INFECTIONS

**Bacterial Skin Infections**

Bacterial skin infections are very common, especially in children and persons with penetrating injuries, immune deficits, ischaemia etc. They are 'always' due to *Streptococcus pyogenes* (Group A β-haemolytic) or *Staphylococcus aureus*; very rarely they are due to enteric Gram-negative bacilli or anaerobes.

Cellulitis, erysipelas and lymphangitis are all basically the same thing; the distinction is cellulitis is not sharply demarcated, erysipelas is, and lymphangitis spreads along lymphatics. Classically the causative agent was thought to be *S. pyogenes*, but in fact *S. aureus* and other *Streptococci* are equally common.

1. Clinical features – spreading erythema (may blister), moderate pain, 'no' pus, systemic symptoms
2. Treatment – need to cover both *S. pyogenes* and *S. aureus*
   a. Flucloxacillin 250-500mg po qid, some use intravenous then oral penicillin (if severe)
   b. Erythromycin 500mg po q6h (if allergic to penicillin)
   c. Rest and elevation

**Folliculitis, boils, carbuncles and abscesses** are infections of hair follicles and damaged tissues. They are 'always' due to *S. aureus*.

1. Clinical features – central pus surrounded by erythema, pain
2. Treatment
   a. Drain pus and culture for MRSA
   b. Flucloxacillin 250-500mg po qid (vancomycin, clindamycin or fucidic acid for MRSA)
   c. Hand hygiene

Impetigo (‘school sores’) is a very common skin infection caused by *S. pyogenes* with *S. aureus*.

1. Clinical features – localised superficial lesions, honey-coloured crusts, no systemic symptoms
2. Treatment:
   a. Hygiene essential – soap and water and adequate drying
   b. Topical e.g. chlorhexidine or mupirocin, occasionally oral e.g. flucloxacillin 250-500mg po qid

**Fungal Skin Infections**

Fungal infections are a common and minor skin and mucosa problem – they are rare elsewhere, but may be serious. Mucous membrane infections are 'always' *Candida* albicans, skin rash is usually a dermatophyte or *Malassezia* furfur, and deep tissue infections are either *C. albicans* or Aspergillus (never dermatophytes).

1. Yeasts – single-cell organisms, round or oval, reproduce by budding
   a. *Candida albicans* – mucosal and skin infections, deep infections in immunocompromised
   b. *Malassezia furfur* – dandruff, seborrhoeic dermatitis, pityriasis versicolour
2. Moulds – multicellular filamentous organisms, tubular hyphae, reproduce by spores
   a. Dermatophytes (*Trichophyton sp, Epidermophyton sp, Microsporum sp*) → tinea
   b. *Aspergillus fumigatus* – lung and brain disease in severely immunocompromised

Four fungal infections:

1. Tinea of scalp, body, feet and onychomycosis is caused by dermatophytes.
   a. Clinical features vary from non-inflamed scaly areas to oedematous, vesicular lesions
   b. Skin – topical azole e.g. clotrimazole, econazole, ketoconazole, miconazole
   c. Nails – oral terbinafine (250mg po od x3-4/12) or itraconazole (400mg po od, 1 week in four, for 3-4 months)
2. Candidiasis may be due to overgrowth 2° to antibacterial therapy, immune suppression, hormonal effects or foreign bodies
   a. Clinical features – oral/vaginal ‘thrush’, cutaneous/nail, catheter-related bladder infection
   b. Treatment usually topical, sometimes oral – nystatin suspension or pastilles, amphotericin B pastilles, azole pessaries or cream
3. Pityriasis versicolour is probably the most common superficial mycosis, caused by *M. furfur*.
   a. Clinical features – hypopigmented finely scaled macules on chest, shoulders and back
Infectious Diseases

4. **Seborrhoeic dermatitis** is caused by *Pityrosporum* sp.
   a. Clinical features – greasy facial rash, dandruff
   b. Treatment with a topical azole

- **Septic Arthritis and Osteomyelitis**

**Bone and joint infections** may be infections in ‘native’ joints, infections in ‘normal’ bone, or infections associated with implanted devices (e.g. joint replacements, metal plates).

**Septic arthritis** is most common in children under 2 years of age. Differential diagnosis should include non-infectious inflammation (rheumatoid arthritis, gout) and other infections (e.g. rubella, mumps).

1. **Aetiology** – haematogenous spread most common, then local spread, then penetrating injury
   a. *S. aureus* 50%, *Streptococci* (Group B, pneumonae) 30%, Gram-negative bacilli 15%, others 5%
   b. By age – infants mostly *Staphylococci and Haemophilus*, children mostly *Staphylococci and Streptococci*, 16-50 years mostly *N. gonorrhoea*, >50 years mostly *Staphylococcus*

2. **Clinical features** – usually only one joint is involved (90%), usually large and weight bearing (knee 50%, hip 20%, ankle 10%, elbow 5%).
   a. History of pre-existing arthritis (50% adults, 0% children), trauma (30% adults and children), infection elsewhere (10% adults, 25% children), no risk factors (10% adults, 45% children)
   b. Symptoms – pain, inflammation (may not be apparent e.g. hip), fever (spiking or low-grade)
   c. Investigations – FBC (↑ESR ± ↑WBC), joint fluid (WBC >50,000/mm³, >75% PMNs, ↑protein and ↑glucose – latter two are non-specific; 33% Gram stain positive, 90% culture positive)
   d. Radiology – joint distension (may be sole finding), destructive changes (>2 weeks), CT/MRI

3. **Treatment:**
   a. Drain and lavage – may need to be repeated 2-3 times (prevents cartilage destruction)
   b. Empirical antimicrobial – e.g. flucloxacillin, cefazolin
   c. Definitive antimicrobial – IV for at least 1 week; 3 weeks total

4. **Prognosis:**
   a. Complications include avascular necrosis, contractures, degeneration, osteomyelitis, mostly in patients where treatment delay is >1 week
   b. <1% mortality, up to 30% have secondary morbidity

**Osteomyelitis** is an infection of bone, almost always bacterial, characterised by pain, loss of function and possibly severely deforming changes that may chronically recur for many years. It is more common and more virulent in developing nations, affecting mainly children (often with underlying predisposition).

1. **Classification:**
   a. Haematogenous (especially in children)
      i. *S. aureus, Streptococci*, Gram-negative bacilli
      ii. Most commonly long bones (femur, tibia, occasionally adjacent vertebrae)
      iii. Severe local tenderness, reduced mobility, fever
   b. 2° to injury (especially diabetic foot)
      i. Diabetic foot – *Streptococci and Staphylococci*; then mixed Gram-negative bacilli and anaerobes
      ii. Puncture wounds of feet – *Pseudomonas aeruginosa* (from shoe interior)
   c. Recurrent infection associated with dead bone (sequestrum)
      i. Follows non-curative treatment of acute osteomyelitis; inflammation spreads to periosteuim leading to suppuration, abscess and new bone formation (involucrum)
      ii. Organism is identical to original isolate but a variety will grow from sinus pus draining via cloacae from periosteuim

2. **Management:**
   a. Investigations - ↑ESR, X-ray (swelling, curious sequestra, diffuse decalcification if pyogenic, abscess), Tc bone scan (hot spot); blood cultures 50%, aspirate 60%, biopsy 90% positive
   b. Surgical – drainage of abscess/pus, debridement of bone, removal of sequestra
   c. Medical – antibiotics depending on culture; requires at least 6 weeks (3 weeks high dose IV)

**Prosthetic joint infection** occurs in about 1% of joints (hips and knees), most commonly due to *Staphylococcus* (coagulase positive and negative) but also due to *Streptococci, Enterococci* and Gram negative bacilli. Note that if the infection occurs within 2 years of insertion, it was probably introduced at the time of surgery

1. **Aetiology:**
   a. Early infection – surgical contamination; *S. aureus*
   b. Late infection – bacteraemia from other sources; *S. epidermidis*, Gram-negative bacilli
Infectious Diseases

2. Treatment:
   a. Removal of metalware is required – the joint can be replaced straight away with gentamicin cement and 6 weeks of antibiotics post-surgery, or a spacer can be used in the interim
   b. Antibiotics – vancomycin for coagulase-negative Staphylococcus.

• Infections in Travellers

Pre-travel advice – the six ‘I’s:
1. Injuries – commonest cause of death after IHD; remind travellers to be careful (avoid being obvious targets, use safe vehicles with seat belts, avoid night-time travel)
2. Insects transmit many diseases, bit with avoidance the only protection for many (e.g. dengue) – cover up, wear light colours, DEET-containing repellent, screen at night or permethrin bednet
3. Ingestion – water should be either boiled, carbonated or treated; food should be thoroughly cooked and still hot. Avoid tap water, ice, salads, buffets, raw or rare meat, or shellfish.
4. Immersion (especially schistosomiasis in Africa) – avoid rivers and lakes; if contact unavoidable can be serologically screened 3 or more months after exposure
5. Indiscretions – many STDs are more prevalent in the developing world; travel increases risk of sex with casual partners (remind travellers of risks and that condoms work)
6. Immobility – move around as much as possible, drink plenty of water, exercise calf muscles; moderate risk consider compression stockings, high risk consider low molecular weight heparin
7. Insurance – is a good thing

Fever in the returned traveller – always need to consider/exclude malaria; note that patients may have more than one problem which may not necessarily be tropical (e.g. UTI, pneumonia). Investigations should include malaria film (may need repeating), FBC/LFTs, blood cultures, urine or stool culture as indicated; appropriate serology (dengue, hepatitis, syphilis, Epstein-Barr, amoebic etc.)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Region</th>
<th>Incubation</th>
<th>Clinical</th>
<th>Lab</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malaria</td>
<td>Tropics</td>
<td>10 days to years</td>
<td>Splenomegal</td>
<td>Blood film</td>
<td>Quinine or chloroquine</td>
</tr>
<tr>
<td>Dengue</td>
<td>Pacific, Asia, Central &amp; Sth America</td>
<td>5 to 8 days</td>
<td>Muscle ache, rash, headache, back ache ‘break-bone fever’</td>
<td>Low platelets &amp; WBC, serology</td>
<td>Supportive</td>
</tr>
<tr>
<td>Typhoid</td>
<td>India, SE Asia</td>
<td>10 to 14 days</td>
<td>Constipation, drowsiness, headache, abdominal pain</td>
<td>Blood culture (S. typhi)</td>
<td>Quinolones; complications 10-20%, 2-5% carriers</td>
</tr>
<tr>
<td>Hepatitis A</td>
<td>Global</td>
<td>30 days</td>
<td>Fever, anorexia, nausea, abdominal discomfort (1-7 days); icteric phase 2 weeks to 2 months</td>
<td>HAV IgM</td>
<td>Supportive</td>
</tr>
<tr>
<td>Rickettsial</td>
<td>Various</td>
<td>Various</td>
<td>Fever, headache, myalgia, rash; painless eschar, lymphadenopathy</td>
<td>Serology</td>
<td>Doxycycline</td>
</tr>
<tr>
<td>Amoebiasis</td>
<td>Tropics</td>
<td>2 to 5 months</td>
<td>Bloody diarrhoea, pain; fever, RUQ tenderness</td>
<td>Stools, USS, CT, serology</td>
<td>Metronidazole</td>
</tr>
</tbody>
</table>

Traveller’s diarrhoea affects up to 50% of travellers in some countries, often in the first few weeks of travel. It is usually mild and self-limiting, usually caused by enterotoxigenic Escherichia coli (preventable by careful oral intake) but also less frequently by parasites and viruses.

1. Clinical features – 3-10 loose stools per day, abdominal pain and cramps, fever in 10-20%, vomiting in 10-20%; lasts over 1 week in 10%, over 1 month in 2%
2. Investigations – malaria film, FBC, LFT, blood culture, MSU, faecal culture, serology, CXR
3. Treatment:
   a. Oral rehydration with WHO formula or Gastrolyte
   b. Loperamide for mild-to-moderate diarrhoea
   c. Norfloxacin is severe e.g. high fever, bloody stools

Malaria is an acute and chronic protozoan infection transmitted by Anopheles mosquitoes to humans; it is widespread in tropical countries with major morbidity in Africa and parts of the Pacific (Papua New Guinea, the Solomon Islands and Vanuatu). Plasmodium vivax is the most common (never kills), followed by P. falciparum (sometimes kills), and rarely P. malariae or P. ovale.

1. Pathogenesis
   a. Female anopheles mosquito injects sporozoites into the bloodstream, which migrate to the liver and multiply there asymmetrically (hypnozoites in P. vivax and ovale stay for ages)
Infectious Diseases

b. Merozoites invade red blood cells and become trophozoites then schizonts; the red blood cells then rupture releasing many more merozoites → symptoms and periodicity

c. Some parasites in red blood cells develop into male and female gametocytes which are ingested by female mosquitoes; sexual cycle in mosquito gut produces sporozoites

2. Clinical features are due to immune response to the infection as well as cell lysis.

   a. *P. vivax* – incubates up to 12 months, relapses months after initial infection; characteristic periodic fever every 48 hours, malaise, headaches, swinging fever, chills, rigors, nausea

   b. *P. falciparum* – incubates 12-14 days (up to 3 months), high fever every 48 hours for more than 2/12; anaemia if large load, thrombocytopenia, vascular collapse, end-organ damage

   c. Investigations – ↑LFTs, ↑LDH (>50%), ↓WBC, ↓platelets, anaemia; malarial smear (examine RBCs looking for trophozoites or schizonts) repeated at 12-24 hour intervals

3. Treatment:

   a. Supportive – watch for thrombotic complications (especially *P. falciparum*)

   b. Treatment:

      i. *P. falciparum* – admit, give quinine and doxycycline (as chloroquine resistant)

      ii. *P. vivax*, others – chloroquine ± primaquine for hypnozoites

   c. Prophylaxis:

      i. Mosquito bite avoidance in the evening and nighttime

      ii. Chloroquine weekly (central America or middle east); mefloquine weekly, or daily doxycycline or malarone – atovaquone/proguanil (Africa, PNG, Solomons, Vanuatu)

      iii. Note risk is high in Africa and malarious Pacific; intermediate in India and variable depending on activities and location in SE Asia and South America.

SEXUALLY TRANSMITTED INFECTIONS

- Management of Sexually Transmitted Infections

General principles

1. Accurate diagnosis:
   a. History of presenting complaint
   b. Sexual and risk history
   c. Physical examination
   d. Microbiological and/or serological investigations

2. Effective treatment and prevention:
   a. Drug therapy
   b. Notification of sexual contacts
   c. Education regarding risk reduction (e.g. safe sex, contraception)
   d. Follow up – compliance, resolution, re-infection, notification, recurrence/prophylaxis

3. Common presenting symptoms:
   a. Urethral discharge ± dysuria, scrotal/testicular pain
   b. Vaginal discharge ± vulval irritation, pelvic pain
   c. Genital skin lesions, ulceration, lumps and rashes

Urethral discharge

1. History of presenting complaint:
   a. Duration, circumstances, previous occurrence
   b. Description – colour, consistency, volume, blood
   c. Associated symptoms – dysuria, urethral irritation, scrotal pain ± swelling (which side)

2. Sexual history – construct sequent for last 3 months
   a. Number, gender and type of partners (regular/casual) and whether symptomatic
   b. Type of sexual contact – vaginal, oral, anal (receptive/insertive), condom use
   c. Overseas travel/contact
   d. Associated risks – alcohol, drugs, past STI history

3. Examination:
   a. Confirm presence of discharge
   b. Associated findings – meatitis, scrotal/testicular mass or tenderness
   c. Other abnormalities – skin, mouth, nodes, joints, eyes

4. Investigations:
   a. Urethral Gram stain – quantify PMNL per HPF, is there presumptive gonorrhoea (Gram-negative intracellular diplococci)?
      i. Yes – treat for gonorrhoea and give empirical treatment for Chlamydia
      ii. No – treat for non-gonococcal urethritis (NGU)
   b. Specific – gonorrhoea culture, first-void urine for Chlamydia testing (nucleic acid amplification), may do serology
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5. Management:
   a. Gonorrhoea – single-dose anti-gonococcal therapy AND treatment for Chlamydia coinfection
      i. Presumptive gonorrhoea – ceftriaxone 250mg im
      ii. If sensitivity testing indicates sensitivity – ciprofloxacin 500mg po
   b. NGU or confirmed Chlamydia – doxycycline 100mg bd x7d OR azithromycin 1g po stat
   c. Risk reduction (behaviour change, condoms), notification of sexual contacts

Vaginal discharge
1. History of presenting complaint:
   a. Duration, circumstances (relation with period, LMP, pregnancy), previous occurrence
   b. Description – colour, consistency, volume, blood, malodour (c.f. normal physiological d/c)
   c. Associated symptoms – vulval irritation/pain, pelvic pain or deep dyspareunia, menstruation
   d. Others – contraception (COC, IUCD, diaphragm), tampon use or douching, antibiotic use
2. Sexual history – as per previous
3. Examination:
   a. Confirm presence and type of discharge – colour, consistency, odour, pH, blood
   b. Vulval signs – rash, ulceration etc.
   c. Cervix – ectopy, mucopurulent discharge, bleeding, ulceration
   d. Bimanual – cervical motion tenderness, uterine tenderness, adnexal tenderness, mass
4. Investigations:
   a. Vaginal/cervical fluid – Gram stain, wet smear, KOH smear (looking for PMNL, hyphae or yeasts, trichomonads, gonococci, bacterial vaginosis)
   b. Specific – vaginal culture (Candida, Trichomonas vaginalis), endocervical culture (Gonorrhoea), endocervical swab (Chlamydia nuclear amplification), cervical cytology (Pap)
5. Management:
   a. Treat for specific conditions, modify if pregnant:
      i. Candidiasis – antifungal (topical or systemic)
      ii. Bacterial vaginosis – nitroimidazole
      iii. Trichomoniasis – nitroimidazole (state dose)
      iv. Gonorrhea/chlamydia – see urethritis management plan
   b. Address risk factors (hygiene, no douching, reduce physical and chemical trauma, exclude background disorder) and notify contacts if STI (trichomoniasis, Gonorrhoea, Chlamydia)

Genital skin lesions:
1. History of presenting complaint – ulcer, lump or rash
   a. Description – number of lesions, changing?, irritation, pain
   b. Duration, relationship with intercourse, previous occurrence (pattern of recurrence
   c. Other symptoms:
      i. GU – vaginal/penile discharge, inguinal tenderness, urinary symptoms, pelvic pain
      ii. Extranagenital – joint pain, leg pain, fever, headache, myalgia
   d. Other medical conditions (diabetes, skin disorder), pregnancy, contraception, smoking
2. Sexual history – as per previous
3. Examination:
   a. Genital skin – also perianal, perineum, anorectal, cervix and vagina
   b. Extragenital skin, eyes, joints, mouth
4. Investigations:
   a. Ulcer-specific – HSV culture, dark field/direct fluorescence and serology for syphilis (known contact, sex overseas or recent immigrant), swabs (bacteriology, yeasts if appropriate)
   b. Others – swabs (bacteriology, yeasts), skin scraping (fungi), biopsy, Pap smear
5. Treatment:
   a. Ulcer:
      i. HSV – acyclovir 200mg 5 times per day for 5 days
      ii. Syphilis – refer for specialist management
   b. Lump:
      i. Genital wart – ablative therapy
      ii. Molluscum contagiosum – ablative therapy
      iii. Abscess – antibacterial, lance
      iv. Scabies – scabicide
   c. Rash:
      i. Pubic lice – miticide
      ii. Scabies – scabicide
      iii. Fungal – antifungal (topical or systemic)
      iv. Dermatological condition (e.g. eczema, psoriasis) – topical steroid
Infectious Diseases

HIV Infection

HIV infection in the past led inexorably to AIDS, and survival after diagnosis of AIDS was measured in months. The introduction of highly active anti-retroviral therapy in 1997 led to a complete change in prognosis – HIV replication could be controlled, and AIDS and death were delayed (or potentially prevented).

1. Pathophysiology:
   a. After infection, the level of HIV in tissues and blood rises – however many HIV-producing cells are killed by cytotoxic T lymphocytes (causing a brief seroconversion illness)
   b. Number of T-helper lymphocytes in blood falls, levels of HIV in blood falls, and B lymphocytes start producing antibodies; HIV levels remain stable for several years
   c. T-helper lymphocyte numbers continue to fall until depletion is severe leading to presentation of AIDS illnesses and increasing levels of HIV in blood

2. Presentations:
   a. Seroconversion illness – 1-2 months after infection, similar to glandular fever (fever, sore throat, lymphadenopathy, rash, aseptic meningitis)
   b. Asymptomatic – prior risk behaviour (e.g. homosexual, heterosexual in 3rd world country)
   c. Early illnesses – oral candidiasis, dermatophyte, shingles, TB, persistent lymphadenopathy
   d. Late illnesses – *Pneumocystis carinii* pneumonia, cerebral toxoplasmosis, cryptococcal meningitis, *Mycobacterium avium* bacteraemia, CMV retinitis, Kaposi’s sarcoma, lymphoma

3. Diagnosis:
   a. Clinical suspicion based on epidemiology and presentation
   b. Absolute lymphopenia (<1.0x10^9/L) may be present
   c. HIV antibody test – very sensitive/specific, needs informed consent (infection carries stigma)
   d. Tests for secondary illness – microbiology, histopathology etc.
   e. Advise about HIV testing for past contacts, and counselling to prevent future transmission
      i. Man-to-man unprotected sex risk – 1:20 to 1:1,000
      ii. Man-to-woman or woman-to-man unprotected sex risk – 1:100 to 1:1,000
      iii. Sharing infected injecting equipment – 1:100
      iv. Needle-stick injury – 1:300

4. Prognosis:
   a. Without treatment average duration of infection to AIDS is 10 years, and before current treatments average survival after diagnosis of AIDS was 2 years
   b. Most opportunistic infections can be clinically cured then suppressed; however unusual illnesses (CNS lymphoma, progressive multifocal leukoencephalopathy) may be incurable
   c. The helper lymphocyte (T4, CD4) count is the best predictor of risk of AIDS or other opportunistic infections within the next few months
   d. The HIV viral load (RNA copies/mL) is the best predictor of the rate of T4 count decline over the next months to years, and is the best predictor of risk of AIDS in the next few years

Treatment of HIV infection:

1. General principles:
   a. Routine care involves an outpatient clinic appointment every 3-6 months with a CD4 lymphocyte count (cost $160) or HIV viral load (cost $425)
   b. Treatment aims to lower viral load and replication to undetectable levels in order to prevent resistance to the antiretroviral agents (otherwise resistance develops within months)
   c. Long-term suppression of viral replication is associated with recovery of T4 count, reduction in risk of AIDS diseases and return to good health.

2. Anti-retroviral drugs – usually 2 reverse transcriptase inhibitors plus 1 protease inhibitor or 1 NNRTI
   a. Reverse transcriptase inhibitors (RTIs)
      i. Nucleoside analogues (NRTIs) e.g. zidovudine = azidothymidine (AZT)
      ii. Enzyme deforming (non-NRTIs) e.g. nevirapine and efavirenz
   b. Protease inhibitors (PIs) e.g. indinavir, nelfinavir
   c. Future drugs – inhibitors of HIV binding, DNA integration, protein assembly and packaging

3. Treatment issues:
   a. Resistance:
      i. Due to selection pressure – HIV replication in the presence of drugs
      ii. Inevitable consequence of inadequate compliance (<95% of doses taken)
      iii. Associated with mutations in reverse transcriptase or protease genes
   b. Adverse effects:
      i. Lipodystrophy – fat redistribution (severe cosmetic effects)
      ii. Hyperlipidaemia – expect vascular effects
      iii. Others – e.g. mitochondrial toxicity
   c. Materno-fetal transmission – risk is 20-40% without treatment
Infectious Diseases

i. Transmission occurs during pregnancy, at delivery and during breastfeeding
ii. Treatment of the mother during last months of pregnancy, delivery by elective caesarean and treatment of the infant in the first 6 weeks of life reduces risk to <1%

**IMMUNITY AND INFECTION**

• Immunisation

**Classification of common vaccines:**

1. Whole-cell bacterial – pertussis vaccine, typhoid vaccine, BCG
2. Toxoids – tetanus, diphtheria, poliomyelitis, measles, rubella
3. Capsular polysaccharide – pneumococcal, meningococcus, *H. influenzae* type B

<table>
<thead>
<tr>
<th>Non-replicative (killed/inactivated)</th>
<th>Replicative (live attenuated)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example</td>
<td></td>
</tr>
<tr>
<td>Bacterial (DTP), some viral (IPV, influenza)</td>
<td>Viral (MMR, OPV, yellow fever)</td>
</tr>
<tr>
<td>Principle</td>
<td></td>
</tr>
<tr>
<td>Preformed antigenic mass</td>
<td>Self-replicative</td>
</tr>
<tr>
<td>Passive antibody</td>
<td></td>
</tr>
<tr>
<td>No inhibitory effect</td>
<td>May prevent immunisation</td>
</tr>
<tr>
<td>Duration</td>
<td></td>
</tr>
<tr>
<td>Relatively short, requires boosters</td>
<td>Relatively long (mimics natural infection)</td>
</tr>
<tr>
<td>Main mechanism</td>
<td></td>
</tr>
<tr>
<td>Humoral IgG</td>
<td>Humoral IgG, local IgA</td>
</tr>
</tbody>
</table>

In New Zealand, there is currently no population-based strategy to ensure that individuals are registered for vaccination and tracking, or to provide reliable coverage details to avoid pockets for future epidemics. We have failed to progress compared with Australia, the UK or the USA and epidemics continue as coverage is low in the general population, and even lower in low socioeconomic areas.

See relevant paediatrics tutorial for information about specific diseases, and the immunisation handbook for details about immunising under special circumstances (page 19), the ID slides are really hard to decipher.

• Infection in the Immunocompromised Host

The **immune system** helps to prevent infection progressing to disease, and hastens recovery from infection. Deficits increase the frequency and severity of disease – hence strategies to prevent disease, and effective treatment of infection, are especially important in patients with impaired immunity

**Deficits in immunity:**

1. Less immunocompetent tissues
   a. CSF has no antibody, acute phase proteins or leukocytes
   b. Heart valves – neutrophils are rarely found in endocarditis vegetations
2. Damaged barriers to infection – increase risk of infection
   a. Burns and wounds disrupt normal skin defences
   b. Mucositis enhances entry of oral flora
   c. Invasive devices provide a pathway for infection – e.g. urinary catheter, ETT, IV cannulae
   d. Cystic fibrosis is associated with reduced clearance of secretions
3. Implanted devices – prevent eradication of infection
   a. Joint replacements, prosthetic heart valves
   b. Almost impossible to cure, though treatment usually leads to clinical improvement
4. Congenital, acquired or iatrogenic defects in systemic immunity:
   a. Some rare deficits of neutrophil function cause fatal childhood disease
   b. Common variable immune deficiency leads to progressive decline in antibody production
   c. HIV infection leads to loss of helper T lymphocytes
   d. Treatment with steroids or immunosuppressive agents impairs cellular immunity, while cytotoxic drugs decrease neutrophil and lymphocyte numbers

**Management of infection in the immunocompromised host:**

1. Consider immune deficiency
   a. Why this unusual infection?
   b. Why recurrent infections?
   c. Why now?
2. What deficit is it?
   a. Local – e.g. recurrent UTI 2° to indwelling catheter
   b. Impaired cellular immunity – e.g. recurrent intracellular protozoal, bacterial or viral infections
3. **Rapid, aggressive efforts to diagnose infection and identify the organism**
   a. Identify the site if possible – symptoms and signs are often due to the immune response, so patients with immune deficiencies may have much less marked features (trivial findings help)
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b. Early use of invasive procedures to obtain specimens, alert the laboratory to the possibility of unusual pathogens (which shouldn’t be discounted as contamination/insignificant)

4. Early broad spectrum antimicrobial therapy with maximal efficacy
   a. Perform initial investigations rapidly
   b. Empiric treatment – broad spectrum, adequate levels
   c. Extra attention to physiological support – oxygen, fluids etc
   d. Consider correction of the immune compromise – catheter out, Ig infusion, G-CSF

Febrile neutropenia (T >38.3°C or T>38°C for 1 hour) is due to an altered inflammatory response, giving a modified presentation. There has been a shift in pathogens over the last 2-3 decades, and anatomical source/site may vary. Risk generally increases with decreasing neutrophil count.

1. Empiric antimicrobials – wide variation of agents, local sensitivity should influence decision
   a. e.g. cefpirome plus genamicin used for febrile neutropenia in ADHB adult haematology
   b. Amphotericin B and/or vancomycin may be added if fever persists

2. Vascular access devices:
   a. May leave if infection due to coagulase negative Staphylococcus or S. aureus
   b. Removal advisable – bacteraemia (Bacillus sp, P. aeruginosa, C. jeikeim, Candida, Acinetobacter sp, Stenotrophomonas)
   c. Removal essential – tunnel infection, port infection, emboli, blocked catheter

3. Antibiotic prophylaxis:
   a. TMP/SMX, fluoroquinolones – effective in reducing infective episodes but resistance
   b. Fluconazole – effective in bone marrow transplant patients
   c. Itraconazole – emerging efficacy data