene
- ‘flesh-eating bugs’ (tabloid press).

The prognosis is poor and the mortality as high as 80 per cent, not surprising in an unfit population. The survivors tend to be young with a clearly defined and treatable cause.

Investigation

Clinical diagnostic indicators

Diagnosis is initially clinical. The patient is always very unwell, with signs of septicaemia. The local signs are a dusky erythema of the skin, which may turn purple and become blotchy. The underlying necrosis of fascia and muscle is always much more widespread than the skin changes suggest.

Microbiology

Investigation should include serial blood cultures.
Management

The treatment is similar to that for gas gangrene, given above (Table 3.4), namely:

- High doses of the appropriate antibiotics, always including penicillin to cover streptococci, and metronidazole to cover anaerobic organisms.
- Total surgical excision of the necrotic areas. This may be impossible if the process is extensive. Such patients invariably die. Large areas of skin may have to be removed, and survivors may need extensive tissue reconstruction. Failing to excise all dead tissue inevitably leads to further spread of the infection.
- Hyperbaric oxygen may have a role for patients who have had a surgical excision. It increases tissue viability and reduces the likelihood of further necrosis.

ACQUIRED HOSPITAL INFECTIONS

Two varieties of infection are found almost exclusively in hospitals because of the extensive use and abuse of antibiotic therapy over the past 50 years. Methicillin-resistant Staphylococcus aureus (MRSA) is a strain of staphylococcus that has developed resistance to most antibiotics, and has prospered by natural selection. Clostridium difficile is an organism, sometimes carried in the normal gut, which becomes a dangerous pathogen when broad-spectrum antibiotics alter the balance of the flora in the intestine.

METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

When penicillin, the first antibiotic of the beta-lactam group, was introduced in the 1940s it was found to be dramatically effective against infections caused by Staphylococcus aureus, but even then there were some strains of the organism that had the ability to produce beta-lactamase, an enzyme which destroys penicillin. These gradually became predominant and widespread as penicillin was overprescribed.

In the 1960s methicillin, a new beta-lactam antibiotic that was not destroyed by the enzyme, was introduced. Unfortunately there were some strains of staphylococcus that were resistant to methicillin and these also became widespread, particularly in environments where penicillin and methicillin were being widely used, i.e. hospitals.

Methicillin requires parenteral administration and has been replaced by flucloxacillin, which is well absorbed when taken orally. However, the term methicillin-resistant Staphylococcus aureus (MRSA) has persisted. It is now used to describe any Staphylococcus aureus resistant to a beta-lactam antibiotic (‘multiple resistance’).

Staphylococcus aureus is found in the nostrils and on the skin of approximately a quarter of the population. Patients bring it into hospital with them and many hospital staff are carriers. The organisms are transferred very easily from staff to patient and from patient to patient, usually by hand contact. In the population outside hospitals, only 2–3 per cent of nasal and skin staphylococci are resistant to methicillin, but in hospital staff resistance rates have been reported of 10 per cent.

Most patients with MSRA are therefore colonized by it rather than infected by it and it is only pathogenic in certain circumstances. MSRA infections are found in patients with the risk factors set out in Table 3.5.

The site of infection may be anywhere in the body. It has been argued that most of the morbidity in patients with MSRA infection is related

<table>
<thead>
<tr>
<th>Table 3.5</th>
<th>Risk factors for methicillin-resistant Staphylococcus aureus infection in hospital patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Immunosuppression</td>
</tr>
<tr>
<td></td>
<td>Burns</td>
</tr>
<tr>
<td></td>
<td>Open wounds</td>
</tr>
<tr>
<td></td>
<td>Surgical drains</td>
</tr>
<tr>
<td></td>
<td>Urinary catheter</td>
</tr>
<tr>
<td></td>
<td>Invasive monitoring</td>
</tr>
<tr>
<td></td>
<td>Admission to an intensive care unit</td>
</tr>
<tr>
<td></td>
<td>Increasing age</td>
</tr>
<tr>
<td></td>
<td>Previous or prolonged hospital stay</td>
</tr>
<tr>
<td></td>
<td>Antibiotic treatment, particularly when prolonged</td>
</tr>
</tbody>
</table>
to the underlying condition that introduces the risk of infection by what are normally commensal organisms. However, those studies that have been adjusted for the underlying disease present convincing evidence that MRSA infection alone increases mortality and prolongs hospital stay.

**Diagnosis of MRSA infection**

This is usually made on routine culture of the infected area. There should always be suspicion of an MRSA infection if any of the risk factors are present.

**Treatment of MRSA infection in a patient**

Various antibiotics are available. Vancomycin and tigecycline are glycopeptide antibiotics that require parenteral administration. Both have toxic side-effects. Regrettably, resistance to these drugs has been seen and new agents and regimens are under development.

**Treatment of MRSA in an institution**

- Isolate infected patients, using barrier-nursing techniques with gowns and masks.
- Transfer any infected patient to their home at the earliest opportunity, if practicable, or to a specialized unit with isolation facilities.
- Seek the source of the infection by obtaining nasal swabs from the staff. Those infected may be treated with topical mupirocin and should be excluded from the hospital until they are shown to be clear of MRSA.
- Educate hospital staff in the importance of hygiene and hand washing after every patient contact.
- Consider screening patients before admission.

**Prevention of MRSA infection**

Although MRSA is endemic in large hospitals in Europe and North America, its incidence is much lower in those institutions which have:

- single rooms for patients
- washing facilities beside every patient and in every examination and treatment area

- **high standards of cleaning** of carpets, furniture and fabrics
- **lower occupancy rates** that allow time for rooms to be properly cleaned between patients.

The student will realize that the implementation of many of these measures requires the application of an ever vigilant and unrelenting clinical discipline.

**CLOSTRIDIUM DIFFICILE**

*C. difficile* is an anaerobic toxin-producing spore-forming organism found in about 2 per cent of healthy individuals in the community. It is found in the faeces of about 20 per cent of hospital inpatients, very few of whom have symptoms. The spores are omnipresent in the hospital environment and spread by hand contact. When antibiotics are given, the normal bowel flora alters in a way that favours *C. difficile* proliferation. About one in 200 inpatients develop symptoms of severe diarrhoea, with the incidence being higher in elderly people (Table 3.6).

The antibiotic-induced bowel disease caused by *C. difficile* is commonly called *pseudomembranous colitis* (PMC) because of the macroscopic and microscopic appearance of the exudate found in many cases. Koch's postulates proving *C. difficile* to be the causative agent were first fulfilled in 1977. The condition existed before that and was attributed to staphylococcal infection.

The mortality is significant, up to 30 per cent, although it is difficult to separate it from that associated with the underlying condition, as with MRSA. The most dangerous complication is toxic

<table>
<thead>
<tr>
<th>Table 3.6 Diagnosis of Clostridium difficile infection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical</strong>, with a high index of suspicion in those who develop diarrhoea and have a history of exposure to antibiotics</td>
</tr>
<tr>
<td>Repeated testing of faeces for the two toxins A and B</td>
</tr>
<tr>
<td>Direct culture is difficult (hence the name) and rarely necessary</td>
</tr>
<tr>
<td>Flexible sigmoidoscopy with biopsy is occasionally useful</td>
</tr>
</tbody>
</table>
facilities for its staff and high standards of domestic hygiene with adequate time for cleaning between patient discharge and the next admission.

**BLOOD-BORNE VIRUS INFECTIONS**

**HUMAN IMMUNODEFICIENCY VIRUS**

HIV is the cause of acquired immune deficiency syndrome (AIDS). It is a retrovirus that, by various mechanisms, attacks cell-mediated immunity. The virus is active in the circulation and in seminal fluid.

The highest risk of transmission is from a transfusion with infected blood. Many sufferers from haemophilia who were given frequent blood transfusions died of this disease before it was properly recognized and infected blood and blood products eliminated.

At least one-quarter of children born to infected mothers acquire the disease at birth. In developed countries these methods of transmission have been virtually eliminated.

The two other routes of infection are from needle sharing by abusers of intravenous drugs, and by sexual intercourse. Anal intercourse carries a much higher risk of transmission than vaginal intercourse. The risk from the latter is increased by violence and the co-existence of other sexually transmitted infections. Further discussion of the epidemiology, treatment and prevention of AIDS is beyond the scope of this book. There is no cure or vaccine for HIV infection, but with modern treatment the interval between the infection and the development of AIDS can be prolonged.

HIV infection compromises the immune response and is a risk factor for all infections.

AIDS presents to the surgeon in many and varied ways and should be suspected in any patient in any of the risk groups. The manifestations fall into three main areas:

- **Common infections which become refractory or recurrent** after treatment, e.g. tuberculosis. The commonest type of surgery required in the UK for patients with AIDS is for perianal sepsis, accounting for one-quarter of all operations on those with the HIV infection.

---

**Table 3.7**

**Treatment of *Clostridium difficile* infection**

<table>
<thead>
<tr>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Withdraw the causative antibiotic, replacing it with another if necessary</td>
</tr>
<tr>
<td>Replace fluid and electrolyte losses</td>
</tr>
<tr>
<td>Isolate the patient</td>
</tr>
<tr>
<td>Treat with the appropriate antibiotics:</td>
</tr>
<tr>
<td>■ First line – metronidazole</td>
</tr>
<tr>
<td>■ Second line – vancomycin (given orally)</td>
</tr>
<tr>
<td>Look out for relapses requiring treatment</td>
</tr>
<tr>
<td>Be aware of the risk of toxic megacolon</td>
</tr>
</tbody>
</table>

*megacolon*, which is treated in the same way as when caused by other diseases (see Chapter 19).

The symptoms are diarrhoea, abdominal pain and, in half the cases, fever. There will always be a history of having taken antibiotics. The condition is particularly associated with broad-spectrum agents but has been connected with almost every antibiotic, including paradoxically some of those used to treat it. It is particularly common after treatment with lincomycin and clindamycin and has led to these agents being used only rarely. The interval between the administration of the antibiotic and the onset of symptoms is usually about a week, but can be shorter or much longer.

The treatment of *C. difficile* infection is set out in Table 3.7.

**Prevention of *Clostridium difficile* infection**

*C. difficile* infection is an antibiotic-related disease and will be less likely to occur if there is sensible and appropriate use of antibiotics, in particular stopping them after a standard course of treatment has been completed.

To develop the disease the gut must contain the spores. Most patients ingest these when in hospital. Isolate and barrier – nurse sufferers, with safe disposal of their faeces and proper cleaning of their environment.

Its frequency would be reduced and its spread curtailed if hospitals had single rooms, good washing facilities for its staff and high standards of domestic hygiene with adequate time for cleaning between patient discharge and the next admission.
Unusual infections particularly associated with AIDS, e.g. oesophageal candidiasis.

AIDS-related tumours, principally lymphomas and occasionally the classical Kaposi sarcoma.

HEPATITIS

Approximately 2 per cent of the population carry one of the various hepatitis viruses. The surgical importance lies in the risk that the surgeon may acquire the infection from an infected patient.

Hepatitis B antigen is found in the blood and body fluids of all carriers. The infection is spread by sexual intercourse, sharing needles by drug abusers, and at birth from an infected mother to her child. Immunization is available and effective.

Hepatitis C was known previously as non-A non-B hepatitis and is spread in a similar manner to hepatitis B. There is currently no vaccine available.

There are other varieties of viral hepatitis some of which only proliferate in the presence of hepatitis B infection.

Transmission of HIV and hepatitis to members of the surgical team

This risk can be summarized as:

- surgeon to patient – rarely if ever
- patient to surgeon – significant, depending on the patient’s infection.

Transmission of a blood-borne virus from a patient to a surgeon or any other member of the operating team requires a blood contaminated ‘sharp’ or ‘needlestick’ injury. ‘Sharp’ injury includes stabs with scalpels, cuts from broken glass and injuries from bone spicules and teeth.

There is no risk from inhalation or skin-to-skin contact, but the mucous membranes of the mouth or eyes are vulnerable. The surgeon may have a skin abrasion that has not been noticed which introduces a risk. The chances of transmission vary with the virus (Table 3.8).

It should be remembered that the hepatitis and HIV status of the patient will not be known in most cases. Clearly the risks to operating staff will vary with the demography of the local population but, nevertheless, the risk of contracting these diseases can never be discounted entirely.

General safety measures against virus infections

- Screening and immunization of operating theatre staff against the disease. This is currently available only for hepatitis B.
- General hygiene avoiding contact with blood and removing spilt blood rapidly.
- Universal use of gowns, gloves and other physical barriers.
- Occlusive dressings over minor wounds and abrasions.

Specific precautions in the operating theatre

- Dispose of ‘sharps’ immediately into strong containers.
- Avoid handling anything sharp – use instruments.
- Modify surgical technique so that suture needles are touched only by instruments and not by the fingers. This may not be possible in open abdominal surgery.
- Use blunt needles whenever possible, e.g. when closing the abdomen.
- Stop using sharp-edged instruments whenever possible; for example make incisions with diathermy and use stapling devices on bowel and skin.
- Embrace laparoscopic and other endoscopic techniques where, once the serous cavity is entered, there is a barrier between the surgeon and the surgical site.
- Cover the mouth and eyes. Masks do not reduce patient infections but they do reduce the risk to the surgical team of virus infection.

Table 3.8

Risk of hepatitis and human immunodeficiency virus (HIV) transmission following a sharp or needlestick injury

<table>
<thead>
<tr>
<th>Virus Type</th>
<th>Transmission Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B</td>
<td>1 in 3</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>1 in 30</td>
</tr>
<tr>
<td>HIV</td>
<td>1 in 300</td>
</tr>
</tbody>
</table>
**Action to be taken after an accidental needlestick or sharp injury**

After a significant incident, it may be appropriate to test the patient for HIV and hepatitis status so as to assess the degree of risk. Where the patient is known to carry the virus the measures available are:

- **passive immunization** with immunoglobulin (for hepatitis B)
- **post-exposure prophylaxis**: immunization for hepatitis B, antiretroviral drugs for HIV.

**Remember to file an official accident report.**

It should not need to be stated that the best way for the surgeon to avoid the small but potentially catastrophic risk of acquiring an incurable virus disease from a patient in the operating theatre is scrupulous care and good surgical technique.