**Infections from the Playing Environment (Water or Soil)**

With the exception of tetanus (B. Watt — vide infra), the danger of infection from the playing environment is more theoretical than real. In almost all instances infection occurs because the area of activity is contaminated with infected animal (e.g. dog or rat) and less frequently human, excreta. Examples of such infections include salmonellosis, leptospirosis, giardiasis and cercarial dermatitis and schistosomiasis, all of which have been recorded in participants in water sports.

**Reference**


**TETANUS**

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Tetanus is a serious, potentially fatal infection caused by *Clostridium tetani*. While uncommon in the UK, it is a common cause of death worldwide. It can be prevented by encouraging active immunisation of the population.

*C. tetani* is an obligately anaerobic bacillus that has two important properties — it forms spores and produces a potent neurotoxin. The spores are non-metabolising forms of the organism that can survive adverse conditions of temperature, dryness and pH, as well as being resistant to many antiseptics and disinfectants. They are found in human and animal faeces and can, therefore, easily contaminate soil and dust such as a sports field. The neurotoxin, (tetanospasmin), is a simple protein of molecular weight 150,000 and like other clostridial toxins, is extremely potent; the minimum lethal dose for man is only one nanogram.

Tetanus occurs when spores contaminate a wound in which oxygen levels are low. Such low levels could occur as a result of other bacteria contaminating the wound and "using up" available oxygen, and especially so in deep penetrating wounds such as those caused by thorns, splints, nails or sports equipment or in the presence of tissue necrosis. Under such conditions, the spores revert to vegetative cells which produce tetanospasmin. The toxin, formed at the site of infection, is rapidly transported along peripheral nerves to the central nervous system. There it binds easily to specific ganglioside receptors and acts by inhibiting the secretion of certain neurotransmitter substances.

After a variable incubation period of a few days to two or three weeks, the patient complains of stiffness of the jaws followed by progressive difficulty in swallowing (hence the name “lockjaw”). This may progress to a general increase in muscle tone, followed by the onset of generalised muscle spasms that are initially triggered by external stimuli but latterly occur spontaneously. The patient may develop pneumonia and cardiovascular instability. Death rates for established tetanus vary from 15-70%. It should be noted that even in fatal cases, there may be little or no evidence of local infection — indeed the original site of infection may be impossible to find. It is generally believed that a short incubation period is associated with more severe disease. The subject is well reviewed by Smith (1984).

The main features of treatment relate to:

1. Early administration of antitoxin to neutralise any unbound toxin.
2. Attention to contaminated wounds.
3. Sedation, accompanied by elective muscle paralysis with curare-like drugs and by tracheostomy and artificial ventilation in severe cases.
4. Penicillin is often given concurrently to inhibit further growth (and therefore toxin production) of *C. tetani* at the site of infection. (Penicillin has no effect on the toxin itself.)

It should be noted that patients who recover from tetanus are not immune from further attacks and therefore require to be immunised.

**Prevention**

The administration of a course of "toxoid" (toxin rendered harmless but retaining its antigenicity) renders individuals immune from tetanus by stimulating the production of protective antitoxin. Such active immunisation should be encouraged in sportmen and sportswomen and anyone who comes into physical contact with soil, dust or animal or human faeces. Booster doses are required every 10 years, but if given more frequently there is a danger of developing hypersensitivity reactions. In the case of persons who present with a wound or with tissue damage, it is essential to have a clear policy about the prevention of tetanus. Measures to be used will depend on the type of wound and the immune status of the patient. An excellent schedule for wound management and tetanus prophylaxis is described by Collee et al. (1987).

**References**


**HERPES SIMPLEX, HEPATITIS B AND THE ACQUIRED IMMUNE DEFIENCY SYNDROME**

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Herpes, hepatitis B and AIDS are viral infections, to which increased attention has been given in recent years, not least in relation to sport.

**Herpes Simplex**

Common presentations are stomatitis which is generally a primary infection usually occurring during childhood with fever and toxicity in addition to the painful intra-oral lesions, or localised skin infection (usually reactivation). Reactivation most commonly presents as “cold sores” around the lips which are uncomfortable but not usually associated with systemic features. Reactivation can be precipitated by a variety of factors such as exposure to sun, wind or febrile illness. The genital form of infection similarly occurs in both primary (generally more severe) or reactivation forms.

Infection is transmitted from person to person by droplets or where skin surfaces are directly opposed as in rugby (“scrapox”) or in wrestling (“herpes gladiatorum”), although “exposure” itself may be the precipitating factors when reactivation is involved.

Prevention depends upon hygiene, ultraviolet filter...
creams and the judicious use of the anti-viral drug “acyclovir” which can be used prophylactically although more usually is applied when the first tingling symptoms or signs of rash appear. For treatment “acyclovir” can be applied to lesions or systemically in the more severe forms of infection.

Hepatitis B
This infection should not be confused with hepatitis A which is spread via the faecal-oral route. Hepatitis B is contracted almost exclusively through contact with infected blood or blood products, or sexually. The death rate from the acute infection is less than 1%, with most cases becoming “non-infections” in 3 to 6 months. In contrast those who become carriers initially usually have an asymptomatic illness, thus making it difficult to know which individuals should be taking special care to avoid transmitting infection.

The tendency in recent years has been to improve overall hygiene precautions rather than to concentrate upon those known to be carrying the virus. Any injuries, cuts or grazes that bleed are potentially a source of infection if the blood comes in contact with open wounds of another person. The virus can survive outside the body for some hours, depending upon environmental circumstances and this can lead to infection for example through the sharing of toothbrushes or razors.

Transmission through bleeding into communal bath water theoretically may occur. Both dilution and the presence of chlorine will reduce this risk although showering is undoubtedly safer, and more hygienic! The risk of spread through sporting activities themselves must be small although reports have been made of cases possibly due to blood transfer between the feet of barefoot cross-country runners or via contaminated thorn-pricks during orienteering events. When people are known to have been exposed or may knowingly become exposed, immunisation either passive or active or a combination of both is available. This may perhaps be considered for certain types of athletes competing in countries known to have a high carriage rate of virus among the local populations.

Acquired Immune Deficiency Syndrome (AIDS)
Routes of transmission of human immunodeficiency virus (HIV) are almost identical to those of the hepatitis B virus. Persons with HIV are probably most infectious shortly after the original exposure and again when the symptoms of AIDS are beginning to appear. It seems probable from the evidence available that the infection is more difficult to transmit by needlestick injuries than hepatitis B. Transfer of infection within a household, unless factors such as sexual exposure or drug abuse have been involved, are very rare (Friedland et al, 1986). Similarly the spread of infection through normal sporting activities must be unusual. As with hepatitis B, detecting the asymptomatic carrier can be virtually impossible and an awareness of the need for overall good hygiene especially when dealing with wounds and care to avoid sexual exposure, are essential.

Herpes, hepatitis and AIDS are all more or less avoidable, depending very largely on personal behaviour and practices. Herpes is the most highly infectious, but poses the least threat to life. In contrast AIDS would appear to be invariably fatal, but does not spread readily from person to person and scarcely at all amongst those leading a normal life-style (Sharp, 1987).

References


M.E. — POST VIRAL FATIGUE SYNDROME
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Myalgic encephalomyelitis (ME) is the latest name to encompass a syndrome complex that involves all or part of the muscles, the brain and its myelin covering. Post-viral fatigue syndrome suggests a possible viral cause.

As an illness ME occurs spectacularly more often in females in outbreaks, although it is otherwise evenly distributed between the sexes (Fegan et al, 1983). It has a predilection for age groups 25-35 and is more noticed in the social classes I and II. The symptoms presented can be enormously varied. Muscle symptoms can be pain on exertion, twitching or just simple rapid and excessive fatigue. The central nervous symptoms initially consist of vertigo with associated sleep disturbance, nightmares, lack of concentration, occasional dysphasia, memory loss, mood disturbance and severe anxiety states; other symptoms can vary with paraesthesia and hyperacusis. Other features appear to be a cyclical and prolonged convalescence with only 60% of affected patients being better after two years.

The primary contact in an illness is usually the general practitioner but equally may well be that of the coach, physiotherapist or team manager. From this unique position and knowledge of individuals they can spot change. The diagnosis however is an exclusion diagnosis often made retrospectively after at least a period of six months.

Positive findings include partial hypogammaglobulinemia, depressed IgG secretion by lymphocytes, reduced helper-suppressor cell ratio, altered immune suppression, occasional absolute and atypical lymphocytosis. Other findings are abnormal muscle metabolism with increased lactic acid, while 75% of biopsies show muscle fibre necrosis and neuro-physiological abnormalities (Arnold et al, 1984, Jamal and Hansen, 1985). Aetiology appears to be related to the immune response to infection with differing viruses producing different end-organ effects and the concept of a chronic viral illness must now be recognised (Yousef et al, 1988). Various viruses (Behan et al, 1985) have been incriminated including the Coxsackie group, Epstein-Barr, varicella, influenza and measles viruses. The Coxsackie virus in particular appears to be incriminated and attracts attention as it is known to be both myotropic and neurotropic and there are numerous anecdotal cases of sudden death in sportsmen during exercise (Roberts, 1986) with the Coxsackie virus being recovered post-mortem from myocardial tissue.

The illness seems to affect high achievers and the link between stress and susceptibility to infection has always been of interest.