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

Ludlam, H. and Cookson, B., 1986 "Scrum kidney: epidemic pyoderma caused by nephritogenic *Streptococcus pyogenes* in a rugby team". *Lancet* II: 331-333.

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, splinters, 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 sportsmen 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

- Collee, J. G., Macleod, D. A. D. and Little, K., 1987 "Wound Management and tetanus prophylaxis". *Communicable Diseases Scotland Weekly Report* 21 (50): 7-9.
- Smith, J. W. G., 1984 "Tetanus" in Topley and Wilson's *Principles of Bacteriology, Virology and Immunity*, edited by G. R. Smith, 7th Edition: 345-368, Edward Arnold, London.

HERPES SIMPLEX, HEPATITIS B AND THE ACQUIRED IMMUNE DEFICIENCY 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 ("scrumpox") or in wrestling ("herpes gladiatorum"), although "exposure" itself may be the precipitating factors when reactivation is involved.

Prevention depends upon hygiene, ultraviolet filter