AN ATLAS OF POTENTIALLY WATER-RELATED DISEASES IN SOUTH AFRICA

Volume 2

Bibliography

Report to the

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by

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BIBLIOGRAPHY

1. INTRODUCTION

As many professionals involved in the management of water resources do not have a medical or public health background, it was thought that an explanatory bibliography of the major water related diseases in South Africa would be of use.

Each section of the bibliography has (where appropriate) the following sections:

1. Aetiology
2. Transmission
3. Preventive and curative steps
4. South African data
5. References

The MEDLINE data base of the national Library of Medicine, Washington DC, and the WATERLIT data base of the CSIR were searched. These were supplemented by South African journals and reports not appearing in the MEDLINE data base.

2. AMOEBIASIS

2.1. Aetiology[2,7]

The protozoan parasite *Entamoeba histolytica* which causes amoebiasis may exist as a hardy infective cyst or a more fragile and potentially pathogenic trophozoite (the "amoebic" form). Amoebiasis most commonly affects the colon and rectum as primary sites of infection, with extraintestinal (most commonly liver abscesses), local or systemic, spread possible if not treated early. *E. histolytica* may cause a harmless luminal colonisation of the colon or rectum, or may invade the bowel wall causing amoebic dysentery (diarrhoea containing blood and mucous). Most infections are asymptomatic. The ability to bind and invade colonic epithelium is under investigation [1].

The virulence (ability to cause disease) of the organism tends to correlate with the isozyme phenotype. Both pathogenic and non-pathogenic zymodemes have been identified [3,6]. Cyst-passers that are asymptomatic generally carry non-pathogenic strains [4].

Diagnosis is made by microscopic examination of fresh stool specimens (or other specimens depending on extent of extra-intestinal spread) in order to demonstrate trophozoites or cysts. Cultures and serological tests are also available [5,8,9].

2.2. Transmission[2,7]

Amoebiasis is almost exclusively a human infection, and its presence in a community depends on the transfer of cysts between individuals. For this to take place food and water supplies need to be contaminated with faecies.

Outbreaks of disease generally result from the ingestion of faecally contaminated water containing amoebic cysts [11]. Only the cysts are infective, and dysenteric patients who pass the fragile trophozoites pose no hazard to others. Endemic transmission under poor hygienic conditions occurs via hand-to-mouth transfer of faecies, contaminated raw vegetables (common where gardens are fertilized with human faecies), the soiled hands of food-handlers, and occasionally by water.

Asymptomatic may pass cysts for a long time. This time period may be associated with the pathogenicity of the infecting organism.

2.3. Preventive and curative steps[11]

Specific measures related to the individual patient should include: sanitary disposal of faecies and hand washing after examining the patient and handling bed linen. Patients should be excluded from foodhandling activities. Close contacts should be given stool microscopic examinations. The specific treatment of cases involves taking metronidazole, and followed by iodoquinol in order to prevent cysts passing once the initial disease has been cured.

Adequate, dependable and safe water supplies for drinking, bathing and household uses forms the basis of the prevention of amoebiasis in the community. For safe water supplies to be used in preference to polluted open water sources, it should be available in the homes and workplaces of the population at risk. The use of human faecies for fertilization in vegetable gardening should not be allowed. The sanitary disposal of faecies and protection of public water supplies are also central to prevention.

Sanitary food preparation, which includes handwashing after defaecation, should receive attention.

2.4. South African Data

Amoebiasis is not a notifiable condition, and basic passive surveillance data is therefore not available for review of temporal and geographic trends.

A number of specific investigations have contributed to understanding the local epidemiology of the disease. A number of endemic "pockets" of disease, where sanitation and water supplies are inadequate, exist throughout the country. Endemic areas are known to exist in parts of KwaZulu/Natal and the Eastern Transvaal. During an 8 month period in 1980, 48 adult cases from Soweto were admitted to Baragwanath Hospital. 38% of these had extra-intestinal complications, and the mortality rate was 13%[10]. In the endemic Durban area 1% of asymptomatically infected individuals were infected with pathogenic zymodemes. 10% of these progressed to amoebic colitis in one year. Infection with pathogenic zymodemes occured in family units, highlighting the potential for transmission[4]. An oubreak of amoebiasis in a non-endemic farming comminity in Cape Town (Philippi) clearly illustrated the potential for transmission under poor hygienic conditions. Farm workers were living under harsh conditions, with the majority having no toilet facilities and using open

water sources for drinking purposes. The introduction of a pathogenic strain into this environment by a foreigner, resulted in a large scale outbreak [11].

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3. ARTHROPOD-BORNE VIRAL DISEASES

3.1. Aetiology

A large and ever increasing number of arthropod-borne virus diseases (arboviral diseases) are known to cause disease in man [7]. The most prevalent arboviral diseases where water is an important component in the transmission cycle are members of three families: *Togaviridae* (Chikungunya, Sindbis); *Flaviviridae* (Dengue, West Nile, Yellow Fever); and *Bunyaviridae* (Rift Valley Fever) [1,2,8,12].

3.2. Transmission

These viruses cause zoonoses (disease in amimals) in a variety of vertebrate hosts which serve as reservoir for infection. In the diseases listed above mosquitoes transmit the virus which is maintained in a continuous vertebrate-mosquito cycle. Infections in people are accidental, and man is an unimportant host in the cycle.

Transmission is by the bite of an infective mosquito, and species vary according to the specific virus: Chikungunya - *Aedes aegypti*; Sindbis - *Culex* spp.; Dengue - *Aedes aegypti*; West Nile - *Culex* spp.; Yellow Fever - *Aedes* spp.; Rift valley fever (affecting mainly livestock) - *Culex* and *Aedes* spp., some of which may be infected transovarially resulting in maintenance of the virus in enzootic foci [10]. Rift valley fever in humans may also (and quite frequently) result from handling of infective material from animal origen during necropsy or slaughtering [13].

All these mosquito vectors are dependant on the availability of open water sources for breeding. Rainfall and inadequate drainage (often related to infromal settlements) are central to the transmission of these arboviral diseases. Heavy rainfall resulting flooding of livestock drinking pans, hatching of vertically infected mosquitoes and leading to epizootic and epidemic infections is well documented in the case of Rift Valley fever [2].

3.3. Preventive and curative steps

Patients should be isolated in a screened room and given general supportive therapy. Blood and body fluid precautions should be instituted.

Preventive measures include: education of the public, elimination of breeding sites and improved drainage, destroying larvae, killing adult mosquitoes by resifual and space spraying, and all relevant barrier methods for preventing mosquito bites (see malaria), including day-biting mosquitoes. Breeding sites for *A. aegypti* (often containers near human habitations, such as old tyres) should be identified and eliminated. A highly effective vaccine is available for yellow fever [5].

3.4. South African data

Yellow fever does not occur south of the Zambezi valley, and only a few imported cases have been reported in South Africa. Since the 1926-1927 Dengue epidemic in South Africa, only a few imported cases have been noted [3,4]. The potential for Dengue transmission is however great due to the following factors: the susceptibility of the population, the presence of the main vector (mainly KwaZulu/Natal north coast), and the occurence of imported cases [3,11]. Rift Valley Fever is enzootic in the inland plateau west of the Drakensberg escarpment and the subtropical coastal forest of Natal, with most cases seen in January to May [6]. An epizootic of Rift Valley fever in South Africa in 1974/1975 resulted in a large number of human cases, both as a result of handling animal carcasses and via mosquito transmission [13]. Between December 1983 and April 1984 a large epidemic consisting of 28 confirmed cases of Sindbis virus infection and 5 confirmed cases of West Nile virus infection were reported in the Witwatersrand-Pretoria region [9].

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4. CHOLERA

4.1. Aetiology [1,13]

Cholera is an acute bacterial infection of the small intestine by *Vibrio cholerae* that characteristically results in watery diarrhoea. Asymptomatic infection (usually eltor biotype) is much more frequent than clinical disease, and mild cases with only diarrhoea are common among children. Severe disease manifests with profuse painless watery stools, some vomiting, rapid dehydration, acidosis and circulatory collapse.

Serogroup O1 *Vibrio cholerae* is the causative agent, and includes two biotypes - cholerae (or classical) and eltor. Eltor biotype is responsible for the current pandemic and results in a very high proportion of asymptomatic cases. Both biotypes produce a similar enterotoxin which causes the watery diarrhoea. Non-O1 serogroup *V. cholerae* are not associated with large outbreaks and epidemics and mostly follow the eating of inadequately cooked seafood.

4.2. Transmission [1,13]

Man is the only reservoir of disease. As the organism poorly tolerates drying and sunlight, transmission mostly takes place through ingestion of water contaminated with faeces of patients and carriers [7,14]. For this reason the occurence of this disease is highest during the summer rainfall season [12]. Unrefrigerated food contaminated by water, faeces, and soiled hands may play an important role in developing countries (Africa in particular) where personal hygiene is of low standard due to insufficient household water supply [11]. Storage of water (collected from open sources) in household containers may afford the vibrios adequate opportunity for multiplication and reaching infective concentrations [14].

4.3. Preventive and curative steps

Because a large proportion of cholera cases are mild or asymptomatic (and dfficult to distinguish from other causes of diarrhoea), isolation and quarantine have no role to play in cholera prevention and control [1]. Efforts should be directed at the rehydration of cases using oral glucose-electrolyte solution (and intravenous fluids in severe cases). In severe cases antibiotics (tetracyclines given orally) may reduce the volume and duration of diarrhoea, and the period of vibrio excretion [5,15].

At the community level activities must be directed at safe disposal of faeces, a safe water supply, and hygienic and safe food preparation. Health promotion efforts are central to achieving control, and people should be made aware that water can be made safe at home by boiling or adding household bleach. Chemoprophylaxis has limited application and should only be used in preventing disease in susceptible household contacts. Vaccination is not recommended and has been abolished from the International Health Regulations of the World Health Organisation (1973) [15,16].

4.4. South African data

Imported cholera was first detected at a gold mine in 1973/1974, and preventive measures were able to prevent subsequent transmission [10]. Cholera was made a notifiable disease in South Africa in 1965, and the first locally acquired case was reported in September 1980 [4]. The epidemic that ensued in South Africa between 1980 - 1987 (resulting in approximately 25 251 cases) is part of the extension of the seventh pandemic of cholera across Africa [8,11,15,17]. The communities most severely affected during this epidemic were blacks in rural, relatively high rainfall areas and living in conditions of poverty and poor sanitation : KwaZulu/Natal, KaNgwane and the Malelane area (eastern Transvaal) [2]. These areas are all in close proximity to neighboring Mozambique were cholera remains endemic up to the present moment [3]. Very few established urban communities were affected because of the availability of safe and ample water supplies. Cholera demonstrated a seasonal pattern, closely correlated to the ambient local patterns of rainfall and temperature [11]. No outbreaks occurred in areas of s+imilar socioeconomic status in dry arid regions of the country (with 99% of cases in areas with more than 600 mm annual rainfall) [11]. In Moletlane district the consumption of open river water was shown to be a risk factor for contracting the disease [14].

Seven cholera epidemic periods occurred in South Africa between October 1980 and July 1987. The case fatality ratio was 1.4 % for this period [11]. Age specific attack rates were higher in the older age groups, with no consistent difference between sexes. The overwhelming majority of patients were black [11].

The first case following the 1980 - 1987 epidemic was detected in a gold miner in 1991. Subsequently two more cases were detected at Shongwe hospital in KaNgwane in 1992. During 1992 a total of 13 cases were notified, and in 1993 a further 74 [6]. Although a major epidemic has not yet occurred, the presence of

endemic cholera in neighboring Mozambique serves to underscore the importance of primary prevention and continued surveillance of this condition [9]

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5. DIARRHOEAL DISEASE IN CHILDHOOD

5.1. Aetiology[1,5]

Diarrhoea is a clinical syndrome characterised by acute onset of more than three loose or watery stools per day and is often accompanied by vomiting and fever. It is therefore a symptom complex of infection by any one (or combination) of a variety of enteric pathogens[4] In the majority of children with acute diarrhoea is no pathogen can be isoloted from stool with routine laboratory tests and the aetiology is presumed to be viral (rotavirus and Norwalk agent are common). In less developed regions bacterial and parasitic agents are responsible for a significant proportion of infections[2,7] In South Africa the following agents are important: shigellosis[3], salmonellosis, yersiniosis, giardiasis, campylobacteriosis, cryptosporidiosis, enteropathogenic- and enterotoxigenic strains of *Escherichia coli*. Rotavirus is the only undisputed viral agent and enteroviruses and adenoviruses are rarely isolated[4,6].

5.2. Transmission[1,5,7]

Diarrhoea transmission is by ingestion of feacal organisms and is encouraged by conditions where there is feacal contact and spread. The epidemiology and transmission varies significantly according to the aetiologic agent. Certain organisms such as *Shigella* are host specific and require a small inoculum size to cause infection. In such instances person-to-person contact transmission is important and can be controlled by improved personal hygiene and hand washing in particular. In infections such as *Salmonella, E. coli* and *Campylobacter,* with a wide range of reservoirs (including animals) and where higher inocula are required, food and water contamination are of greater importance. In all instances peak transmission occurs in the summer months (November to February)[4].

5.3. Preventive and curative steps.

Primary prevention is based on interruption of the feacal-oral transmission of causative agents and requires behavioural and environmental interventions such as sanitary waste disposal, adequate clean domestic water supplies, refuse removal and improved personal (including hand washing) and food hygiene[6,7]. Local studies have indicated that the absence of an inside toilet and refuse

receptacle, overcrowding, the use of a feeding bottle during weaning, and the educational status of mothers to be important risk indicators [8,12].

The promotion of breast feeding is by far the most important and cost-effective intervention. Not only do specific immune factors in breast milk protect the child, but the child is also not exposed to the likely contamination of the feeding bottle [7].

Oral rehydration therapy (ORT) (consisting of a sugar-salt solution) given as soon as possible after onset of disease, is the intervention of choice to offset the effects of acute diarrhoea: loss of fluid and electrolytes and reduced nutrient intake. ORT has resulted in dramatic reductions in morbidity and mortality due to diarrhoea. In most instances of undifferentiated diarrhoea antibiotic therapy has no value and can even hinder recovery [7,10].

5.4. South African data.

Diarrhoeal disease remains a major cause of morbidity (an estimated 1.5 million cases in 1984) and mortality in South Africa. More than a quarter of all registered deaths in children under 5 years of age are due to diarrhoea (8984 deaths in 1984). The highest risk group are black and coloured children under the age of 1 year. A marked seasonal mortality pattern is evident with peaks between December and March [9,11]. Over the period 1968 - 1985 there has been a steady decline in diarrhoeal disease mortality rates for white, coloured and Indian population groups. No trend is discernable in blacks due to incomplete mortality data [11].

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6. VIRAL HEPATITIS A AND E

6.1. Aetiology

The hepatitis A virus (classified as *Enterovirus* type 72) infection results in acute onset of fever and malaise, followed by jaundice. Many infections, particularly in children, are mild or even asymptomatic [6]. There is no evidence of a chronic form of infection.

Hepatitis E causes a disease similar to that resulting from hepatitis A infection. There is also no chronic form of infection. It is a non-enveloped RNA virus. Although sofisticated diagnostic tests are available, the diagnosis is basically by exclusion of hepatitis A and other forms of hepatitis (using serological tests) [8].

6.2. Transmission

Man is the only reservoir of importance and the mode of transmission is person-to-person via the faecal-oral route. The virus is excreted in the faecies of infected individuals. The contamination of water sources and food then provides the transmission link to uninfected persons. Contaminated water sources and food infected by food handlers have often been reported [1]. Direct transmission amongst male homosexuals has been described.

The reservoir and transmission of hepatitis E is as for hepatitis A.

6.3. Preventive and curative steps

The provision of sufficient safe potable water supplies (for personal use, cooking and handwashing) and the sanitary disposal of faeces are the most basic and important primary preventive measures to be taken. Only once the above is in place, public motivation to encourage good personal hygiene (handwashing) and safe disposal of faecies has an important role to play. Handwashing in pre-school and day-care centeres (particularly if some children are in nappies) should receive attention. The contamination of shellfish (particularly oysters and clams) is common in certain areas and these foods should be thoroughly cooked before eating. Vaccination is available against Hepatitis A, and is particularly indicated during an outbreak situation, or for travellers to endemic areas [5].

The preventive and curative steps for hepatitis E are similar to that of hepatitis A. No vaccine is available for hepatitis E.

6.4. South African data

Hepatitis A is a notifiable condition in South Africa, and has been reported from most regions of the country. A number of hospital based studies have reported on prevalences of disease, but population based data is lacking [3,4,5,6]. In 1990 Natal had the highest rate (11 cases per 100 000), followed by the Western Cape (9 per 100 000). The Eastern Cape, Northern Cape, Southern Transvaal, and Northern Transvaal reported similar rates (4 per 100 000) [2]. On a national level the incidence rate of hepatitis A has increased from 4 per 100 000 in 1986 to 5 per 100 000 in 1990. The reason for the increase in incidence is not clear, and may be a funcion of improved reporting. Age specific data show that the younger age groups (5-9 years) have the highest incidence rates (8 per 100 000) compared to young adults (4 per 100 000) and persons over the age of 40 years (1 per 100 000). Notification rates are highest in whites, followed by asians, coloureds and blacks. These differences are again most likely a reflection of reporting rather than actual disease frequency.

It is expected that more Hepatitis E cases will be diagnosed as diagnostic testing comes into greater use throughout the country [7]. At present hepatitis E is not a notifiable condition and the distribution and prevalence of disease is not known [8]

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7. LEPTOSPIROSIS

7.1. Aetiology

Leptospirosis is a bacterial disease characterised by fever, headache, severe myalgia, conjunctival suffusion, and in some cases jaundice or haemorrhagic rash. Cases are often misdiagnosed as meningitis or encephalitis. Pathogenic leptospires belong to the species *Leptospira interrogans*, and constitute a large group of serovars. Common serovars include *icterohaemorrhagiae, pamona* and *canicola* [1,3].

7.2. Transmission

Leptospirosis is a zoonotic disease with wild (rats) and domestic (swine, cattle, dogs) animals acting as reservoirs for infection. Transmission is via skin contact with water, moist soil, or vegetation contaminated with urine of infected animals. Swimming, accidental, and occupational contact with contaminated water are common modes of transmission [1,2,3].

7.3. Preventive and curative steps

The following are important aspects to consider: 1) preventing public access to potentially contaminated water for recreational and domestic purposes by providing safe alternatives; 2) providing protective clothing to workers in hazardous occupations; 3) control of rodent populations in human settlements (including refuse removal); 4) in problem settings immunisation of domestic animals and humans against specific serovars has been used with some success [1,2].

7.4. South African data

The occurrence of leptospirosis is poorly documented in South Africa, and limited published data is available [2,3].

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8. PEDICULOSIS

8.1. Aetiology

Pediculus capitis (head louse), *P. humanus* (body louse), and *P. pubis* (crab louse) infest the head and hairy parts of the body with resultant itching and excoriation. The body louse has greater public health significance as it can act as the vector of epidemic typhus [1].

8.2. Transmission

Direct contact with an infected person, or indirect contact via the sharing of clothes, headgear, and bedclothes. Crowded living conditions with insufficient household water supply to bathe and wash clothes/linen are central determinants of this infestation. The crab louse is most frequently transmitted by sexual contact [1].

8.3. Preventive and curative steps

Topical treatment with benzyl benzoate (a number of other agents available), removal of all nits from hair, and hot water washing of all clothing and bedding are essential to prevent further transmission. In terms of primary prevention, adequate living conditions (less crowding) and regular washing of clothing and linen will prevent transmission in households [1].

8.4. South African data

Pediculosis is endemic in all regions in South Africa where people have low incomes, live under crowded conditions, and have insufficient water supplies for the maintenance of household hygiene.

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9. MALARIA

9.1. Aetiology

Malaria in man is caused by four different species of the parasite (*Plasmodium falciparum, P. malariae, P. vivax, P. ovale*), each with its own pathology and epidemiological features. In Southern Africa 95% or more of the infections are due to *P. falciparum*, which causes the most severe form of malaria. Most of the remaining infections are due to *P. ovale*, but *P. vivax* and *P. malariae* occasionally occur. *P. vivax* and *P. ovale* are the relapsing forms of malaria but are rarely fatal [9,25].

9.2. Transmission

Man is the only important reservoir of human malaria. Nonhuman primates are naturally infected by a variety of malarial species (other than the four mentioned above) which can infect man, but natural transmission is extremely rare.

Person-to-person transmission is by means of the bite of an infective female anopheline mosquito and is therefore a blood-borne infection at this level. Untreated acute and chronic cases of malaria act as the reservoir for disease. In this regard semi-immune migrants from Mozambique have provided a constant pool of parasites. In rare instances transmission may occur by means of needles contaminated with infected blood and blood transfusions. Congenital transmission occurs rarely.

Currently the main vector in South Africa is *Anopheles arabiensis*, which is an endophilic as well as exophilic feeder [11]. The behaviour and biology of the mosquito vector are directly linked to climatic factors (and rainfall patterns in particular) which are therefore of central importance in deciding disease transmission [32]. Moderate rainfall encourages Anophele breeding, but if excessive may flush out mosquito larvae [27]. In endemic regions falling water levels leave pools which are ideal for *An. arabiensis* breeding. Normal or higher rainfall following prolonged droughts is associated with an increased prevalence of the principal vector with resultant upsurge in disease incidence [17,35].

9.3. Preventive and curative steps[14,16,21]

Malaria prevention and control comprises the following strategies: prompt treatment of acute and chronic cases so as to reduce the parasite reservoir[7,12,15,20,26,31,33,36,37]; taking of prophylactic drugs[8,2]; vector control and avoidence of mosquito bites (personal protection). The latter consists of wearing long sleeves and long trousers, applying repellents, screening windows, sleeping under bednets (preferably impregnated with pyrethroid), and using insecticide aerosols and burning coils. From a public health perspective the basis of malaria control operations consists of intra-adomiciliary residual spraying with DDT and other insecticides[2,3,4]. This method does however have limitations as *An. arabiensis* also feeds outdoors, and contact with the insecticide is reduced[5]. Environmental measures such as ensuring adequate surface water drainage (particularly peridomestic collections of water in rapidly expanding informal settlements) and the provision of protective housing (removed from potential breeding sites) is central to reducing the vector population and man-vector contact[13].

Drug prophylaxis provides additional, but not absolute protection against malaria. Because of the existence of chloroquine resistant malaria in Southern Africa particular care must be taken in the selection of appropriate drugs for individual protection. Recommendations for malaria chemoprophylaxis in Southern Africa have been published[1].

9.4. South African data

Malaria is seasonally endemic (peak transmission during the rainy season - March, April and May) in the less developed regions of the lowveld of the eastern and north-eastern Transvaal and northern KwaZulu/Natal[27,29,34]. The malaria case load for the country during the period 1976 -1992 rarely exceeds 10,000 per annum[6,24]. The major increase in cases over the period 1985 - 1988 corresponds with the detection of chloroquine resistance in South Africa and is partly due to increased agricultural development in malarious areas[10,18,19,23,30]. During the period 1987 - 1992 incidence rates have been highest in Transvaal, Gazankulu, Venda, and KwaZulu. The total incidence rate for the endemic regions was 174 per 100 000 in 1988 (and 119 per 100 000 in 1986)[6].

Approximately 50% of cases occur in the under 20 year age group with peaks in the 5-9 and 15-19 year olds. The 15-19 year age group shows the highest incidence rate (34 per 100 000) while children below 4 years of age are at

minimal risk. After the 15 -19 year peak, the risk of malaria decreases almost linearly with age. Case fatality ratios are under 0.4 in the age groups below 50 years, and well over 1.3 in older age groups [6]. A male/female ratio of 1.2 was experienced over the period 1980-1989 [6].

Approximately 30% of all cases of malaria reported in the last six years are classified as imported into the country. The majority have originated from Mozambique.

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10. POLIOMYELITIS

10.1. Aetiology[1,8]

Poliomyelitis is an acute viral infection with varying severity. The spectrum of disease ranges from inapparant infection to nonspecific febrile illness, aseptic meningitis, paralytic disease and death. Three closely related but antigenically distinct virus strains (types 1, 2, and 3) are the aetiological agents. All three types of virus can cause paralysis, and are classified as picornaviruses belonging to the enterovirus group. Type 1 is isolated from paralytic cases most often, type 3 less so, and type 2 least commonly. Type 1 most frequently causes epidemics. Most vaccine associated cases are due to types 3 or 2.

10.2. Transmission[1,8]

Man is the only host for this infection. Persons with inapparent infections, especially children, serve as a disease reservoir and harbor the virus in pharyngeal secretions and faeces. Direct person-to-person contact by oral-faecal or oral-oral routes is responsible for most transmission of disease. In a minority of instances faecally contaminated foodstuffs and water have been involved as vehicles. There is no reliable evidence to link flies as being important in transmission.

Where sanitation and personal hygiene are poor, transmission occurs more frequently via the faecal-oral route, whereas in settings in which sanitation are better, person-to-person transmission may result more from oropharyngeal spread.

10.3. Preventive and curative steps[1,8]

Improvements in the standard of sanitation and personal hygiene in populations will result in a decrease in faecal-oral transmission of the virus which is so typical of settings with endemic disease. With increased standards of living fewer infants are exposed to the virus early in life when most infections are asymptomatic. The result is that larger numbers of susceptible older children and young adults accumulate with resultant unstable epidemic (no longer endemic) disease and oropharyngeal spread.

Primary prevention by means of vaccination is the only effective means of preventing disease. Two vaccines are available: inactivated poliovirus vaccine

(IPV) and attenuated oral polio vaccine (OPV). OPV is preferred in developing countries (including South Africa) because it simulates natural infection and induces both circulating antibody and intestinal resistance, and protects susceptible contacts by secondary spread [12]. Three doses of trivalent OPV spread over the first six months of life is the recommended protection for infants in developing countries. In high-risk areas an additional dose of monovalent OPV is given at birth. IPV blocks paryngeal excretion but does not prevent intestinal infection.

Isolation of poliomyelitis cases in hospitals is no longer recommended, and quarantine is of no value as most infections are inapparant. The closing of schools and other places of public congregation has no proven value. The most effective method of epidemic control remains the immediate administration of trivalent OPV to those over 6 weeks of age who have not been adequately immunized.

10.4. South African data.

Despite the introduction of vaccines in the late 1950s, outbreaks of increasing magnitude continued to occur in South Africa at regular intervals [7]. Between 1960 and 1975 a 3-yearly cyclical pattern in polio epidemics was clearly distinguishable on a national level. After 1975 a gradual decline in the annual number of reported cases has been observed, with epidemic peaks widening to every fouth year [5]. In 1990 only 4 cases were reported, followed by 2 cases in 1991 and zero cases in 1992 [3,7]. The WHO goal of total eradication of poliomyelitis by the year 2000 therefore seems to be within reach [2,10].

Since 1980 only three major epidemics were reported. These may have resulted due to decreased herd immunity in rural areas [11], and loss in vaccine potency due to a break in the vaccine cold-chain [4]: KwaZulu in 1981 [12]; Gazankulu and Lebowa in 1982 [6,9,13]; Natal/KwaZulu in 1988 [14,15]. It was suggested that massive floods (resulting in disruption of sanitation and water supplies) in the preceading two months may have triggered the 1988 outbreak [15].

Most cases of acute paralytic poliomyelitis occur in children under 5 years of age. The highest age-specific incidence rates during the period 1980 - 1990 occured in the 1-4 year age group (1 to 3 per 100 000). During this period the vast majority of cases have occured in the black population group and polio type 1 has been the predominant virus type isolated [3].

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11. SCABIES

11.1. Aetiology

A disease of the skin caused by the mite *Sarcoptes scabiei*. The mite penetrates the skin resulting in papules, vesicles, and linear burrows containing mites and their eggs. This infestation leads to intense itching and secondary infection common due to scratching. Compromised patients (immunosuppressed or senile) often develop "Norwegian scabies" - a more generalised dermatitis with extensive scaling and crusting [1,4].

11.2. Transmission

Man is the reservoir for this disease, and transmission is by direct skin-to-skin contact (including sexual contact). Transfer via clothes and bedclothes to family members is common in households with insufficient access to water for maintenance of basic standards of hygiene [1].

11.3. Preventive and curative steps

Specific curative treatment consists of the application of an emulsion of benzyl benzoate to the whole body (except head and neck) and washing of clothes and bedclothes. The latter may be particularly difficult where access to sufficient volumes of potable water is lacking.

Prevention is based on public awareness, early diagnosis and treatment of patients and contacts. Infested patients should be excluded from work or school until the day after treatment, and should be subject to contact isolation for 24 hours after treatment has commenced. The laundering of clothes, underwear, and bed sheets (using hot water) is essential to break the cycle of infection in households. The most important primary preventive measure is having access within (the household) to sufficient quantities of potable water, so as to allow family members to bath regularly and wash their clothes and linen.

11.4. South African data

Scabies is endemic in most regions of South Africa, particularly affecting low-income groups [2,4]. The secondary infection associated with epidemic scabies has been implicated as a major determinant of acute glomerulonephritis [3].

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12. SCHISTOSOMIASIS

12.1. Aetiology

Schistosomiasis (or Bilharzia) is a blood fluke (trematode) infection of humans and animals. The schistosome worms live and lay eggs in the veins of various organs (mainly gut and urinary bladder) for many years. Eggs result in the formation of granulomata where they are deposited by the schistosomes. The number and location of eggs in the human host results in symptoms and signs, with variation according to schistosome species. Three forms of schistosomiasis occur in South Africa:

1) *Schistosoma haematobium* affecting the urinary tract with resultant symptoms of blood in the urine. (*Bulinus sp.* provide the snail intermediate host.)

2) *Schistosoma mansoni* affecting the intestinal tract with resultant diarrhoea containing blood and mucus. (*Biomphalaria sp.* provide the intermediate snail host.)

3) *Schistosoma mattheei* - intestinal bilharzia of animals which occasionally affects man. (*Bulinus sp.* provide the snail intermediate host[6,15,18].

12.2. Transmission

Direct contact of exposed skin or mucous membranes with water containing cercariae (larval forms) results in infection. Eggs are passed out in the excreta of infected humans (and animals), hatch in water extruding miracidia which infect the relevant fresh water snail intermediate hosts (see above). After a few weeks free-swimming larval stages (cercariae) are released which in turn infect man by penetrating human skin during contact with water. Cercaria penetrate the bloodstream, develop and mature into adult worms which migrate to the veins of the abdominal cavity. Eggs that are deposited by adult worms in venules, migrate into the lumen of the gut or urinary bladder, or lodge in other organs, including the liver and lungs.

12.3. Preventive and curative steps

Specific treatment with praziquantel is effective in the early stages of infection. Disease resulting from chronic and repeated infection includes liver fibrosis, portal hypertension, obstructive uropathy and possibly bladder cancer and would require supportive medical and surgical interventions.

The prevention and control of this water-based disease requires a multisectoral approach. During the planning of irrigation schemes, public health experts have a key role to play in preventing the expansion of disease transmission. The provision of safe water supplies and adequate sanitation is pivotal to the success of any intervention [10]. Other aspects that are of importance include: prevention of exposure to contaminated water; control of host snails by biological means [8] or selective molluscicides [17]; improve irrigation and agricultural practices; treatment (including mass treatment) of patients and pre-symptomatic cases in endemic areas; and relevant health promotion [11].

12.4. South African data

The southern part of the former Transvaal, the Orange Free State, Lesotho, the North West Province, and the Western Cape Province are the only areas in South Africa where bilharzia infection is not found [7,12]. Therefore, the main areas in which the disease is found are: Northern Transvaal, Eastern Transvaal, KwaZulu/Natal, the Cape Eastern seaboard from the KwaZulu/Natal border as far south as Humansdorp, and foci along the lower Orange river. Urinary disease is far more widespread than intestinal disease which is found in the Lowveld, Swaziland and Northern KwaZulu/Natal. *S. mattheei* is found over the whole area. Well in excess of three million South Africans are estimated to be infected [5]. Infection rates in school children of over 80% for *S. haematobium* and *S. mansoni*, and over 40% for *S. mattheei* have been reported from most rural areas. Although all ages are affected, the peak prevalence of infection is in children seven to 15 years of age [14,19]. Reductions in infection prevalence in school children due to improvements in water and sanitation services in some rural areas have recently been reported [9]. Apart from blood in the urine, the majority of children infected with *S. haematobium* generally appear healthy. However, about 40% may show urinary abnormalities (calcified bladder, polypoid lesions, obstructive uropathy, renal failure), with bladder cancer being the leading cause of mortality in adulthood [3]. *S. mansoni* results in minimal morbidity in South Africa [1,2,4,13,16].

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13. TRACHOMA

13.1. Aetiology [5]

Trachoma is a chlamydial conjuctivitis of insidious or abrupt onset and the infectious agent is *Chlamydia trachomatis* (serovars A, B, Ba, C) [3]. In hyperendemic regions frequent reinfection takes place resulting in diffuse conjunctival inflammation (particularly on the upper eyelid). Vascularization of the cornea (pannus) and scarring or the conjunctiva ensues. In-turned eye lashes and lid deformaties result, causing chronic abrasion of the cornea and visual impairment with blindness later in life. Associated bacterial infections abound and enhance transmission and severity of disease. Microbiological laboratory diagnosis is possible using conjunctival scrapings [1].

13.2. Transmission [5]

The reservoir of disease is man. Transmission takes place by contact (direct or via fingers and soiled clothes) with ocular and nasopharyngeal discharges. Flies may be of importance in certain instances. As long as active lesions are present (this may be for a few years) the disease is communicable. The severity of disease is related to poor hygienic living conditions and possibly exposure to dust.

Transmission takes place primarily within the household, with the main source of infection appearing to be pre-school children [4].

13.3. Preventive and curative steps

The improvement of basic sanitation, with community access to water in homes is essential. Once this has been achieved, educational programmes highlighting the importance of personal hygiene are important. Transmission is reduced if persons have access to soap and water and avoid common-use towels and toilet articles. Specific treatment involves the use of topical tetracycline and erythromycin ointments. Mass treatment (focussed on children) has been advocated in certain endemic regions. However, sustainable primary health care interventions at household level using community health workers to provide treatment and education, have been shown to provide a more effective alternative [9,10].

13.4. South African data

Trachoma in South Africa is mainly distributed within the Northern Transvaal and KwaZulu/Natal. Between the years 1971 and 1980, 3248 and 392 cases were notified from Northern Transvaal and KwaZulu/Natal respectively [7,8]. The disease has a patchy distribution in the Northern Transvaal [2,4,6]. Blinding hyperendemic trachoma has been shown to exist in the Lebowa and Gazankulu areas. Pre-school children are most affected, with reinfection of adults from this group resulting in blindness presenting mainly in older adults [4]. Females have a higher prevalence of disease (female to male ratio 2:1) [2,4,6].

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14. TYPHOID FEVER

14.1. Aetiology.[3,11]

Typhoid fever is a systemic bacterial disease caused by *Salmonella typhi*. This is a flagellated gram-negative bacillus which is nonlactose-fermenting. Identification is by means of its biochemical properties and the presence of somatic (O) and flagellar (H) antigens. Freshly isolated strains have a capsular (Vi) antigen. More than 106 types are distinguished by phage typing, and is of importance in epidemiological investigations.

14.2. Transmission[2,11]

S. typhi is a natural pathogen only of humans. Following acute illness or mild and even subclinical infections, the affected person may develop an asymptomatic carrier state which may last a few weeks or become chronic, lasting many months and even years. Carriers excrete *S. typhi* in their stools or urine and serve as the reservoir for future infection. A chronic urinary carrier state is common in those with *Schistosoma haematobium* infection.

Transmission is by means of food and water that has been contaminated by the faecies and urine of patients and carriers. Transmission is facilitated by the ability of *S. typhi* to survive in water and sewage for weeks and months.

14.3. Preventive and curative steps[2,11]

Antimicrobial therapy is effective in terminating most cases of clinical illness. Despite some reports of emerging antibiotic resistance the majority of cases of typhoid in SA remain susceptible to ampicillin/amoxycillin and chloramphenicol[3,5,13]. As recent as 1992 cases of typhoid fever resistant to ampicillin, chloramphenicol and trimethoprim-sulphamethoxazole were reported during an outbreak in Northern Natal. In cases suspected of drug resistance it is recommended that third generation cephalosporins or quinolones are used as empiric therapy for typhoid fever[3]. Oral therapy with ciprofloxacin has been reported to be effective in the treatment of multiply antibiotic resistant strains of *Salmonella typhi*[4].

A chronic carrier state may persist after treatment and regular stool and urine cultures should be taken to diagnose this condition and instigate early preventive measures. Additional clinical information related to diagnosis and management is available from recent publications [1,8,12,14,16].

Preventive and control measures include the safe disposal of sewage; adequate supplies of safe water for maintenance of personal hygiene (including handwashing) and drinking; high standards in food handling and preparation; pasteurisation of milk and dairy products and identification and surveillance of carriers. Routine use of typhoid vaccine is not recommended as a control procedure.

14.4. South African data.

The number of cases reported annually ranges from 8276 in 1934 to 2000 in 1961. The peak incidence rate of typhoid fever in South Africa during the eighties was in 1984 (19 per 100 000). Since then the rate has declined to 7 per 100 000 in 1989 and less than a 1000 cases in 1992. [6,7,9,10]. Case fatality ratios have also declined dramatically from 16% in 1945 to less than 1% at present. The areas most affected by typhoid fever are the north-eastern Transvaal, the eastern coastline from Ubombo in the north, along Natal, to Transkei in the south and the districts on the south-western border of Lesotho [6,10]. Typhoid fever is hyperendemic in the old homelands of Venda, Lebowa and Gazankulu. During the period 1976 - 1979 the annual notification rate in these three regions varied between 16 and 110 per 100 000 [15].

The vast majority of cases occur in the Black population group and 60% of cases are in the 5-19 year age group. There is an equal distribution of cases between the sexes. Typhoid fever shows a distinct seasonal pattern with distinct peaks in the rainy months, January through to March [6,10].

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15. INTESTINAL HELMINTHIASIS

15.1. Aetiology

Gastrointestinal helminthiases (parasitic worms) are among the most prevalent and widespread of chronic human infections. More than a quarter of mankind is currently infected with one or more species of gut parasitic worm [2]. A minority of these infections result in serious disease. In general, only individuals acquiring high intensity infections resulting in large worm burdens, develop serious disease [2]. A wide variety of worms can result in infestation. However, the most prevalent genera (of direct importance to the water-and-disease theme of this text) are: *Ascaris lumbricoides, Trichuris trichiura,* and *Hymenolepis nana.*

15.2. Transmission

Humanity is the chief source of infection in all three the worms listed above. Transmission is direct, and only a single host (man) is involved. These helminth infections are most frequent in the 5- to 9-year-old group. Preschool and young school children are more affected because of their more frequent exposure to contaminated soil during play and hand-to-mouth actions. Low income urban and rural populations are most afflicted because of poor hygienic conditions (inadequate access to sanitation and clean water domestic supplies). Infection takes place in the household with the family unit being the unit of dissemination. Small children with worm infestation who defecate in the open (on floors and in playing areas) provide the chief source of eggs. Eggs remain viable in the soil for long periods. Infective eggs are transmitted hand-to-mouth by children who have come into contact with contaminated soil through playthings or dirt eating.

Where night soil is used for the fertilisation of market gardens human infestation (of all ages) results from eating raw vegetables. Even though direct contamination of drinking water is rarely a source of infection, water (or rather the lack of it) is imprortant in transmission. Without access to sufficient quantities of clean household water, high levels of family hygiene can not maintained.

15.3. Preventive and curative steps

Mebendazole is the drug of choice for the treatment of *Ascaris lumbricoides*, *Trichuris trichiura* and mixed helminth infections, and piperazine citrate is effetive in the treatment of *Hymenolepis nana* infestation.

In endemic areas worm infestation has been shown to affect the cognitive function and school performance of children, and undernutrition with growth stunting and iron deficiency anaemia in severe cases [5,8,13]. For this reason the mass treatment of school children with single dose mebendazole at regular intervals has been advocated. Such mass treatment has been shown to be effective in reducing the intensity of infestation, infection prevalence, and incidence of surgical complications due to *A. lumbricoides* [4,10,16].

Additional aspects of importance to the prevention of infection in highly endemic areas are:
1) the sanitary disposal of human faeces;
2) improving family hygiene by making adequate safe water available for hand washing (particularly before meals and after defaecation); and
3) washing and scalding of uncooked vegetables where night soil is used for fertilizer.

15.4. South African data

A number of studies have illustrated the endemicity of helmith infestation in a variety of localities in South Africa. Prevalences for *A. lumbricoides* and *T. trichiura* vary from more than 70% for the South Western Cape Province [3,17] to 50% and more in KwaZulu/Natal [1,15] and between 40% and 55% in the Eastern Cape Province [7,8]. In hot and dry climates, such as the Richtersveld (northern Cape), prevalences of under 6% have been reported [14]. An additional dimension to the public health problem posed by ascariasis is the high prevalence of complications: intestinal obstruction, biliary obstruction, allergic asthma and pneumonitis, and dissemination of larvae [6,9,11,12].

Much lower prevalences have been reported for *H. nana* infestation, varying from 1% to 15% [7,14,15,17].

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