Wolfe Medical Atlases – 17

A colour atlas of Tropical Medicine and Parasitology

Second Edition

W. Peters & H. M. Gilles

Filariasis of the male genitals – from a woodcut by Katsushika Hokusai (Hokusai Mangwa, v. 12, 1834). (By courtesy of the Wellcome Trustees.)

A colour atlas of Tropical Medicine and Parasitology

Second Edition

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Contents

Preface 9
Introduction 11
Part I: Arthropod-borne Infections 14
Arboviruses 16
Rickettsioses 25
Bacterial Infections 30
Protozoal Infections – Malaria and Babesiasis 36
Trypanosomiasis 63
Leishmaniasis 85
Nematodes – the Filariases 108
Part II: Soil-mediated Helminthiases 142
The Hookworm Infections 143
Infection with *Ascaris* and Related Nematodes 155
Trichuriasis 160
Part III: Snail-mediated Helminthiases 162
Schistosomiasis 163
The Intestinal Flukes 189
Liver Fluke Infections 194
Paragonimiasis 203
Angiostrongyliasis 208
Part IV: Infections Acquired through the Gastro-intestinal Tract 210
Viral Infections 211
Bacterial Infections 214
Protozoal Infections 225
Helminthiases 244
Pentastomiasis 274
Part V: Infections Acquired through the Skin and Mucus Membranes 277
Viral Infections 278
Bacterial Infections 284
Protozoal Infections 298
The Superficial Mycoses 301
The Systemic Mycoses 302
Ectoparasitic Arthropods 310

Part VI: Airborne Infections 315
Measles 316
Meningococcal Meningitis 318
Tuberculosis 321
Whooping Cough 321
Part VII: Nutritional Disorders 322
Kwashiorkor and Marasmus 323
Nutritional Marasmus 325
Avitaminoses 326
Part VIII: Miscellaneous Disorders 336
Zoonotic Viral Infections 336
Iatrogenic Parasitoses 339
Neoplastic Conditions 341
Genetic Blood Dyscrasias 342
Venomous Bites and Stings 345
Diseases of Uncertain Aetiology or Epidemiology 349
Life Cycles 356
List of Tables 368
Bibliography 385
Index 386

Preface

The striking contrasts in factors governing morbidity and mortality in the Third World as compared with the developed nations call for a few comments. The two essential features are the increased lethal effects of the commoner bacterial and viral diseases, and the enormous degree of morbidity occasioned by those due to parasites. Simple infections such as nonspecific gastroenteritis and associated disorders acquired through the gastrointestinal tract, account for a high proportion of the world's mortality, probably some 6% of deaths from all causes as compared with the 10% believed to be due to pneumonia. However, it was recently estimated that the disease-specific mortality rates are higher in the poorer countries than in the richer by the following factors: influenza, pneumonia, bronchitis 2.3; respiratory tuberculosis 5; dysentery (all forms) 7.5; typhoid 160; diphtheria 100; whooping cough 300; measles 55. Including the parasitic infections, communicable diseases account for about one third of all recorded deaths, but the degree of mortality caused directly by parasites is difficult to estimate except in the case of malaria where a case mortality rate of up to 1% seems a probable figure.

Parasitic infestation is usually multiple. According to recent estimates intestinal helminths probably affect at least 1,000 million, about 300 million are exposed to schistosomiasis, and 250 million to filariasis, not counting another 50 million at risk of acquiring onchocerciasis and river blindness. Of these helminthiases it is suggested that about 13% are transmitted by arthropods, 46% are soil-borne and seven per cent are transmitted through snails. Malaria, according to a recent WHO estimate, is still endemic in areas occupied by 800 million people, ie 21% of the world's 3.8 billion population. In Africa 50 million inhabitants face the threat of trypanosomiasis, and a similar number are exposed to Chagas' disease in the New World, while at least seven million in various continents live in areas where leishmaniasis is present.

Between 10 and 15% of the world's population is estimated to be undernourished and this is manifested by a spectrum of sequelae ranging from a diminished resistance to infection, to death from acute starvation in times of famine such as we have recently experienced on the African continent and the Indian peninsula. Some estimates put the total figure for undernutrition or frank malnutrition as high as 50%.

Part 1
Arthropod-borne Infections

Numerically speaking mosquitoes are probably responsible for more disease than any other group of arthropods but other insects too are of great importance. While *Anopheles* mosquitoes carry malaria, various viral infections and some types of filariasis, other viruses and filarias are transmitted by culicines. Other biting flies transmit African trypanosomiasis, leishmaniasis, bartonellosis and several other kinds of filariasis. Fleas carry a species of typhus and plague, lice carry epidemic typhus, and mites and ticks other varieties of typhus. Ticks are also responsible for transmission of the haemorrhagic and the relapsing fevers. The arthropod vectors of disease are classified in Table I.

The important arthropod-borne (arbo) viruses considered here are yellow fever, Southeast Asian haemorrhagic fever and Japanese B encephalitis. These are representative of the arbovirus diseases which include many other infections of man in the tropics (*see* **Table II**). Of the Rickettsioses, louse-borne typhus due to *R. prowazeki* is potentially the most important, but tick typhus is fairly common in some areas (eg East Africa) and mite-borne scrub typhus (tsutsu-gamushi disease) in Southeast Asia and the Southwest Pacific.

Plague is still endemic in certain tropical and subtropical areas, and localised outbreaks are not uncommon especially in war situations, eg Vietnam, where there has been an increase in recent years. Relapsing fever in Africa and Bartonellosis (Carrión's disease) are geographically limited in extent.

Among the protozoal infections, African trypanosomiasis is increasing both in West and East Africa but, in numerical terms, is still mainly of importance for its effect on domestic animals. South American trypanosomiasis (Chagas' disease) extends through much of the sub-continent and appears to be responsible for considerable morbidity in parts of its distribution. Malaria and leishmaniasis are widespread and cause severe morbidity and mortality in many countries.

Of the tissue filariases, those due to *Wuchereria bancrofti* and *Brugia malayi* may produce serious deformity, while the skin dwelling parasite, *Onchocerca volvulus*, causes blindness in parts of tropical Africa and Central America.

While the diagnosis of the viral and bacterial infections must be confirmed by appropriate serological and cultural techniques, the protozoal and helminthic infections are readily recognised, if not on clinical grounds alone, then by fairly simple techniques designed to demonstrate the presence of the actual causative parasites.

THE ARBOVIRUSES

Yellow Fever

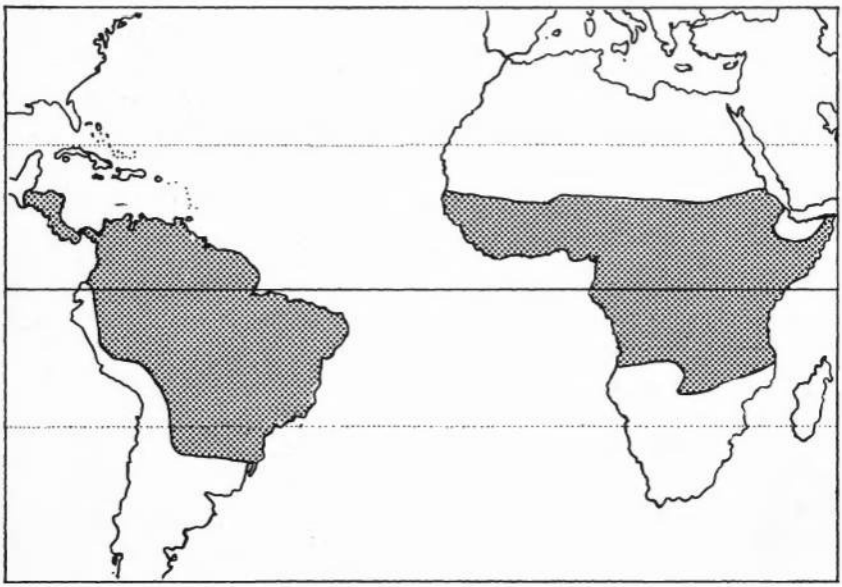
1 Distribution map of yellow fever virus Yellow fever occurs today in South America and tropical Africa. It is mosquito transmitted. Vaccination provides a high level of protection for 10 years and is a legal requirement for travellers entering endemic countries. The main differential characters of the common types of mosquito vectors of disease are shown in the next figures (*see also* **62, 216–223**).

2–4 Mosquito eggs *Culex* (**2**) eggs are deposited on the water surface in 'rafts'. *(× 25)* *Aedes* (**3**) eggs are laid singly. They often have a conspicuously sculptured surface. *(× 25)* *Anopheles* (**4**) eggs have lateral floats. They tend to aggregate on the water surface forming 'Chinese figure' patterns. *(× 25)*

5–7 Mosquito larvae The larvae of *Culex* (**5**) and *Aedes* (**6**) are suspended under the air water interface by their siphons. Those of *Anopheles* (**7**) lie parallel to the surface. *(× 4½)*

8 & 9 Mosquito pupae Pupae of culicines (**8**) and *Anopheles* (**9**) are very similar. They obtain air through siphons on the cephalothorax. *(× 6)*

1



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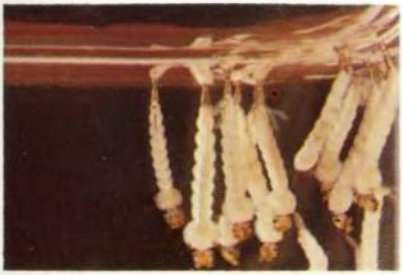

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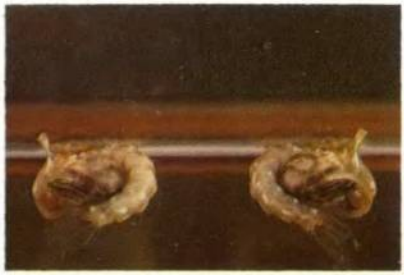

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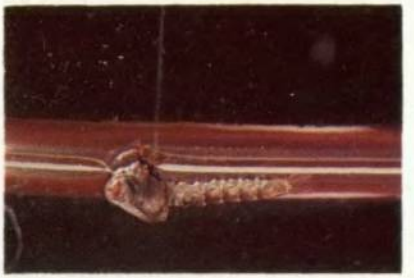

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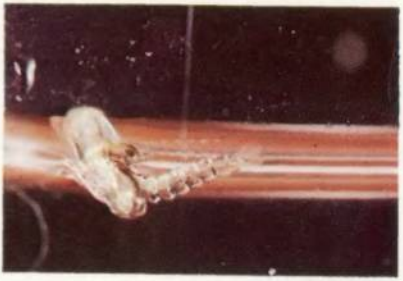
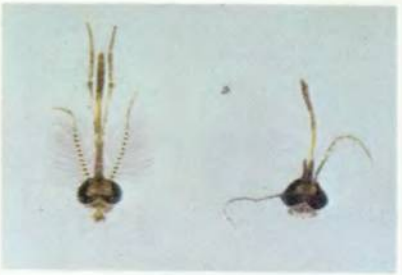


12

10–12 Adult female *Culex* hatching from pupa Mature pupa, *(× 5)*, **(10)**; moment of hatching, *(× 5)*, **(11)**; newly emerged female **(12)**, *(× 4)*

13–15 Heads of adult culicine and anopheline mosquitoes Adults of the different families and genera are recognised by the form of the antennae and palps. *Culex* ♂ ♀ **(13)**; *Aedes* ♂ ♀ **(14)**; *Anopheles* ♂ ♀ **(15)**. *(× 25)*

13



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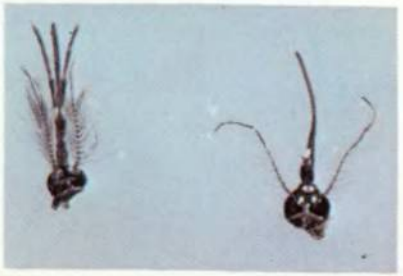

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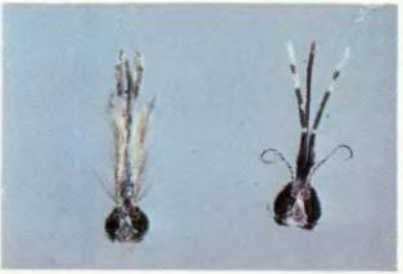

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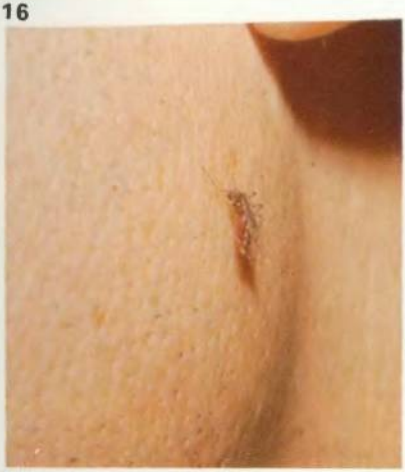

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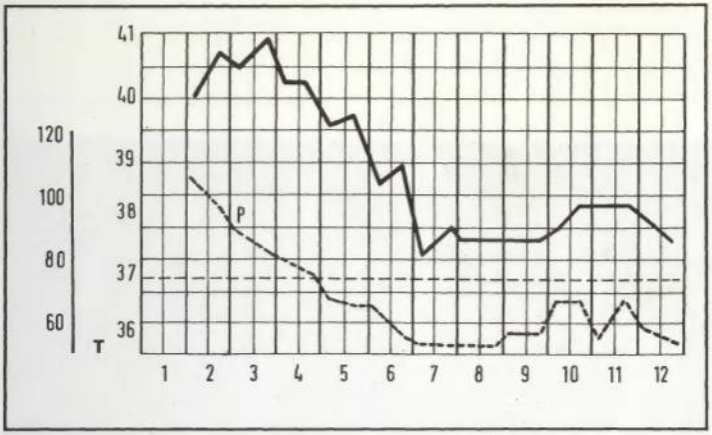
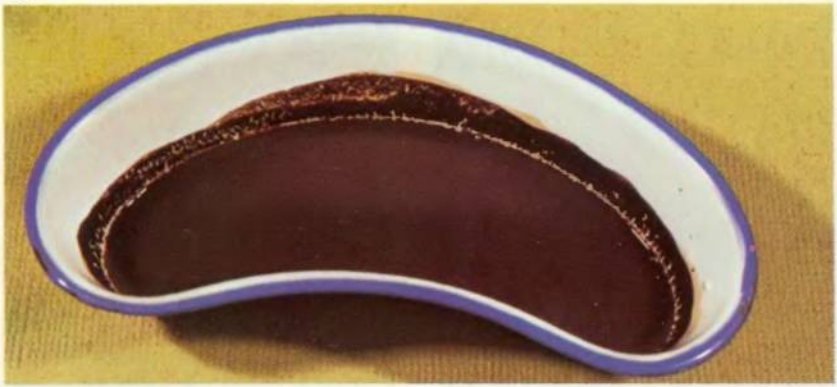


16 *Aedes* vector biting Photograph taken at midday in rain forest near Belém at the mouth of the River Amazon. Mosquitoes of the genera *Aedes* (*Stegomyia*) and *Haemagogus* transmit the virus from forest monkeys, which form a sylvatic reservoir for yellow fever virus, to man, and subsequently from man to man.

17 Water containers near houses – vector breeding sites *Aedes aegypti* breeds in domestic water containers and is responsible for urban epidemics.

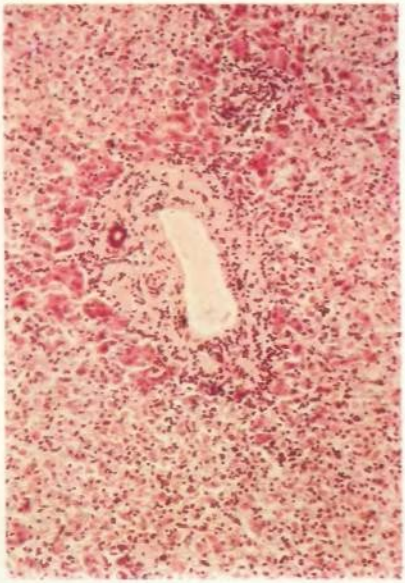
18 Temperature chart of yellow fever case The increasing slowness of the pulse relative to the temperature (Faget's sign) is of clinical diagnostic value. D refers to day of illness, P refers to pulse and T to temperature °C.

19



19 'Black vomit' of yellow fever Despite the name, jaundice is usually not marked in yellow fever. Bleeding from the gut is a grave portent, and vomiting of material resembling coffee grounds such as that shown here occurs.

20



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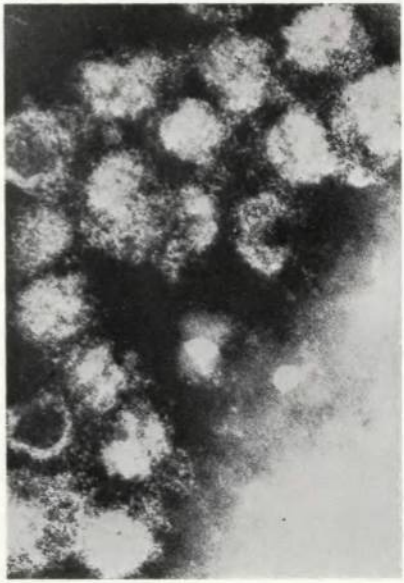
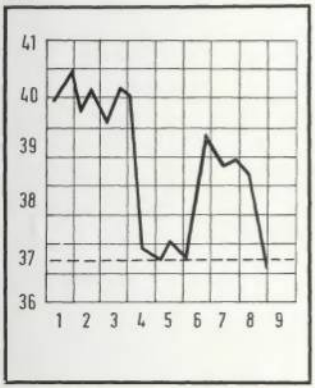


20 Section of liver from fatal yellow fever case Midzonal necrosis and Councilman bodies are characteristic histological features. *(H&E × 90)*

21 Electron micrograph of yellow fever virus This virus, one of the RNA Group B arboviruses, is shown here after negative staining. *(× 210 000)*

Dengue

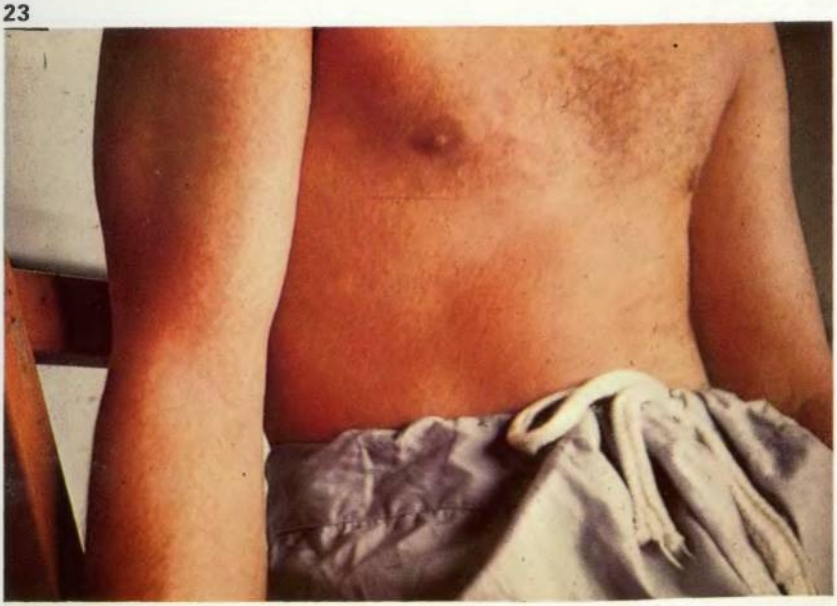
22



22 Dengue temperature chart Dengue is a mosquito-borne disease of wide distribution in the tropics and subtropics. It is of short duration with a characteristic 'saddle-back' fever.

23 Rash of dengue An erythematous generalised rash appears, usually during the second bout of fever.

23



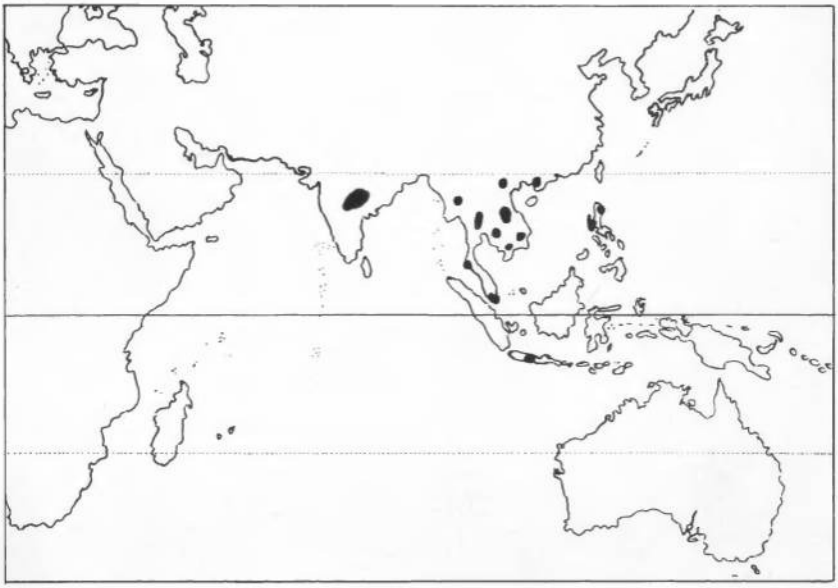
Southeast Asian Haemorrhagic Fever

24 Distribution map of SE Asian haemorrhagic fever During the last two decades epidemics of dengue haemorrhagic fever (DHF) have occurred in SE Asia. All four known types of dengue virus have been isolated in almost all the countries involved and *Aedes aegypti* has been identified as the main vector.

25 Haemorrhagic rash on arms Cutaneous haemorrhagic manifestations ranging from petechiae to gross ecchymoses characterise the infection, especially in children.

26 Marked ecchymoses in a Chinese boy.

24



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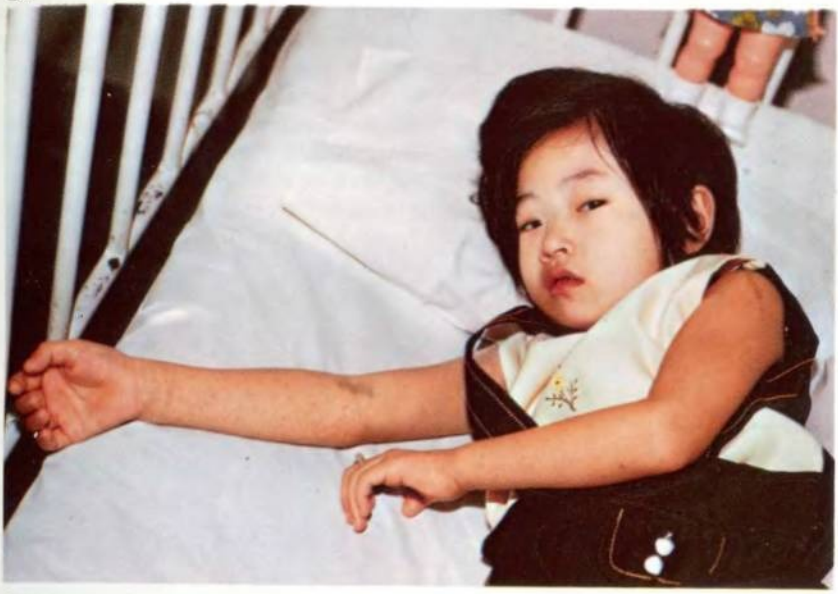

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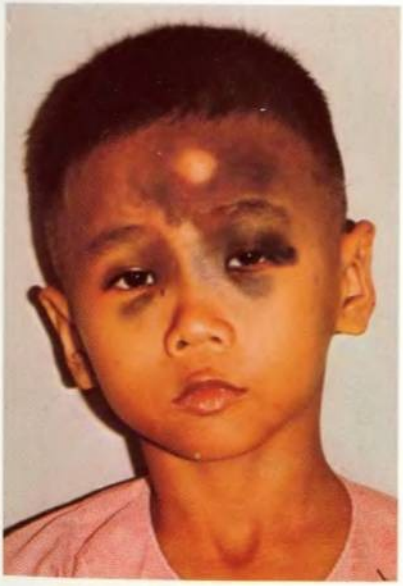


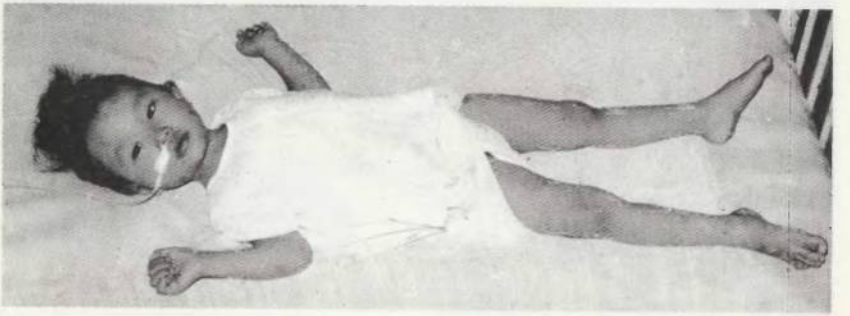
Japanese B Encephalitis

The distribution of Japanese B Encephalitis is similar to that shown in **24**. The virus is spread through birds, and transmitted by various species of *Culex* mosquitoes. These are commonly found in surface water such as flooded paddy fields. Pigs form a reservoir for the virus.

27 Encephalitis due to Japanese B virus Encephalitis is severe and results in serious sequelae such as mental retardation. This eight-year-old Chinese boy had conjugate deviation of the eyes to the right and required tube feeding.

28 Section of brain showing neuronal damage Neuronal degeneration and necrosis are commonly seen in many parts of the brain. A striking change is destruction of the Purkinje cells in the cerebellum. *(× 200)*

27



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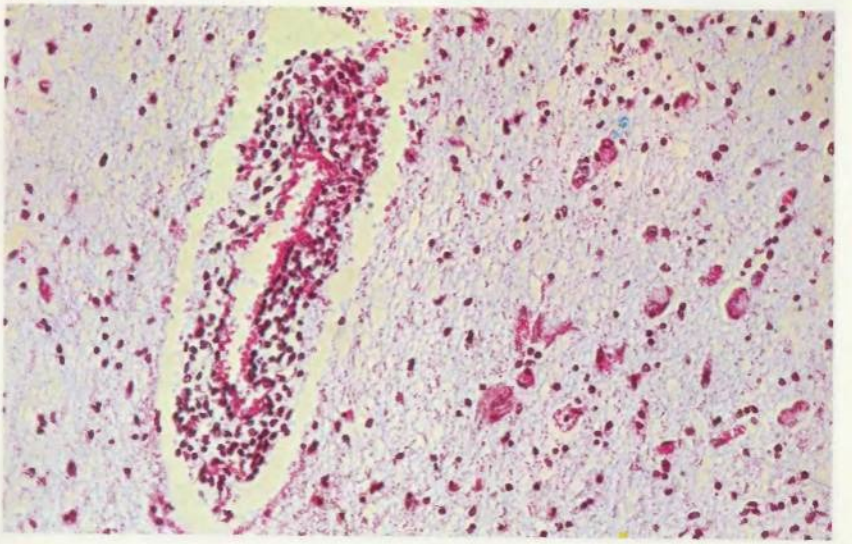

THE RICKETTSIOSES*

Louse-borne Typhus

29 Body louse The body louse *Pediculus humanus* transmits typical epidemic typhus due to *Rickettsia prowazeki*, and Trench Fever. Rickettsial infection is cosmopolitan. The use of DDT for disinfectation of louse-infested communities is a primary control measure in epidemic situations. *(× 10)*

30 Ultrastructure of *Rickettsia prowazeki* Electron micrographs show that the rickettsial organisms have structural affinities to the bacteria. *(× 55 000)*

31 Rash of typhus in an Ethiopian The generalised macular or maculopapular rash is similar in all types of rickettsial infections. The discrete rash shown here has a typical purplish colour.

29

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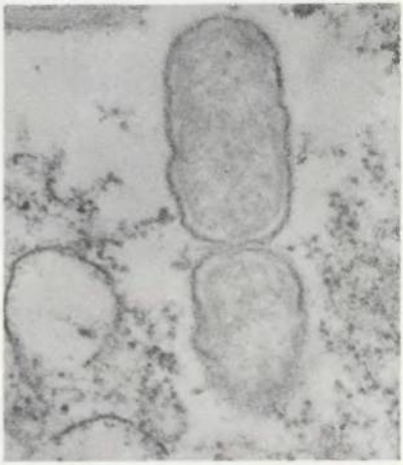

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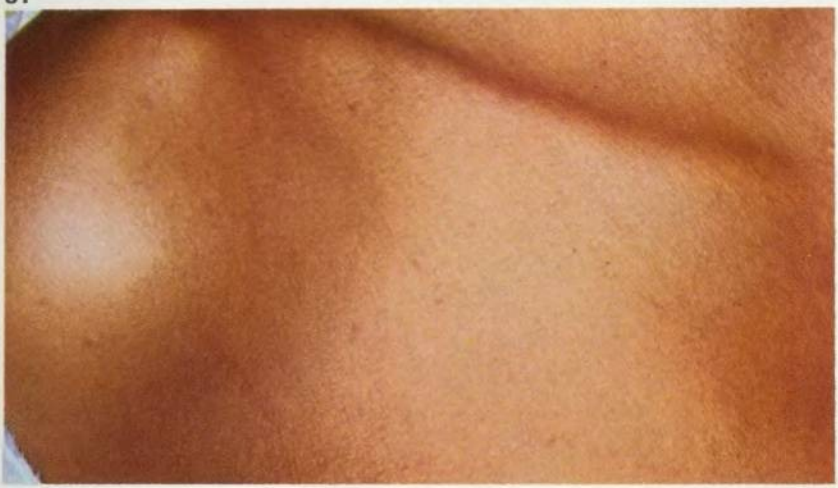

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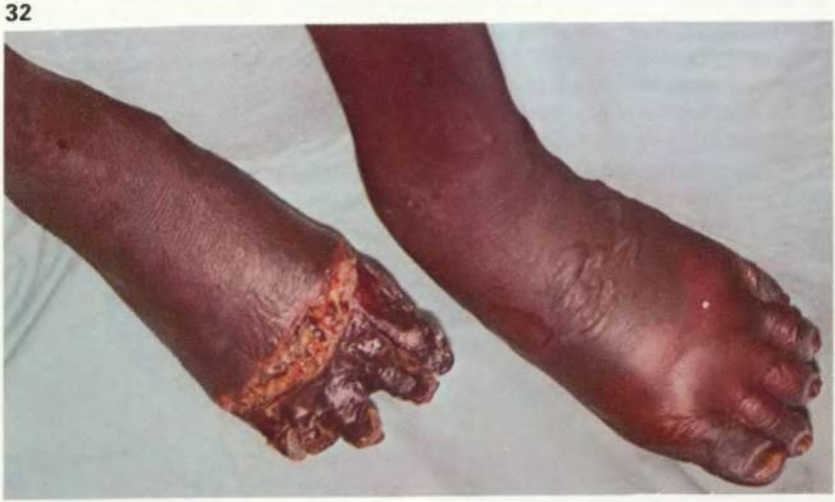

32 Peripheral gangrene in severe typhus One of the characteristic features of typhus is the severe toxicity of the infection. Gangrene of feet and hands occasionally occurs.

*(*See* **Table III**)*

Scrub Typhus

33 Distribution map of scrub typhus Scrub typhus occurs in the Indian subcontinent, SE Asia, the Far East and parts of the Southwest Pacific. *R. tsutsugamushi* is present in trombiculid-infested rodents in specialised ecological niches known as mite islands particularly in Southeast Asia and islands of the Western Pacific. Not far from Kuala Lumpur, for example, *Leptotrombidium deliensis* is found in the forest, and *L. akamushi* in the grass ('lalang').

34 Larva of *Leptotrombidium* Larval mites transmit the infection from rodent to man when he comes into accidental association with mite infested terrain. The mites act as reservoirs since transovarial transmission of the *Rickettsia* occurs. *(× 125)*

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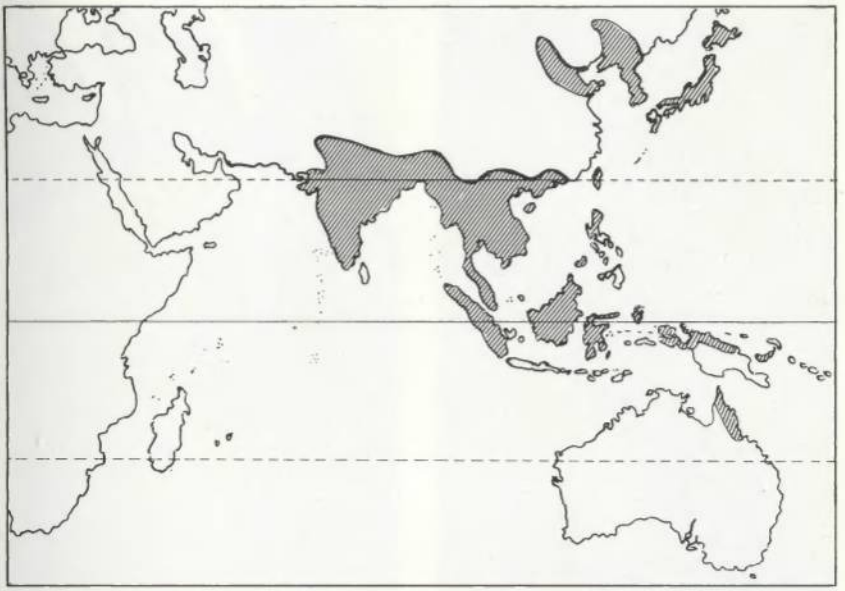

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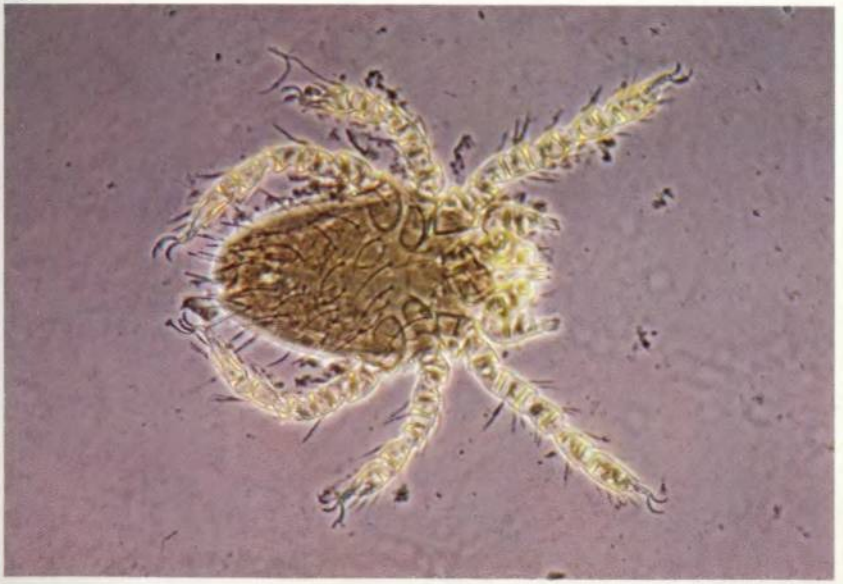

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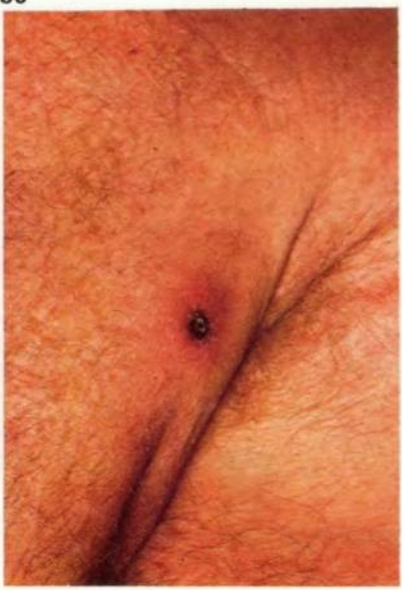

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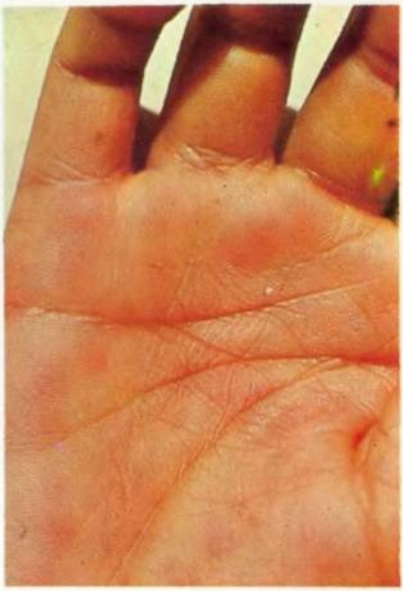

35 Eschar of mite bite A typical 'eschar' forms at the site of the trombiculid bite and precedes the fever.

36 Scrub typhus rash The maculopapular eruption appears on about the sixth or seventh day of the illness, and lasts 3 or 4 days. While seen mainly on the trunk, upper arms and thighs, it may also appear on the face, hands (as shown here) and feet.

Tick Typhus

37 Adult female *Amblyomma hebraeum*, a vector of East African tick typhus Various species of hard ticks transmit a variety of *Rickettsia* species, eg *R. conori* var. *pijperi* in Africa. *(× 7)*

38 Eschar at site of tick bite As with mite-borne typhus an 'eschar' forms at the site of the infective tick bite.

Q Fever

39 Cross section of *Coxiella burneti* Q fever due to *Coxiella burneti* of ruminants is cosmopolitan. It may be spread to man by ticks, but is mainly acquired by direct contact with milk. *(× 57000)*

Murine typhus may be acquired when rickettsia-infected rodents harboured in deserted farmhouses, transmit infection by their fleas to newcomers who, for example, utilise such buildings as holiday homes.

37

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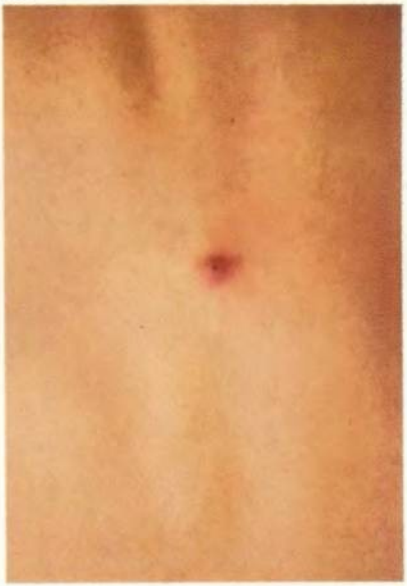

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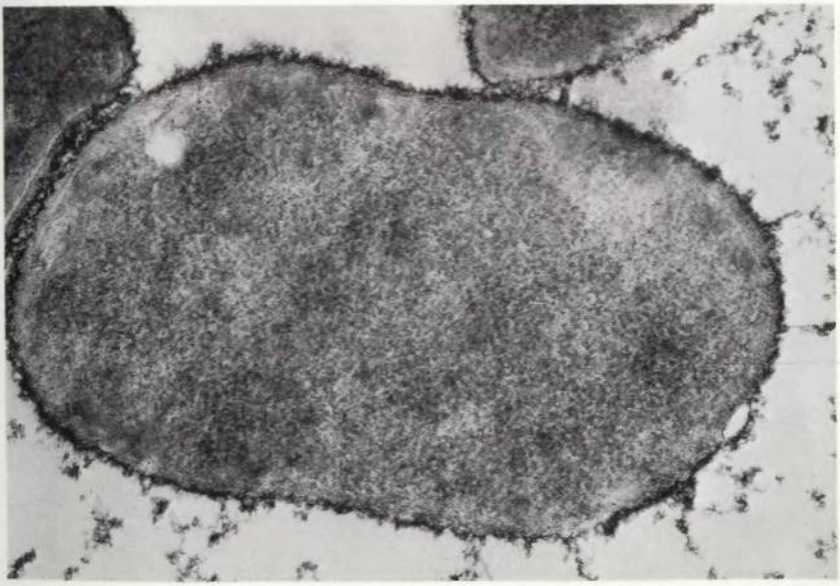
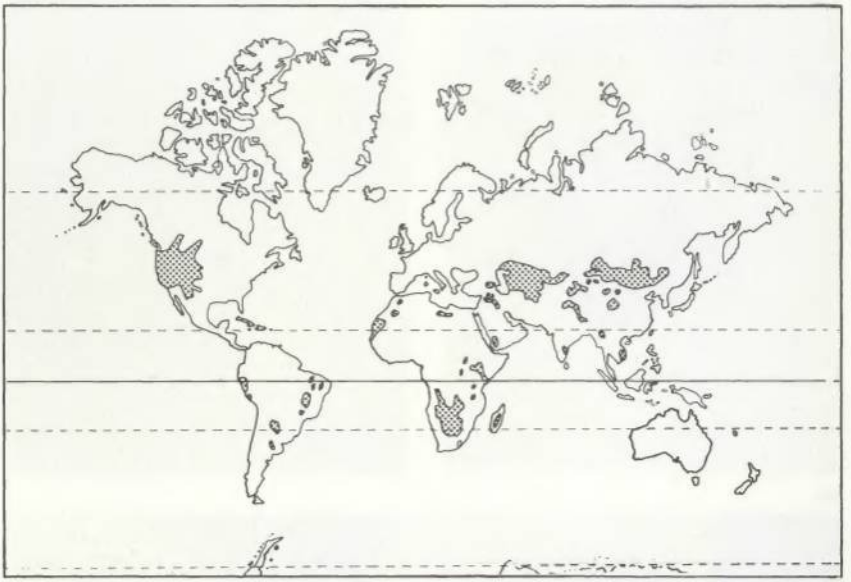


BACTERIAL INFECTIONS

Plague

40 Known and probable foci of plague Plague is now largely focal in distribution.

40



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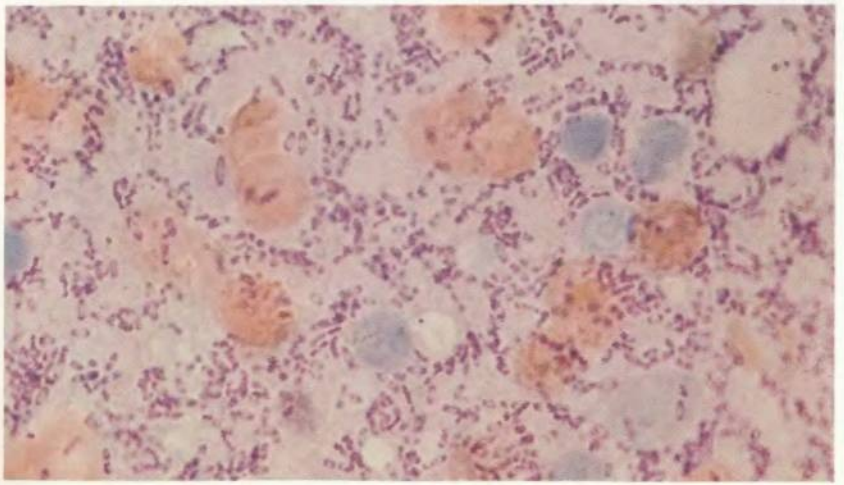
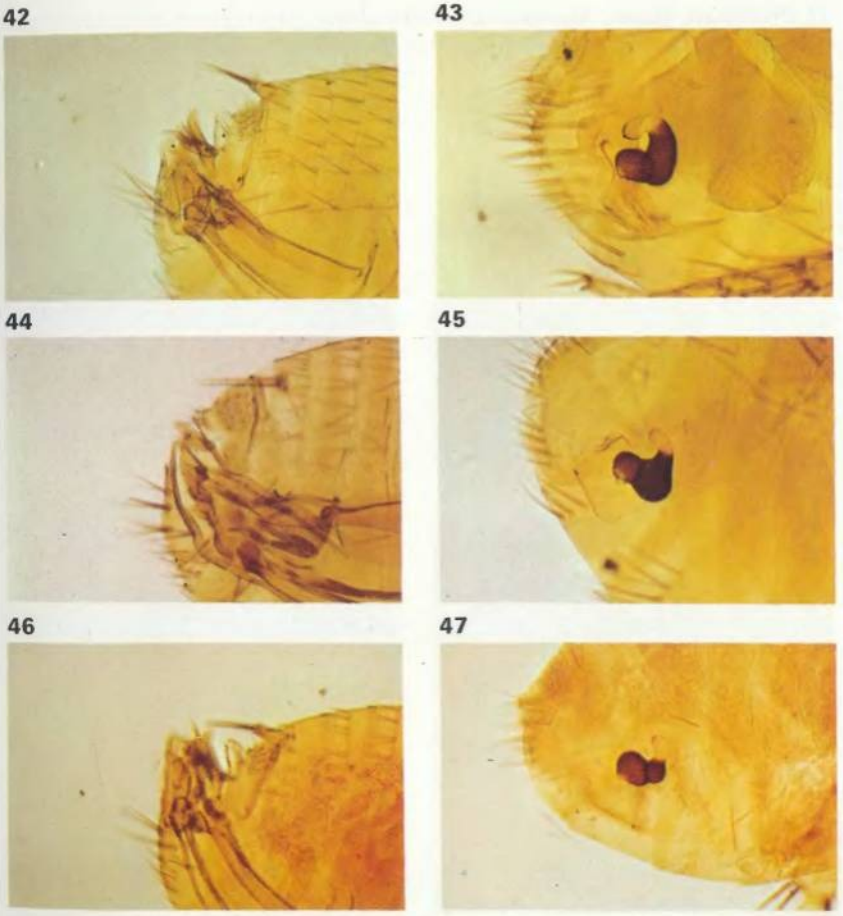


It spreads rapidly in conditions of war and other catastrophes, eg earthquakes. Recent epidemics have occurred in Vietnam.

41 *Yersinia pestis* in liver smear *Y. pestis* (syn. *Pasteurella pestis*) is a gram negative cocco-bacillus with bipolar staining. It is normally enzootic in rats. *(× 1 250)*

42–47 Male and female *Xenopsylla* fleas compared Plague is transmitted by fleas of the genus *Xenopsylla*. *X. cheopis* ♂ ♀ (**42 & 43**); *X. astia* ♂ ♀ (**44 & 45**); *X. braziliense* (**46 & 47**). The rat flea *X. cheopis* is the main vector. *(× 60)*



48 Proventriculus of *X. cheopis* blocked by plague bacilli Hungry fleas leave dying domestic rats (*R. norvegicus*, *R. rattus*) often with their foreguts blocked by bacilli. In this condition they will attempt to feed on any animal. If they bite man they transmit the disease. *(× 120)*

49 Metal guards preventing access of rats along ships' hawsers Plague control demands strictly enforced rodent control measures and international quarantine regulations, particularly for shipping.

50 Bubonic plague One of the most characteristic clinical features is lymphadenopathy with suppuration especially in the inguinal and axillary regions.

51 Pneumonic plague Pneumonic infection allows direct spread of bacteria from man to man. The X-ray shows infection in the left lower lobe on the second day of the illness.

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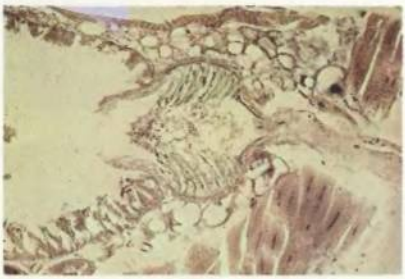

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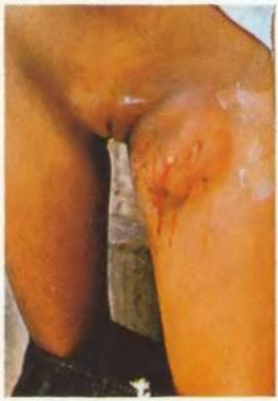

51

52 *Borrelia duttoni* in human blood film from Tanzania Endemic relapsing fever, a cosmopolitan disease caused by *B. duttoni*, occurs in rodents and man. First described from Africa, it occurs also in the Middle East, Mediterranean basin, and the New World including the USA. *(Giemsa – phase contrast × 900)*

53 Soft tick with coxal fluid Soft ticks such as *Ornithodorus moubata* transmit *B. duttoni* from rodents to man, and man to man through infected coxal fluid which enters skin abrasions. As the organism passes from tick to tick by transovarial transmission, the arthropod is itself a reservoir host. *(× 4)* In East Africa *O. moubata* lives in soft earth at the base of mudlined walls.

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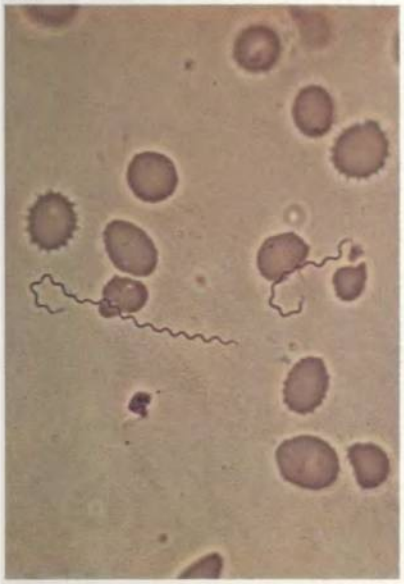

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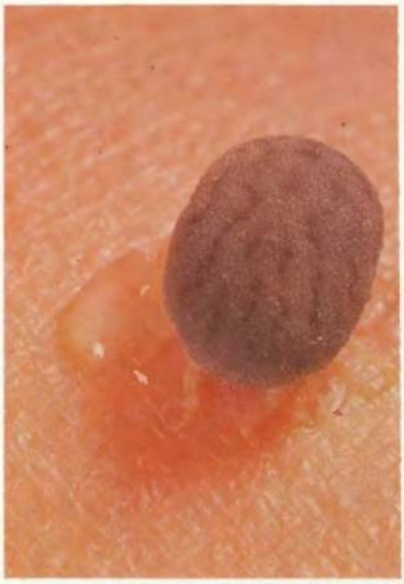
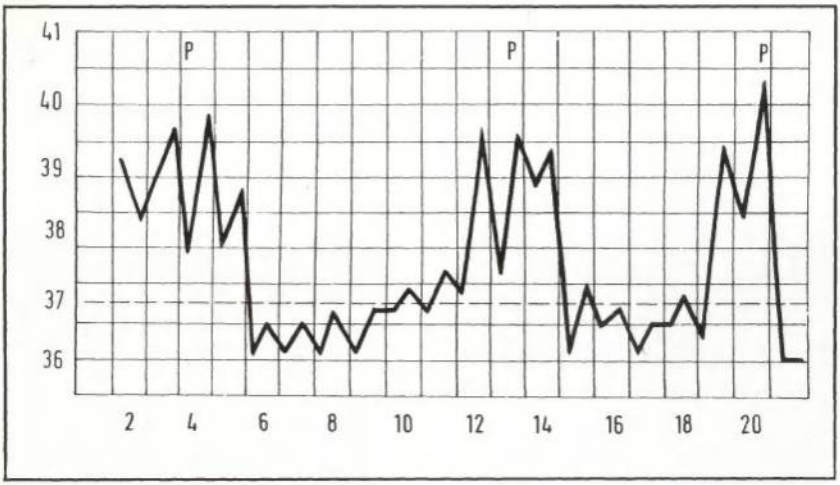


54 Temperature chart of tick-borne relapsing fever The infection acquires its name from the typical relapsing nature of the fever. *B. duttoni* only appears in the blood during the febrile episodes which are numerous and of short duration.

55 Scanning electron-micrograph of *Borrelia recurrentis* in blood *B. recurrentis* transmitted by body lice occurs in epidemic form in Ethiopia and Eritrea. The febrile periods recur on one or two occasions only, but mortality can be high. Infection is acquired from the body fluids of lice crushed on the skin while scratching. *(× 1100)*

56 *B. recurrentis* in blood film The organisms are readily seen in this Indian ink preparation. *(× 600)*

54



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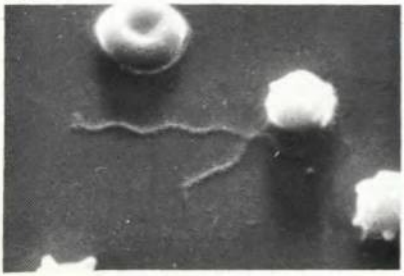

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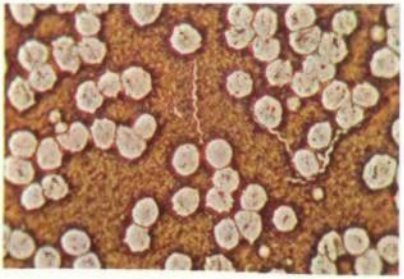

Bartonellosis (Carrión's Disease)

57 Blood film showing *Bartonella bacilliformis* *B. bacilliformis* is a gram negative intraerythrocytic bacillus which produces fever, and acute haemolytic anaemia (Oroya fever) in Bolivia, Peru, Colombia and Ecuador. The bacilli also invade reticulo-endothelial cells. *(Giemsa × 900)*

58 'Verruga Peruviana' The infection sometimes appears as a generalised verrucous eruption, sometimes haemorrhagic in nature. Organisms can be cultured from the cutaneous nodules. The disease is transmitted by the sandfly *Lutzomyia verrucarum* at altitudes of 800–3000 m.

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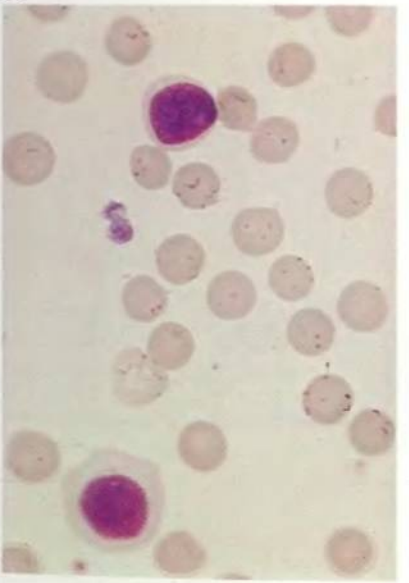

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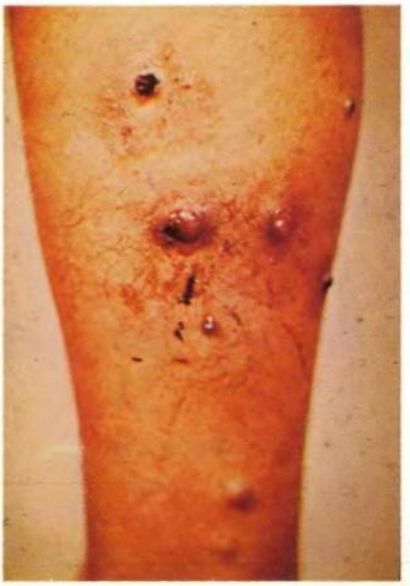


PROTOZOAL INFECTIONS*

Malaria

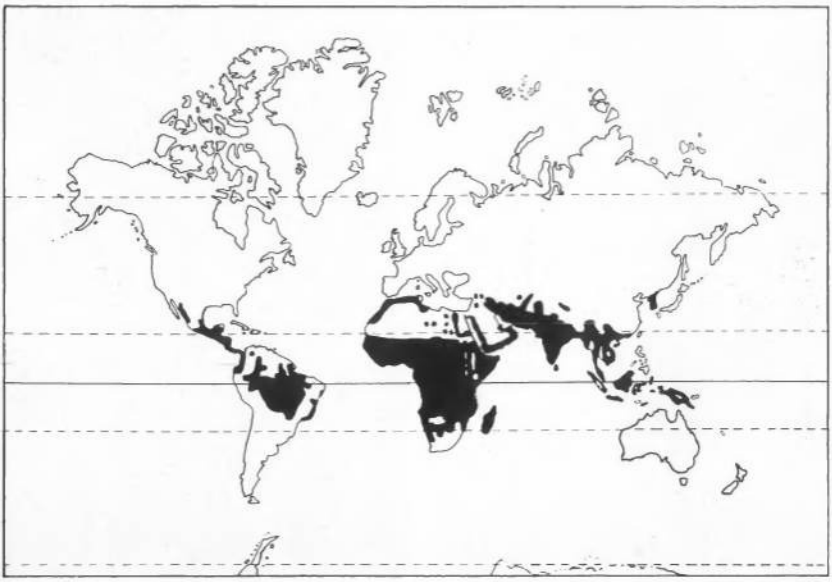
59 Distribution map of malaria In spite of intensive control measures over the last 20 years, malaria is still widely distributed in the tropics and subtropics. *Anopheles gambiae* is the most dangerous malaria vector in tropical Africa. It breeds in small temporary collections of fresh surface water exposed to sunlight and in such sites as residual pools in drying river beds. Most important vectors in other parts of the world are also surface water breeders. Successful malaria control operations in recent years have been based largely on the destruction of house haunting anopheline vectors by spraying DDT and other insecticides on the interior walls where mosquitoes usually rest before and/or after feeding.

*(*See* **Table IV**)

60 Bromeliad leaf axils, sites for specialised anopheline larvae Some South American vectors of the subgenus *Anopheles* (*Kerteszia*) are found in bromeliads. Control of these larvae by insecticides is extremely difficult.

61 Anopheline pupa hatching *(× 4)*

59



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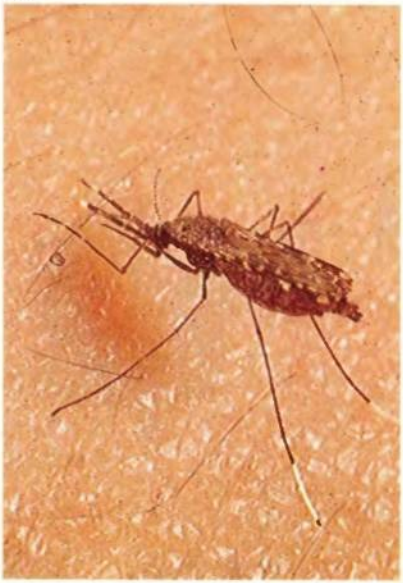


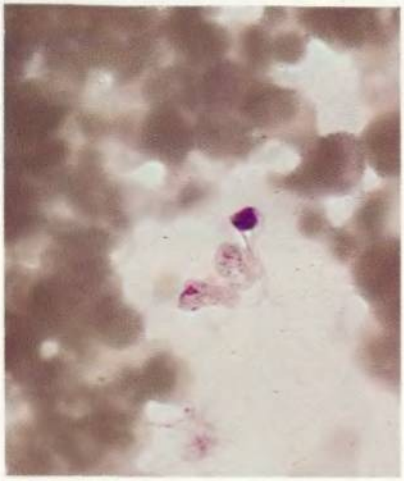
62 Female anopheline biting Malaria is transmitted by female *Anopheles* mosquitoes. Most species bite indoors at night but some are outdoor feeding. The adults are recognised by the antennae and palps (*see also* **15**). *(× 4)*

63 Life cycle of the malaria parasite Male gametes develop by exflagellation from microgametocytes in the midgut of the female *Anopheline*. *(× 900) (see also* **764***)*

64 Ookinetes in midgut Male and female gametes fuse to produce motile ookinetes which enter midgut epithelial cells. *(× 900)*

65 Scanning electronmicrograph of oocysts outside anopheline midgut Infective stages (sporozoites) develop in oocysts which lie on the outside of the mosquito midgut. *(× 125)*

63



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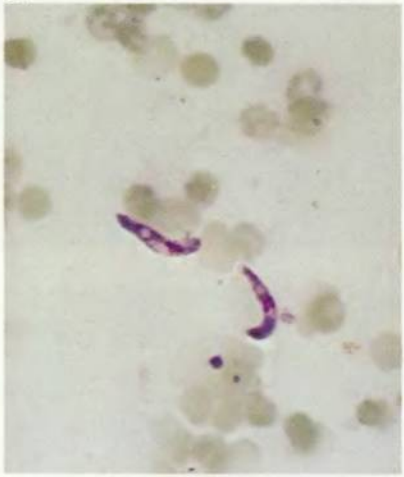
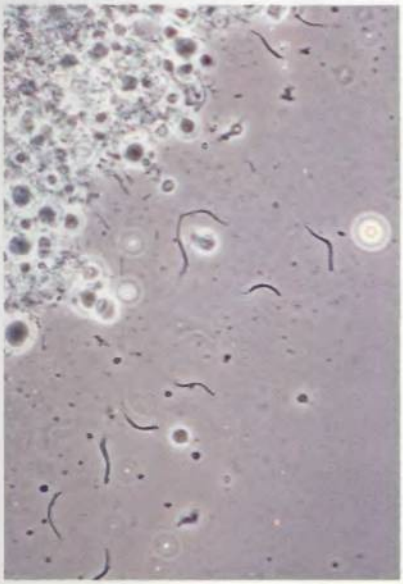


65

66 Living infective sporozoites Sporozoites emerge from the oocysts and enter the insect's salivary glands. They are passed into the skin with saliva when the mosquito next takes a blood meal. *(× 350)*

67 Exoerythrocytic schizont in liver Within 30 minutes the sporozoites enter the parenchymal cells of the host's liver where they form large 'tissue' schizonts. These mature in six to 14 days according to the species, liberating daughter cells called merozoites. *(Giemsa – colophonium technique × 350)*

66



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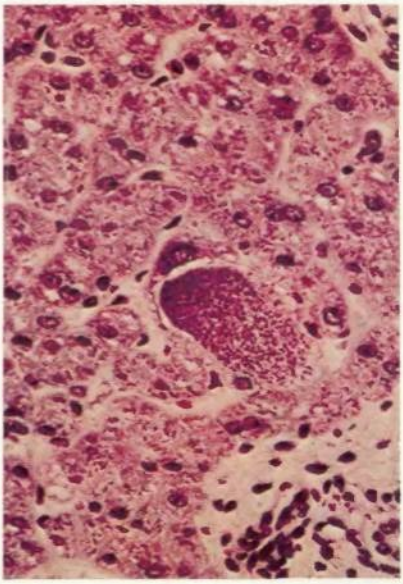
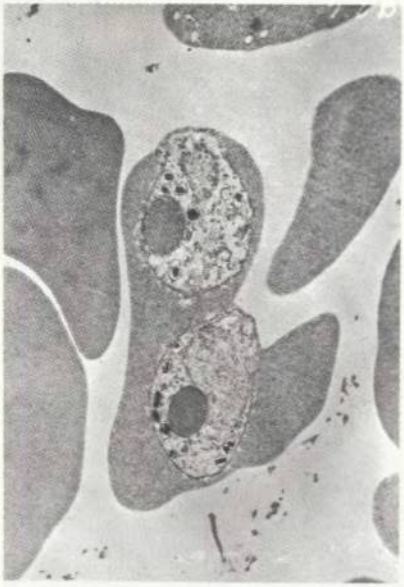


68 & 69 Electronmicrographs of trophozoites and mature schizont of rodent malaria parasite in red cell The merozoites form asexual parasites which grow inside erythrocytes to form the trophozoites (**68**). These feed on red cell contents producing insoluble pigment (haemozoin) as a waste product. When growth is complete the parasites undergo cell division (schizogony) (**69**) and the daughter cells, after rupture of the host, invade new red cells. *(× 11 000)*

70 Tertian and quartan fever patterns The asexual blood stages of *P. falciparum*, *P. vivax* and *P. ovale* require 48 hours to complete their schizogony. Fever is

68



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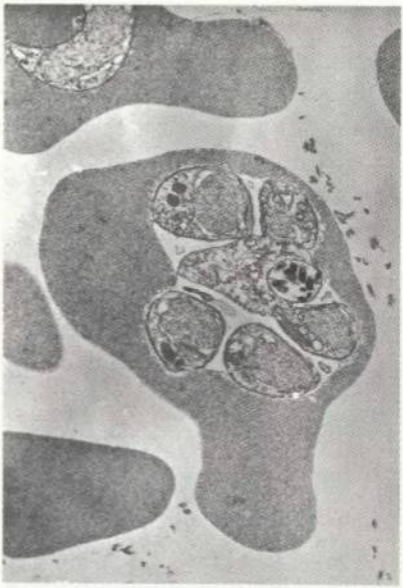

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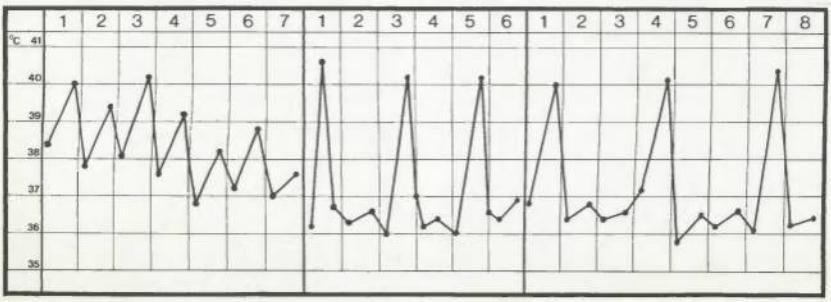
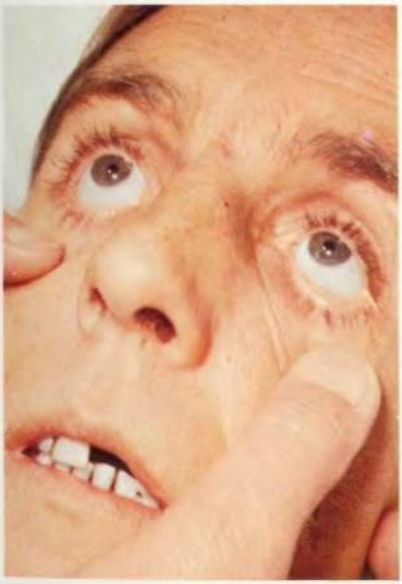


produced when the schizonts mature, ie at 48 hour intervals. This gives the classical tertian periodicity which is however uncommon in a primary attack of *P. falciparum* malaria. *P. malariae* requires 72 hours and is associated with quartan fever, ie 72 hours between paroxysms.

71 Malarial anaemia The growth of intraerythrocytic parasites leads to disruption of the host cells. This (and possibly also auto-immunity) results in severe haemolytic anaemia. Jaundice can also occur.

72 New Guinea child with grossly enlarged liver and spleen Haemolysed red cells and parasite debris are phagocytosed by macrophages particularly of the spleen and liver which become enlarged. This child was seen on a field survey.

71



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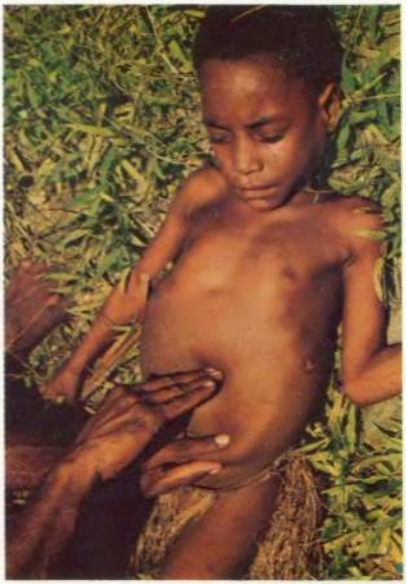

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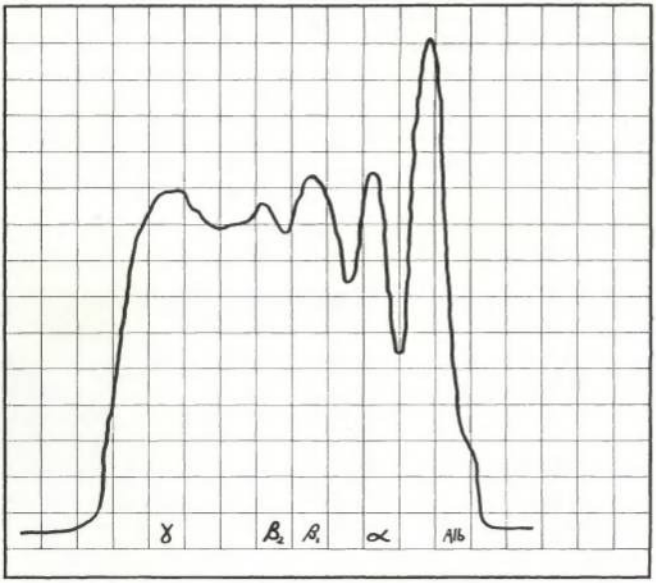


74

75

76

77

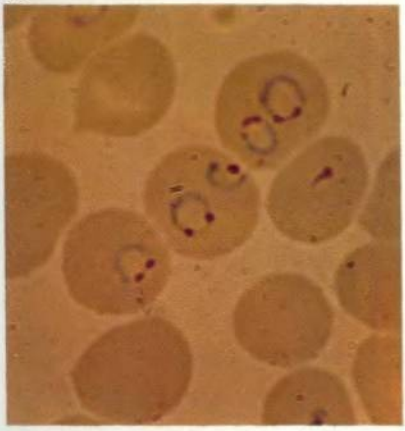
73 IgG increase in malaria The characteristic immunological response is an increase in the IgG level. Cellular immunity also plays an important role.

74–77 Preparation of blood films Diagnosis of malaria is based primarily on the recognition of parasites in well prepared thick and thin blood films stained with a Romanowsky stain (Giemsa, Leishman, Field, etc) at pH 7.2–7.4. A small drop of blood from finger or ear is placed on a clean slide (**74**). The thin film is made by pulling a second slide away from the drop (**75**). Spreading the drop for a thick film (**76**). Comparative thicknesses of thin and thick films (**77**).

P. falciparum

78–81 Life cycle of the blood stages Fine rings (**78**) predominate, mature trophozoites and schizonts (**80**) appearing uncommonly in the peripheral circulation because parasites mature in capillaries of the internal organs. Host cells are not enlarged. Spots of irregular shape and size (Maurer's dots) may be seen in older rings (**79**). Crescent-shaped gametocytes (♂ **81a**, ♀ **81b**) are diagnostic. *(Giemsa × 1 800)*

78



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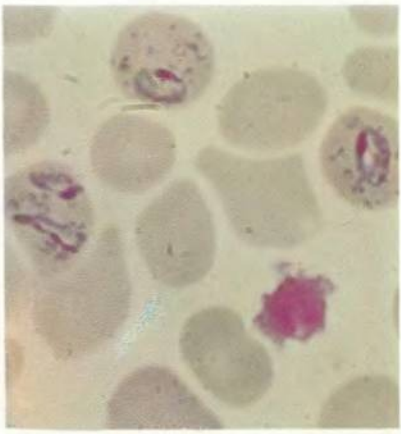

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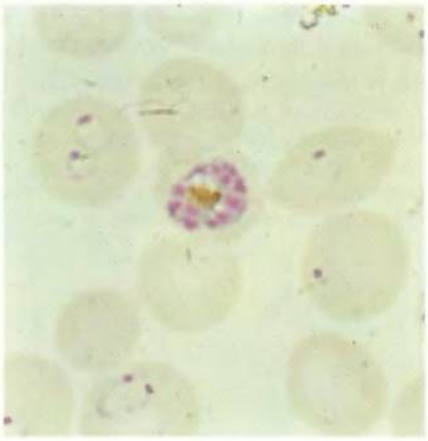
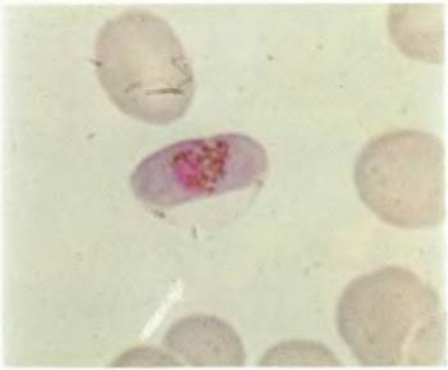
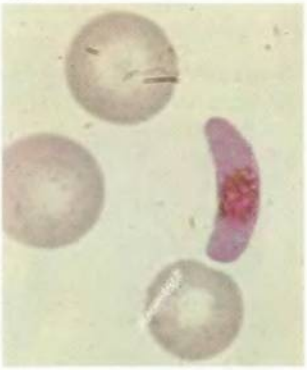


81a



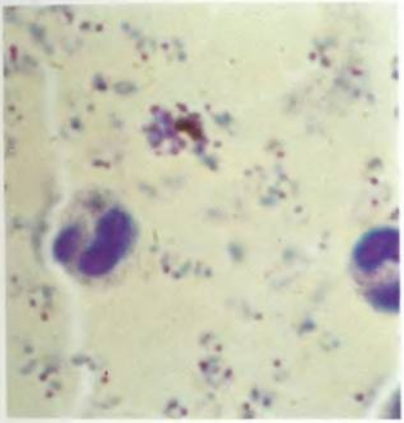
81b



82 & 83 Thick blood film Usually only young rings (**82**) are seen in acute infections sometimes in very large numbers. Heavy parasitaemia leads to severe haemolytic anaemia. Gametocytes (**83**) appear about a week after the onset of the illness. *(Field × 1800)*

84 Blackwater urine and serum taken during course of illness An acute haemolytic crisis resulting in haemoglobinuria occasionally occurs in severe attacks (Blackwater fever). Haemoglobinuria can also be drug induced in patients deficient in the enzyme glucose 6-phosphate dehydrogenase (G-6-PD). A = normal urine; B = patient's urine; C = patient's urine, diluted; D = normal serum; E = patient's serum.

82



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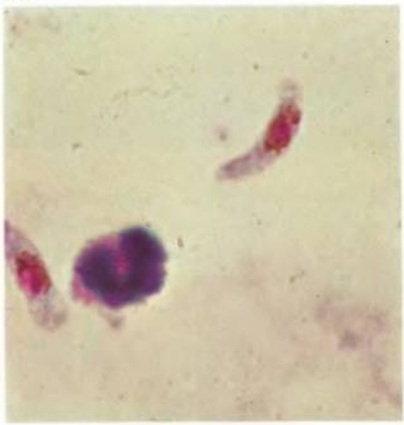

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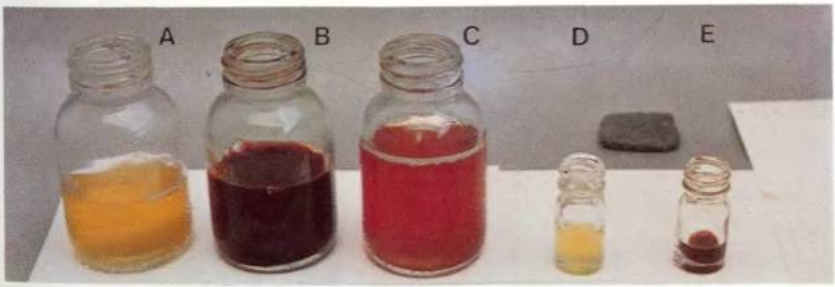
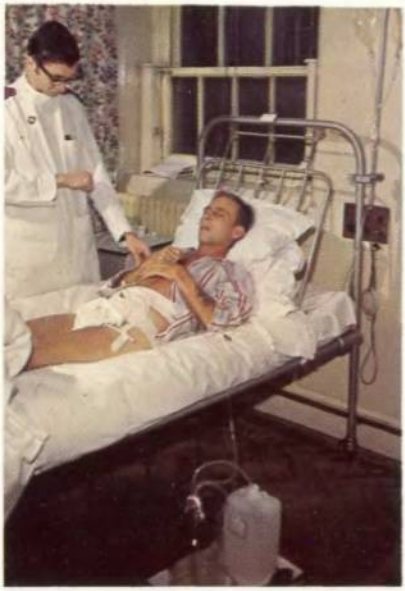
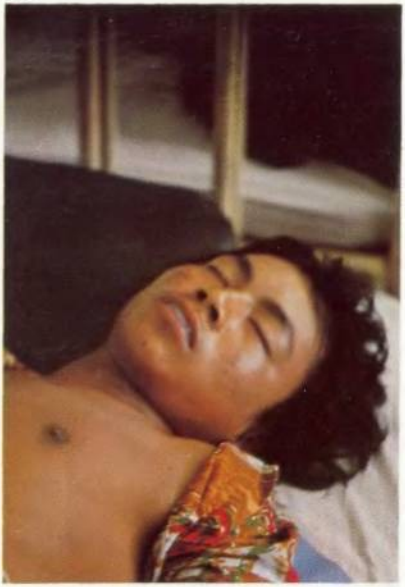


85a



85b

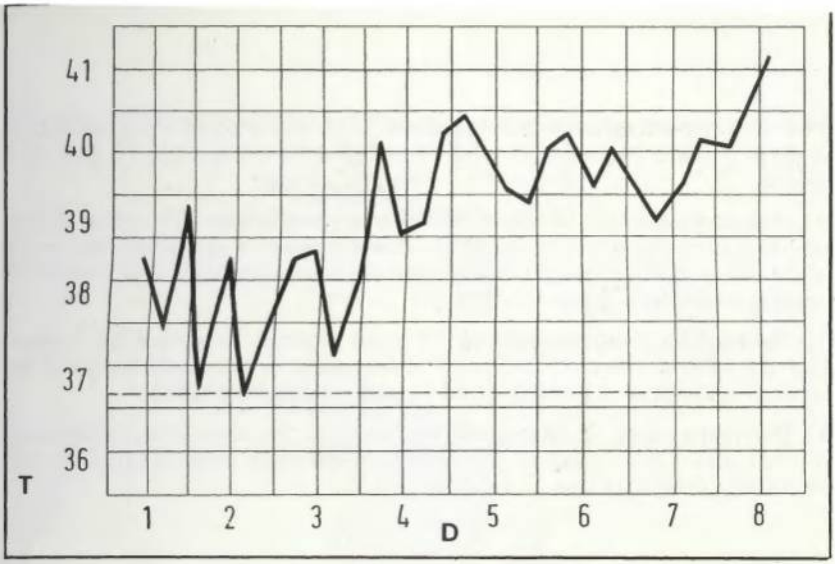


85 Complications of malignant tertian (falciparum) malaria **A** Acute renal failure. Peritoneal or haemodialysis are life saving measures. **B** Coma in cerebral malaria. This is one of the commonest and most lethal complications. Confusion is an early warning sign.

86 Temperature chart In first infections the fever is usually irregular rather than tertian. No relapses occur after adequate treatment with blood schizontocides since no secondary liver schizogony takes place in this species (cf *P. vivax*).

87 Gross section of brain in cerebral malaria Cerebral malaria results when cerebral capillaries are blocked by developing falciparum schizonts. Consequent capillary endothelial damage results in 'ring' haemorrhages. Cerebral malaria is a medical emergency which demands immediate treatment by intravenous administration of suitable antimalarials, eg quinine. Rehydration is often also needed, but overhydration may result in pulmonary oedema.

86



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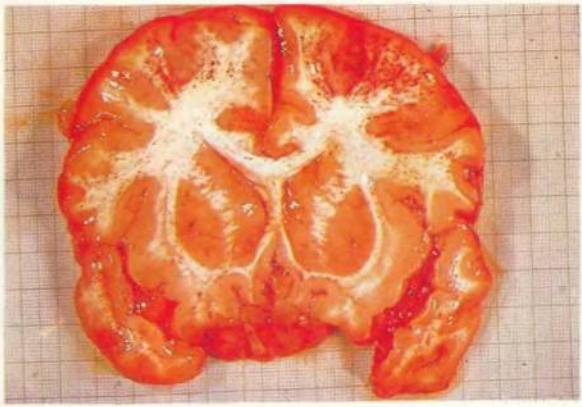

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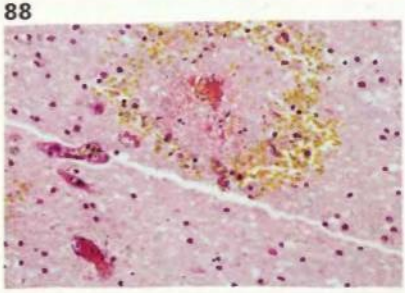


88 Microscopic section of brain *(× 100)*

89 & 90 Liver and spleen in chronic malaria In chronic infection accumulation of malaria pigment (haemozoin) produces a dark brown coloration of liver and spleen.

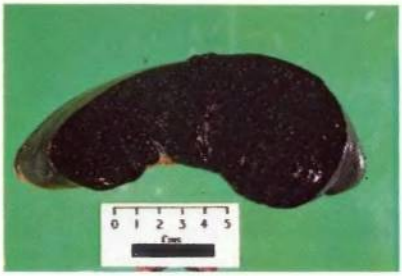
91 Placental smear with falciparum schizonts and macrophage The accumulation of falciparum schizonts in the maternal side of the placental circulation may result in the delivery of underweight infants especially in primigravidae. True congenital malaria is very rare. *(Giemsa × 600)*

92 Haemoglobin S distribution map It is now generally recognised that haemoglobin S (AS) diminishes the severity of falciparum malaria, thus favouring the survival of the gene in tropical Africa ('balanced polymorphism').

93 Distribution map of chloroquine resistance In the areas shown falciparum malaria may not respond to prevention or treatment with chloroquine and alternative drugs may have to be used.

89

90



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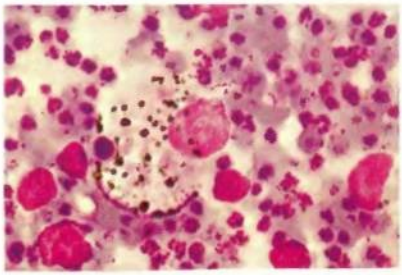

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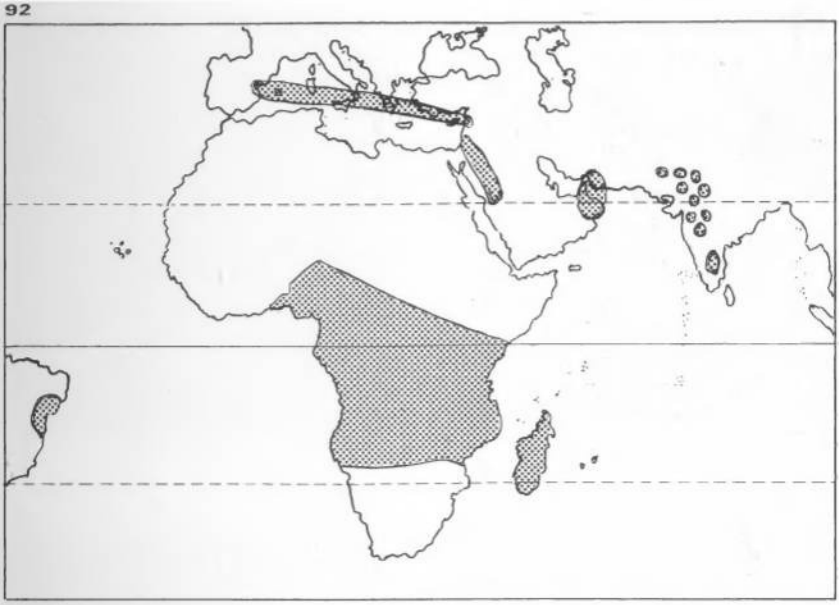

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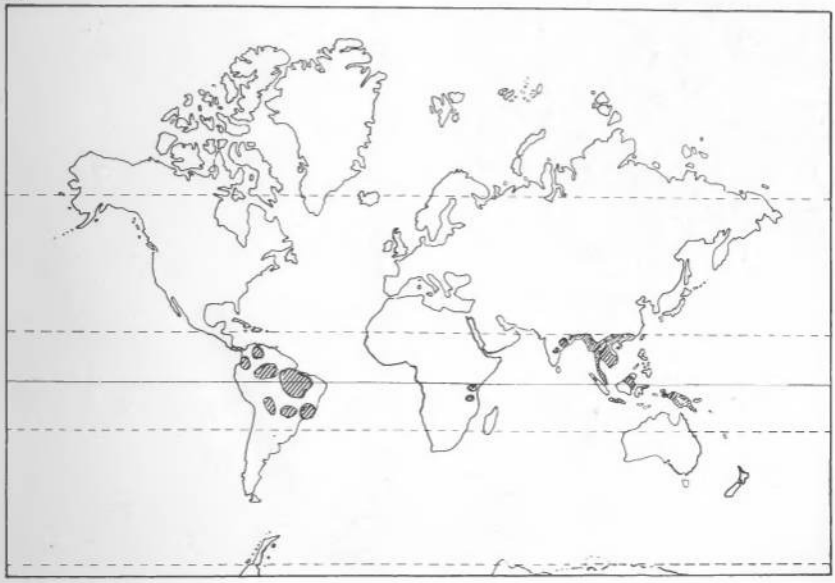
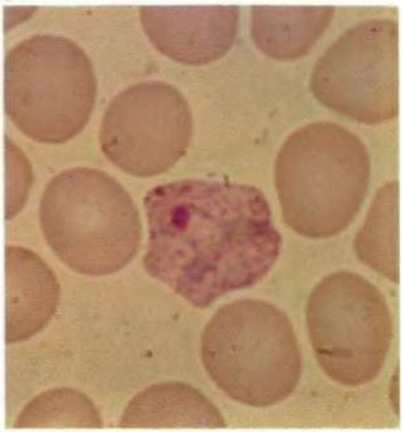


P. vivax

94–98 Life cycle of the blood stages All stages of asexual parasites from young trophozoites (**94**) to schizonts appear in the peripheral circulation together with gametocytes. The parasites are large and amoeboid (**95**), and produce schizonts with about 16 daughter cells (merozoites) (**96**). Pigment is well developed. Host red cells are enlarged and uniformly covered with fine eosinophilic stippling (Schüffner's dots). Gametocytes are round, the male or microgametocytes (**97**) being about seven μm, and the female or macrogametocytes (**98**) ten μm or more in diameter. *(Giemsa × 1800)*

94



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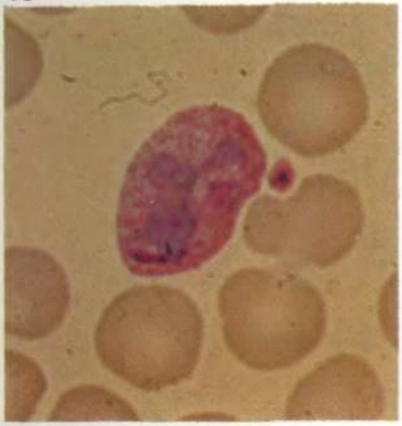

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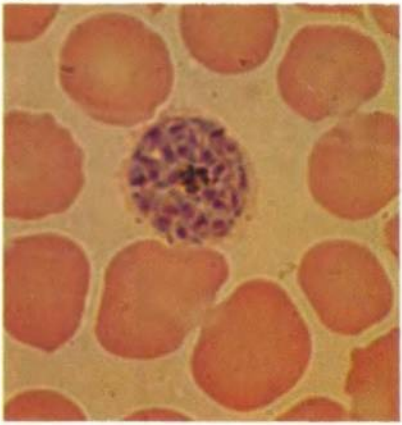

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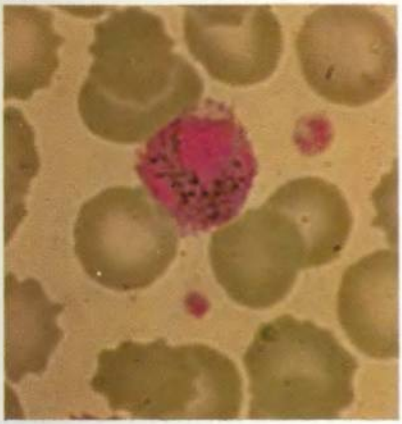

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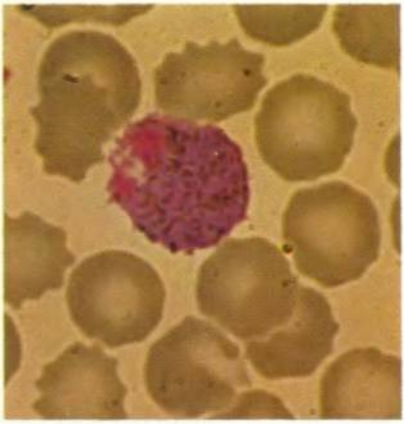
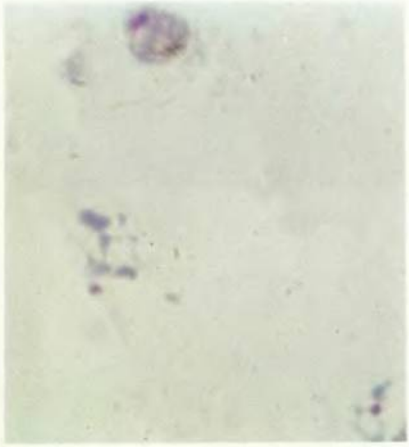


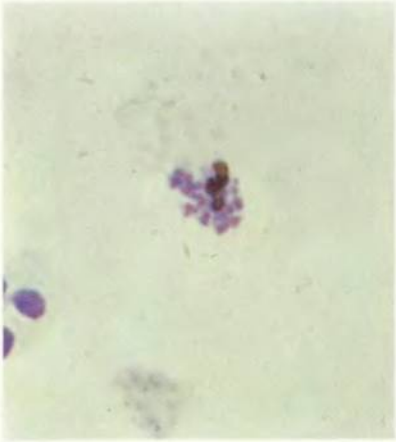
99a, b & 100a, b Thick blood film All stages may be present. Parasitaemia is often less heavy than in falciparum malaria. The parasites seen here are all in a single thick film. In the thicker part are seen an amoeboid trophozoite (**99a**), and a schizont (**99b**). Often the Schüffner's dots can be seen in 'ghost cells' in the thinner parts of the film where the host cell has been haemolysed (**100a**). In **100b** are seen three trophozoites and a macrogametocyte. *(Field × 1800)*

101 Diagram of relapse patterns in vivax malaria Relapses in vivax malaria are due to emergence of new blood forms from maturing secondary liver schizonts. In tropical areas relapses may arise within three to four months of a primary attack, but in subtropical areas usually only after nine months or more. A – Clinical symptoms; B – Overt parasitaemia; C – Subpatent parasitaemia; D – Primary and secondary tissue stages in the liver; E – Sporozoite infection; F – Exoerythrocytic schizogony; G – Radical or spontaneous cure; H – Microscopic threshold; J – Clinical (pyrogenic) threshold rising with the increased immunity; 1 – Incubation period; 1a – Pre-patent period; 2 – Primary attack composed of paroxysms; 3 – Latent period (clinical latency); 4 – Recrudescence (short-term relapse); 5 – Latent period; 5a – Parasitic latency; 6 – Clinical recurrence (long-term relapse) followed by parasitic recurrence; 6a – Parasitic relapse.

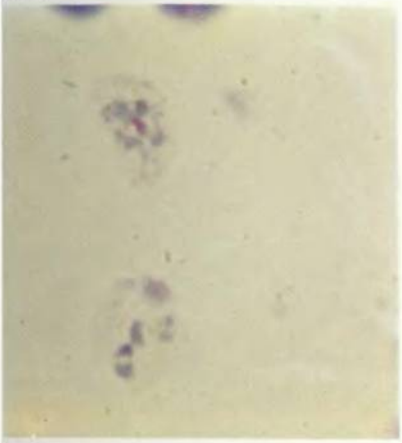
99a



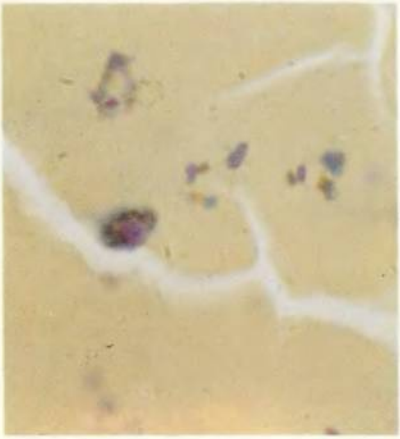
99b



100a



100b



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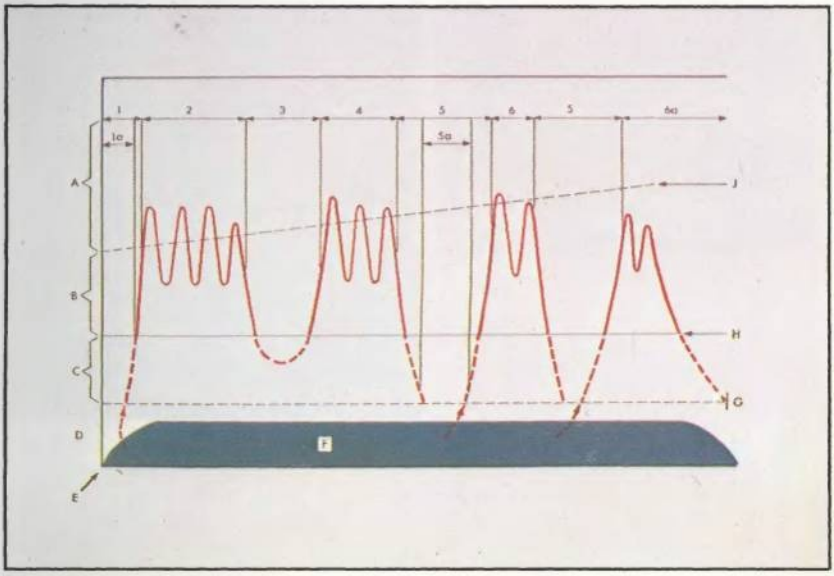
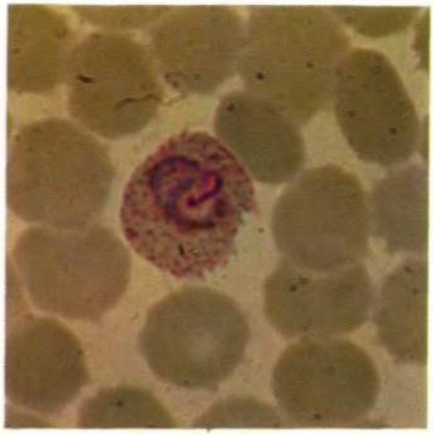


P. ovale

102–105 Life cycle of the blood stages The parasites differ from *P. vivax* in being more compact, and producing about eight merozoites at schizogony (**104**). Host red cells contain Schüffner's dots and tend to be ovoid and fimbriated. Diagnosis is difficult in thick films in which the parasites are easily confused with *P. malariae* (cf **114 & 111**). As in *P. vivax* 'ghost cells' may be seen in thinner parts of the thick film but the contained *P. ovale* are more compact (cf **114 with 100**). Relapses occur as in *P. vivax* but the disease tends to be more chronic. *(Leishman × 1800)*

102



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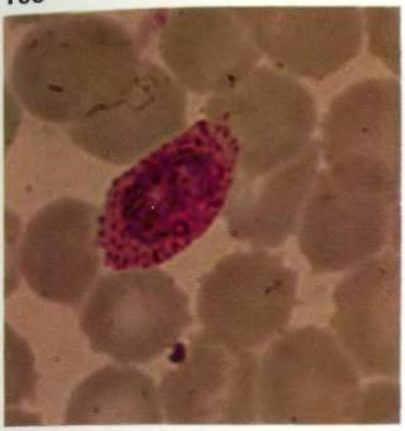

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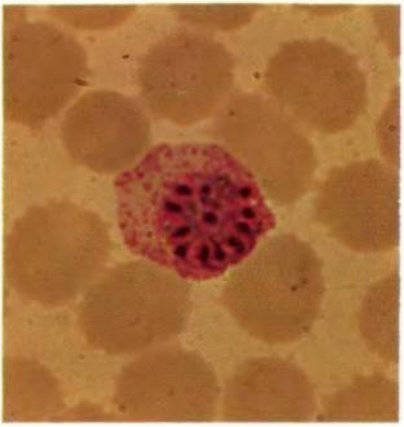
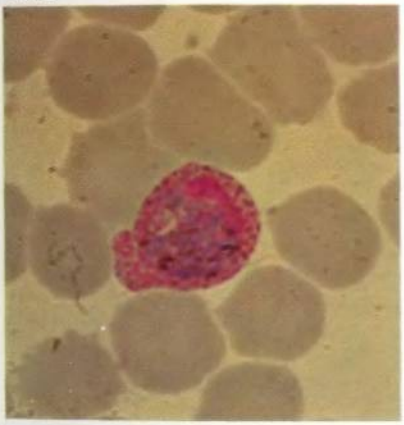
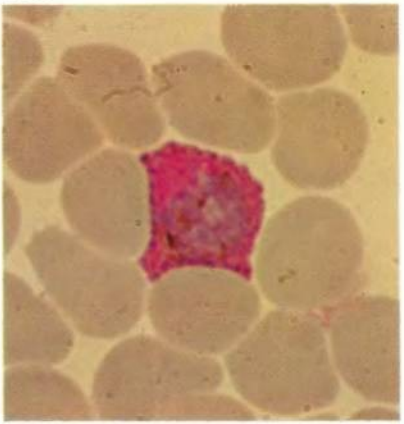


105a



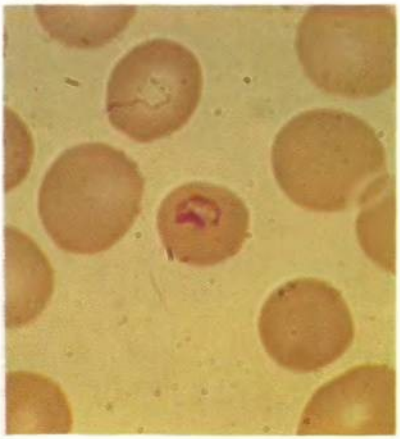
105b



P. malariae

106–110 Life cycle of the blood stages All stages appear in the peripheral circulation from young trophozoites (**106**) to compact schizonts (**108**) with eight merozoites. 'Band forms' (**107**) are common. With special staining a very fine stippling (Ziemann's dots) is sometimes seen. Host red cells are not enlarged. Gametocytes are round and compact with distinct blackish pigment, the females (**110**) usually staining a bluer colour, and the males (**109**) somewhat mauvish. *(Leishman × 1800)*

106



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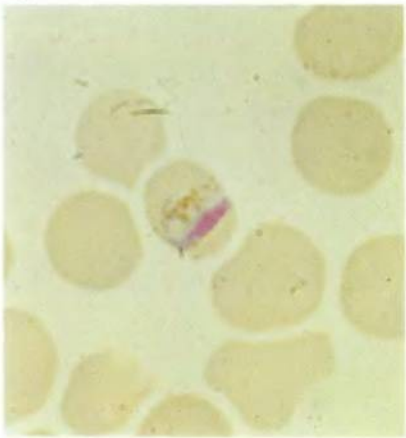

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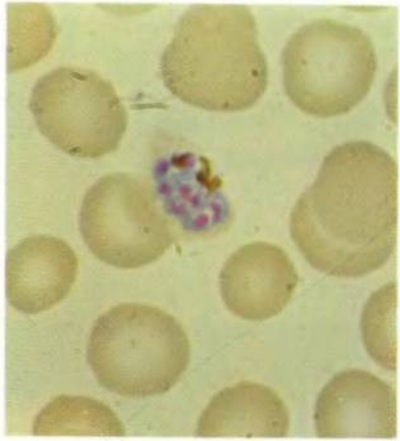

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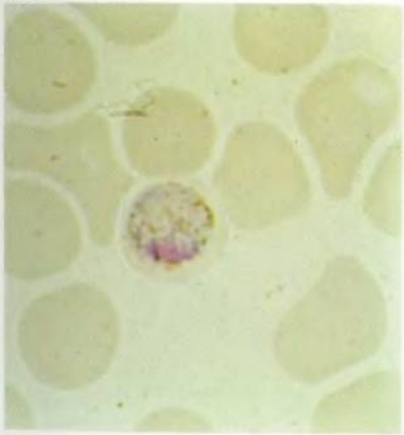

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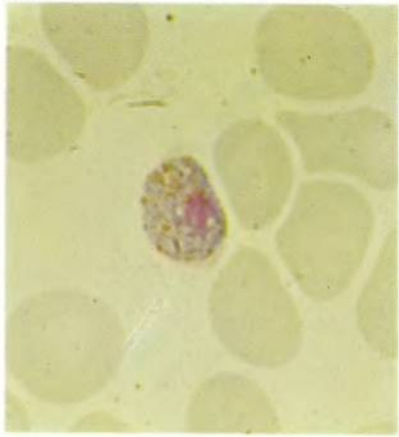
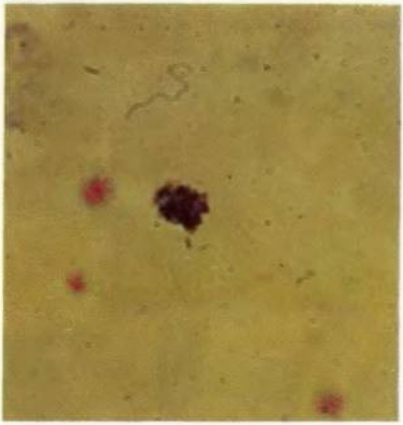


111–114 Thick blood films Younger parasites (**111**) are easily recognised by their heavy pigment which may obscure the inner structure of older trophozoites and gametocytes (**113**). Schizonts containing about eight merozoites with a central mass of pigment (**112**) are characteristic. All stages are very similar to those of *P. ovale*, which can sometimes be identified if these parasites (**114**) can be found at the periphery of the thick film in 'ghost cells'. *(Field × 1800)*

111



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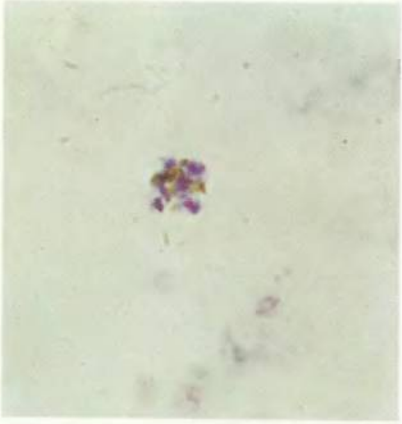

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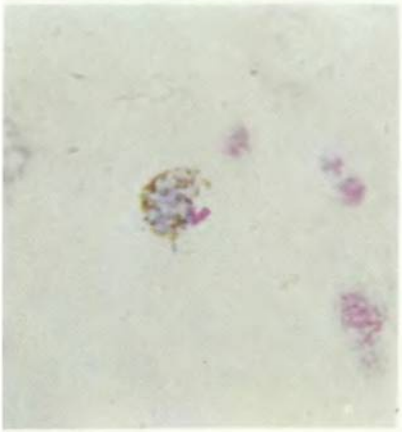

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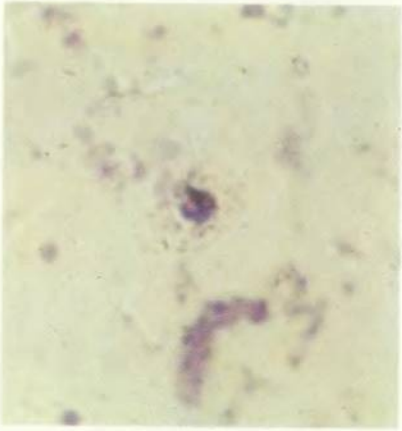
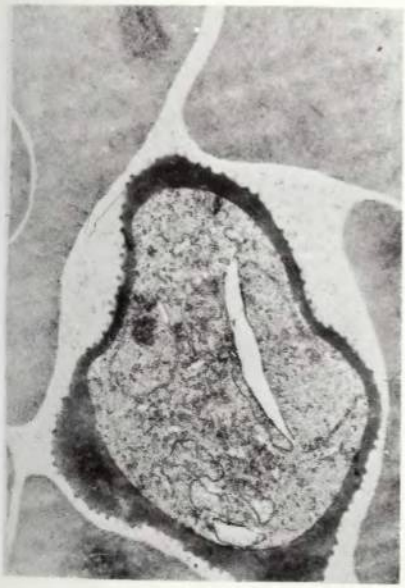


115 Ultrastructure of *P. malariae* trophozoite Very small but regular bosses occur on the surface of the host erythrocyte (possibly corresponding to the Ziemann's dots). *(× 11000)*

116 Nephrotic child with *P. malariae* infection A close association has been established between quartan malaria and the nephrotic syndrome in children. Note the gross oedema and ascites.

115



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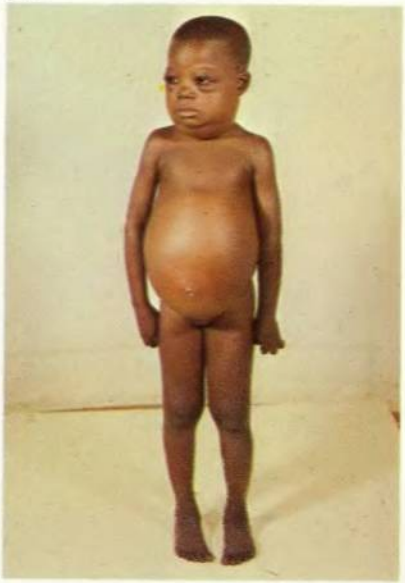


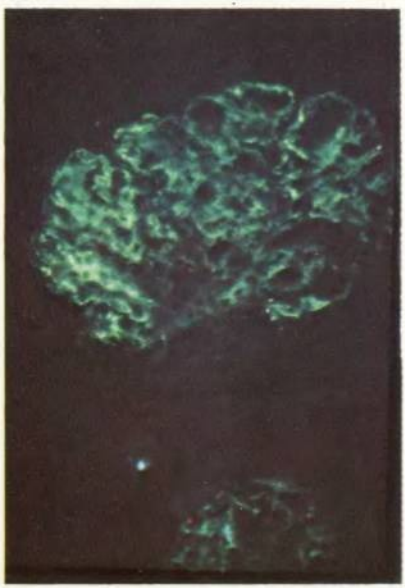
117 Immunofluorescence of immune complexes in kidney Immunofluorescent antibody techniques have demonstrated the deposition of immune complexes on the basement membrane of the glomeruli in 'quartan malarial nephrosis'. *(× 350)*

118 'Tropical splenomegaly syndrome' (TSS) Gross enlargement of the spleen is a characteristic feature of the tropical splenomegaly syndrome (TSS). The syndrome is thought to be due to an abnormal immunological response to malaria infection. High IgM levels are invariably found. Note scars due to application of indigenous medicines.

119 Massive spleen in TSS Regression of the enlarged spleen occurs when long-term antimalarial therapy is given.

120 Section of liver in TSS Liver biopsy shows hepatic sinusoidal dilatation with marked infiltration of lymphocytes and hypertrophy of the Kupffer cells. *(× 500)*

117



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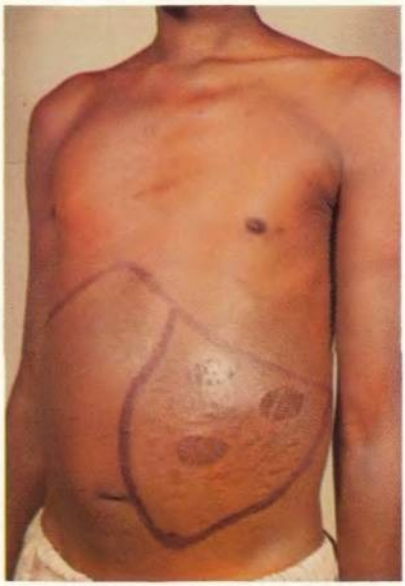

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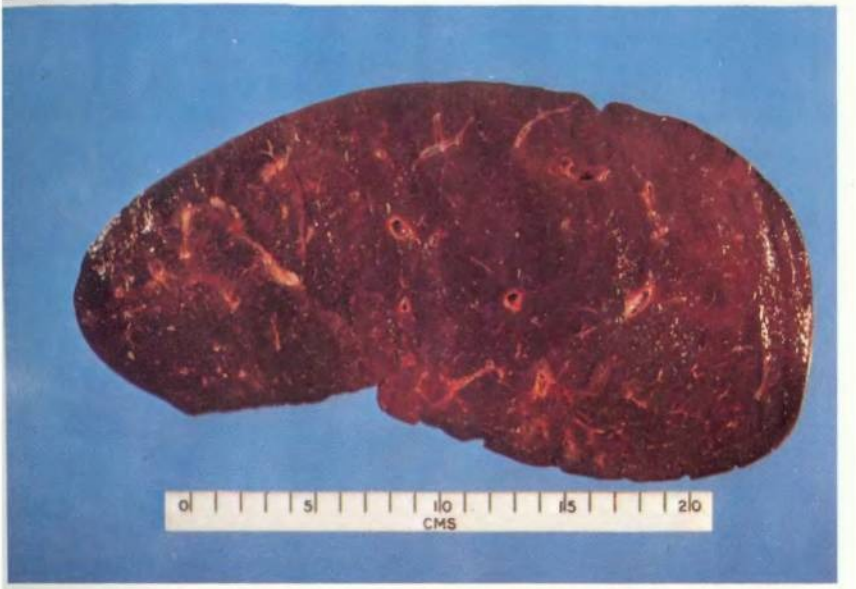

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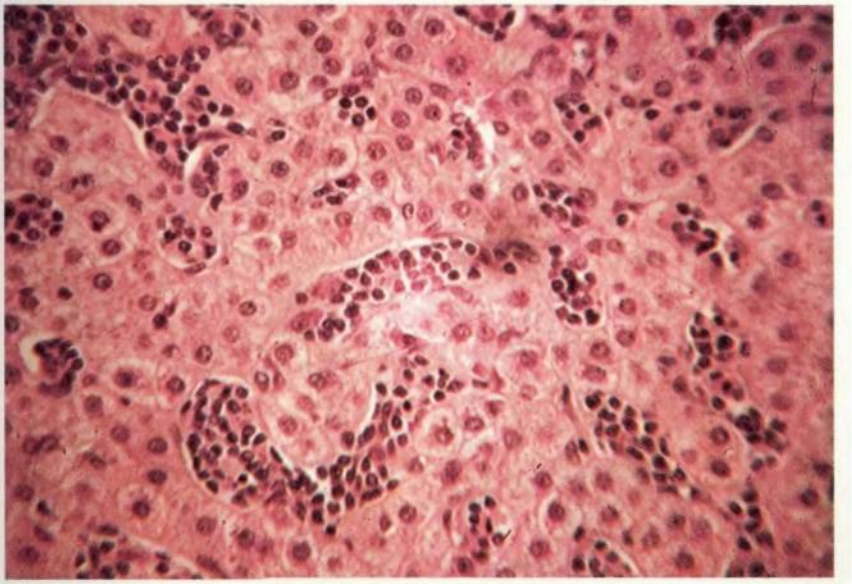
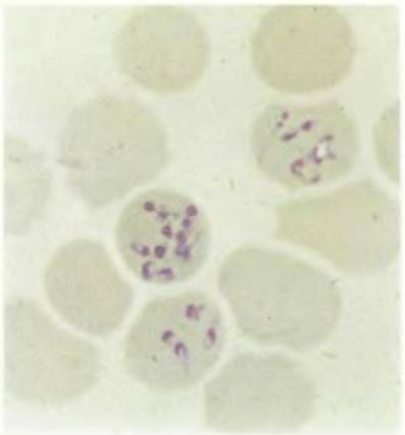


Babesiasis in Man

121 & 122 *Babesia* sp. in thin blood film Infection with species of *Babesia* from cattle or rodents is a rare occurrence in man. Parasites are transmitted by ticks. Heavy red cell infection especially in splenectomised patients may lead to fatal haemolytic anaemia. This patient died from an infection acquired in Scotland. *(Giemsa × 1800)*

121



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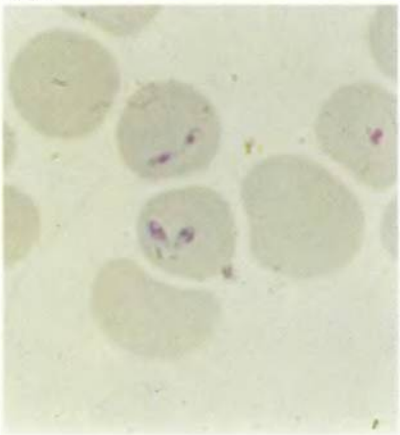
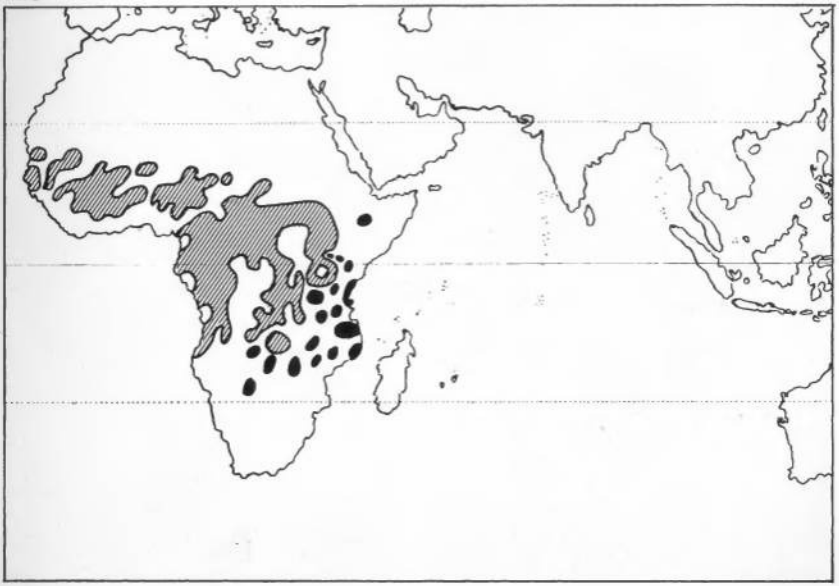


TRYPANOSOMIASIS*

African Trypanosomiasis

123 Distribution map of human disease African trypanosomiasis is confined to equatorial Africa with a patchy distribution depending upon detailed topographical conditions. It is caused by two subspecies of *Trypanosoma brucei*. *T. b. gambiense* infection is widespread in West and Central Africa but *T. b. rhodesiense* is restricted to the East and East Central areas with some overlaps between the two. Slanted lines indicate *T. b. gambiense*; solid areas *T. b. rhodesiense*.

123

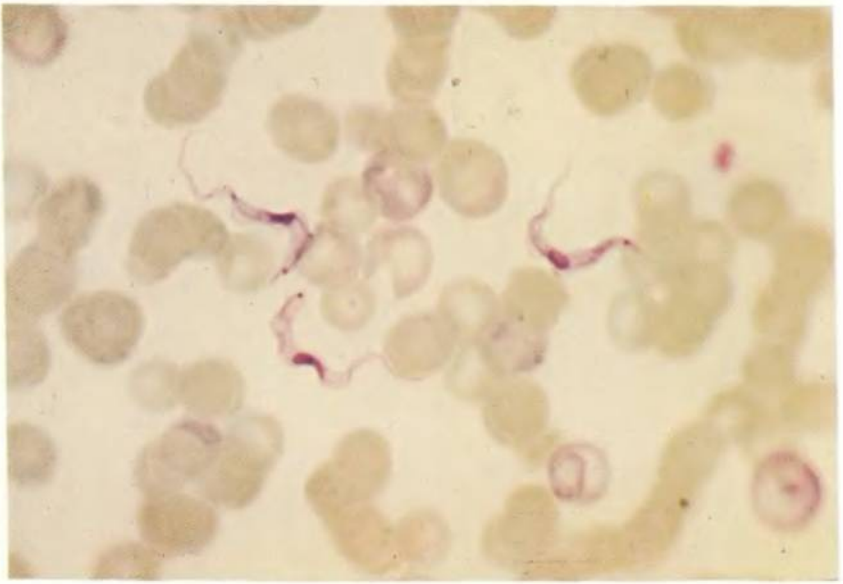


124 & 125 *T. b. rhodesiense* in rat blood *T. b. gambiense*, *T. b. rhodesiense* (and *T. b. brucei* of animals) are virtually indistinguishable in blood films. They are polymorphic; ie long, thin, intermediate and short stumpy forms of trypomastigotes may coincide. Note the small kinetoplast and free flagellum. Both subspecies from man will infect guinea pigs but only *T. b. rhodesiense* is infective to rats. *(× 1 250)*

*(*See* **Tables IV & V**)

126 Diagram of ultrastructure of *T. b. gambiense* A – free flagellum; B – microtubules of pellicle; C – nucleus; D – mitochondrion; E – kinetoplast; F – cytoplasmic granule; G – reservoir; H – basal body of flagellum; I – Golgi apparatus; J – endoplasmic reticulum; K – ribosomes; L – fold of pellicle; M – attached flagellum; N – undulating membrane. *(× 30 000)*

124



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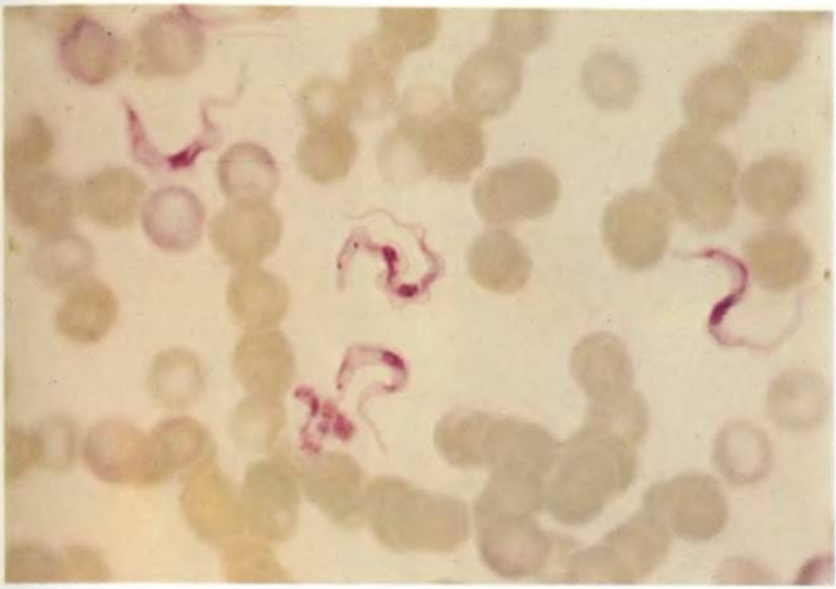

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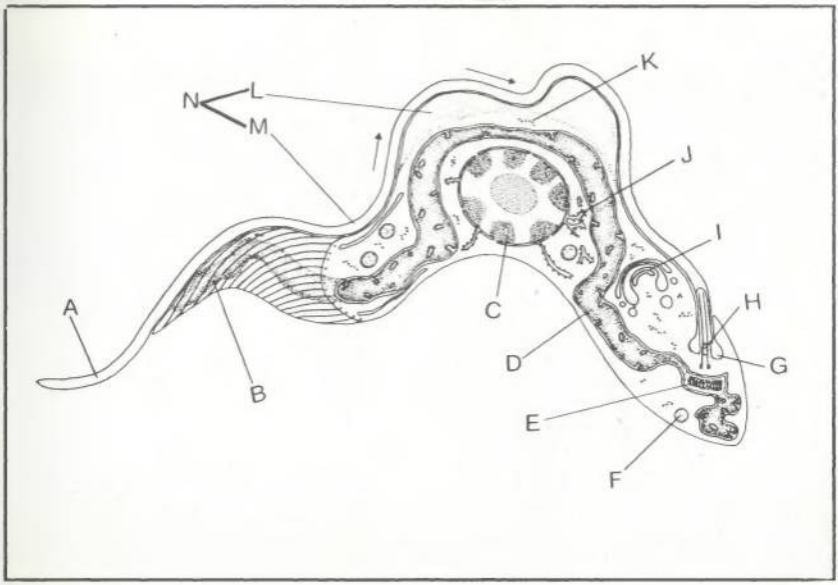


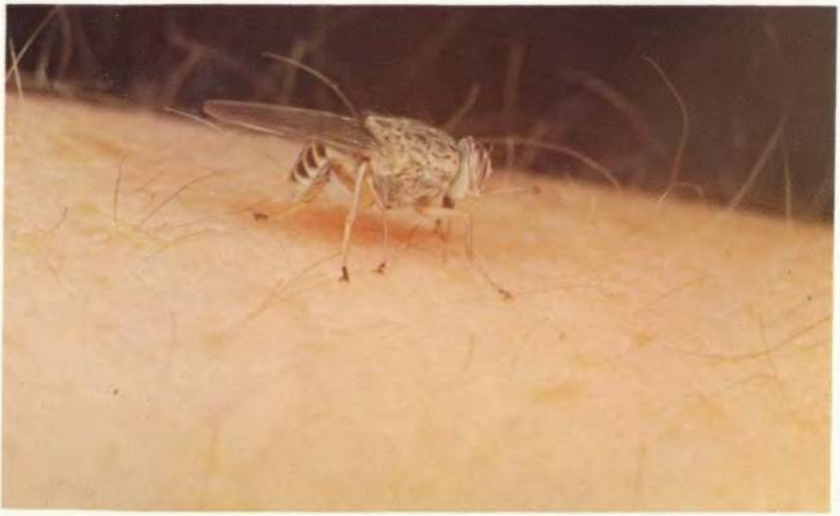
127 Tsetse fly feeding The common vectors of *T. b. gambiense* are *Glossina palpalis* and *G. tachinoides* in West Africa. *T. b. rhodesiense* is associated with *G. morsitans*, *G. swynnertoni* and *G. pallidipes*. Other, secondary vectors have more localised distributions. *(× 4)*

128 Trypanosomes in section of tsetse fly After ingestion by the tsetse fly, the trypomastigotes pass to the midgut. After asexual reproduction the parasites migrate forward between the peritrophic membrane and gut wall to re-enter the pharynx and proboscis. They migrate back into the salivary glands when they transform first into epimastigotes (*see* **148 & 149**), then to the infective stage (metacyclic trypomastigotes). The section shows trypomastigotes massed at the entrance to the midgut ready to enter the proventriculus. *(× 90) (see also* **765***)*

129 Metacyclic trypanosomes in salivary 'probe' The infective stages are passed into the bite together with the saliva when the fly next feeds. They may be observed in saliva expressed from the proboscis of the fly onto a microscope slide. *(× 900)*

130 Larva, pre-pupa and pupa of *Glossina morsitans* A single larva develops inside the female tsetse fly and is deposited when mature in dry soil. Here it pupates and metamorphoses to the adult. *(× 4)*

127



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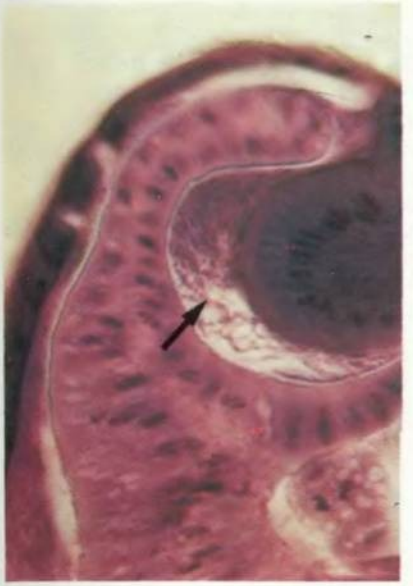

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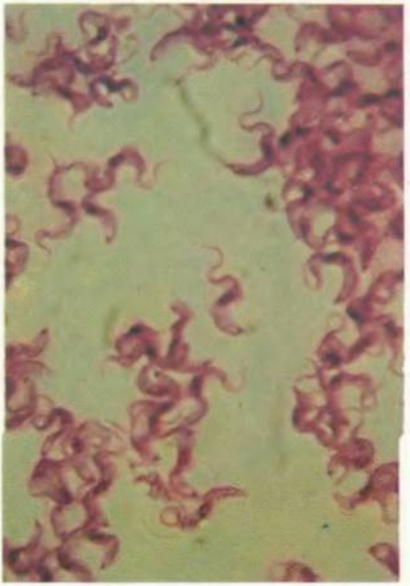

130

131 Ecology of gambiense infection Gambiense trypanosomiasis is transmitted by riverine species of *Glossina* requiring optimum shade and humidity. Shady trees near lakes, river and pools of water are ideal habitats. The figure shows a typical site for transmission by *G. palpalis* in the hinterland of Liberia. Man/fly contact is intimate when villagers congregate around pools for collecting water or washing. *Glossina tachinoides* is second in importance to *G. palpalis* as a vector of sleeping sickness. Rhodesiense trypanosomiasis can occur in scrub savannah country because the *Glossina* vectors are less dependent on moisture. Moreover, in such terrain wild animals and domestic cattle provide alternative feeding opportunities for the fly. Trypanosomiasis is a serious disease of domestic animals, causing great economic loss and depriving human populations of much needed protein. *T. vivax* and *T. congolense* are the commonest parasites involved. Pigs may also be decimated by other species.

132 Bushbuck reservoirs of *T. rhodesiense* A reservoir of *T. rhodesiense* was long suspected in wild animals. The first species found infected with this trypanosome was the bushbuck (*Tragelaphus scriptus*), but other species of game animals have since been found to harbour parasites.

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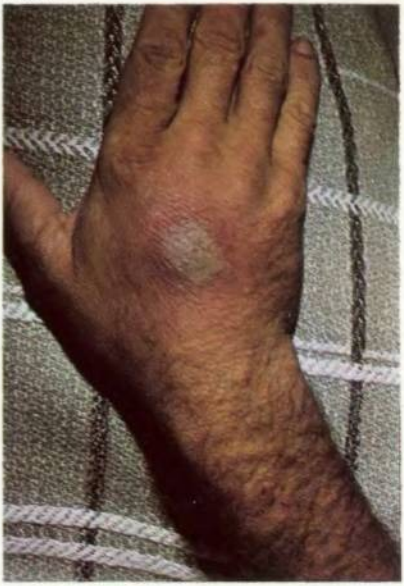
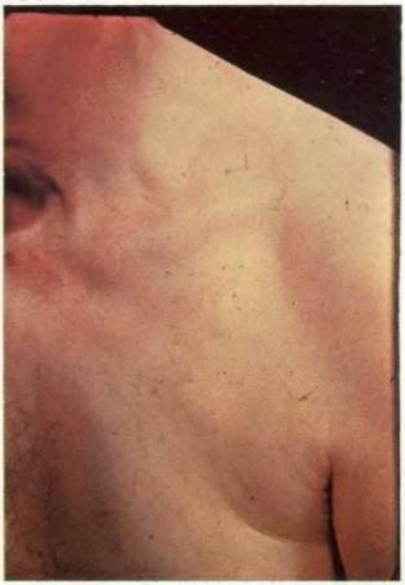
133



133 Trypanosomal chancre The bite reaction, the earliest clinical lesion, is known as a 'trypanosomal chancre'. It resembles a boil but is usually painless. Fluid aspirated from the nodule contains actively dividing trypanosomes.

134 Trypanosomal rash In fair-skinned individuals each peak of fever may be accompanied by a remarkable skin eruption in the form of annular patches of erythema.

134



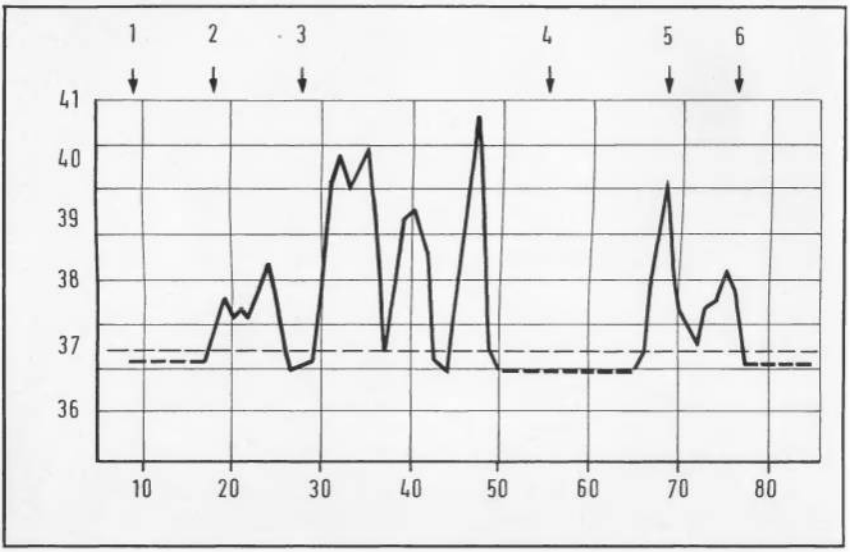
135 Temperature chart in a patient with trypanosomiasis Irregularly occurring episodes of pyrexia are often associated with the rash. Trypanosomes appear in the blood one to three weeks after infection. They may be scanty in gambiense but are commonly numerous in rhodesiense infection which is usually a more fulminating disease. 1 – headache; 2 – trypanosomal chancre; 3 – oedema of l. eyelid; 4 – rash; 5 – rash.

136 Cervical lymphadenopathy – keloid scars following attempted excision Enlargement of lymphatic glands, especially in the posterior triangle of the neck ('Winterbottom's sign') is an important clinical feature of *T. gambiense* infection, and calls for diagnostic puncture of the gland.

137 Gland puncture Gland puncture can provide early diagnosis of trypanosomiasis.

138 Trypanosomes in gland fluid The trypanosomes are easily identified as actively motile organisms in the wet preparation of aspirated gland juice. Their identity can be confirmed by staining. *(× 1 250)*

135



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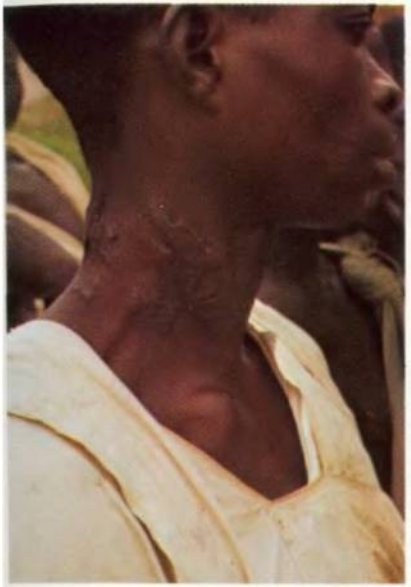

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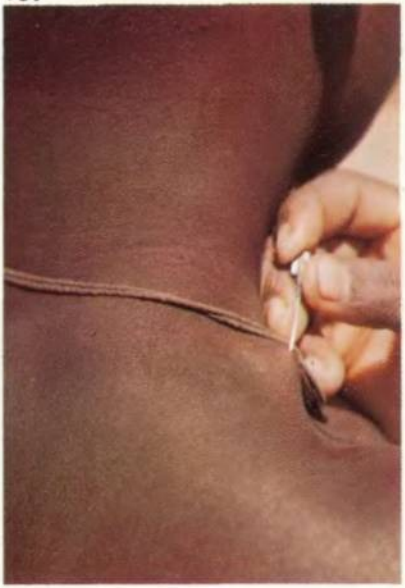

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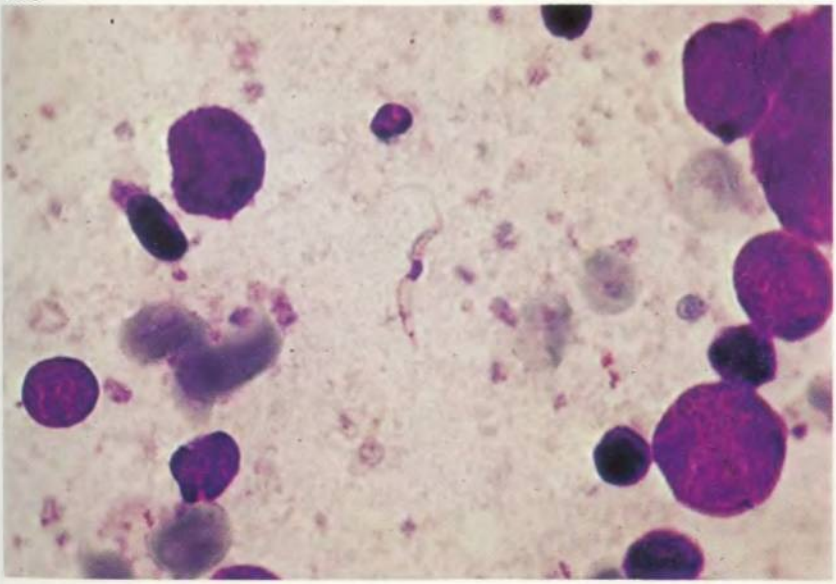

139 Sleeping sickness In the absence of treatment, the patient with gambiense infection becomes progressively more wasted and comatose, finally showing the classical picture of sleeping sickness as the CNS becomes involved. Rhodesiense infection often leads to death from toxic manifestations before CNS changes are evident. This Ghanaian child was diagnosed as having sleeping sickness during a smallpox control campaign; she was very somnolent but not cachetic.

140 Lumbar puncture This procedure should be carried out to determine whether the CNS has been invaded. In such instances, the CSF will reveal a lymphocytic pleocytosis, an increased protein content and trypanosomes may be found in stained films of the centrifuge deposit. In *T. rhodesiense* infection invasion of the CNS may occur within four to eight months, whereas several years usually elapse before meningoencephalitis develops in gambiense disease.

141 Quantitative IgM radial diffusion assay IgM values are raised both in the blood and the CSF in trypanosomiasis. This is an important diagnostic finding both for individuals and for field surveys. Other valuable serological aids are the CFT and FAT. (From left: well 1–55mg%; well 2–110mg%; well 3–220mg%; well – 4 patient's serum 170mg%)

139

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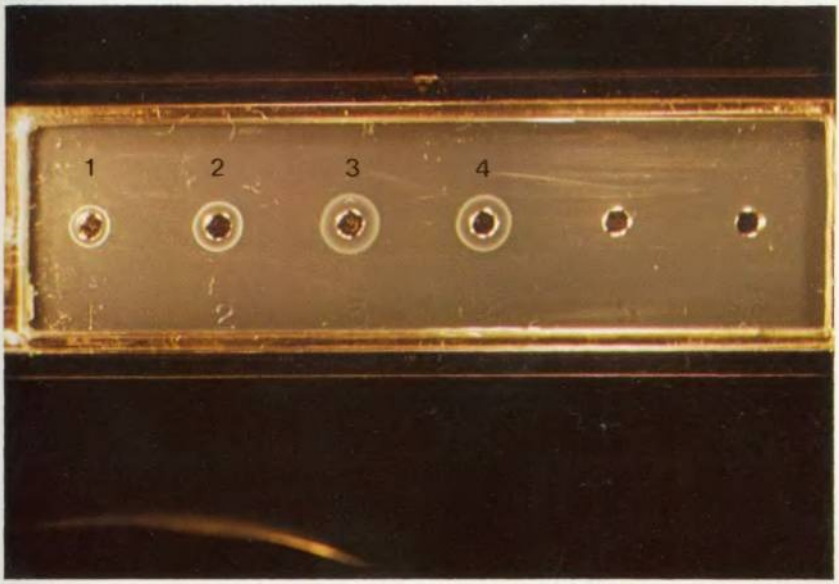
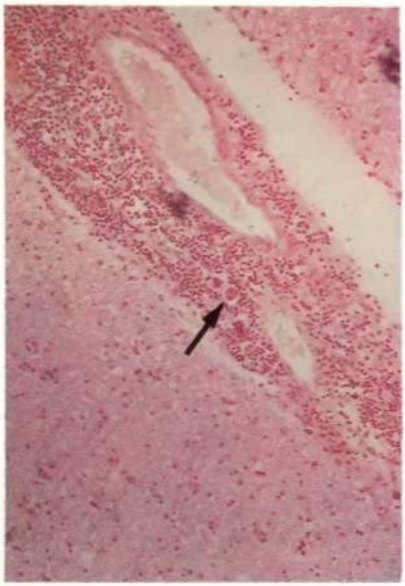


142 Microscopic changes in brain The leptomeninges are congested, there may be oedema and small haemorrhages are commonly present. The basic pathological change is a meningoencephalitis in which perivascular cuffing with round cells is often pronounced. Scattered irregularly through the brain substance there occur large eosinophilic mononuclear cells with eccentric nuclei known as morula cells of Mott. *(× 90)*

143 Morula cell The morula cell of Mott is an IgM-producing plasma cell as shown in this preparation treated with anti-IgM antibody. *(× 900)*

142



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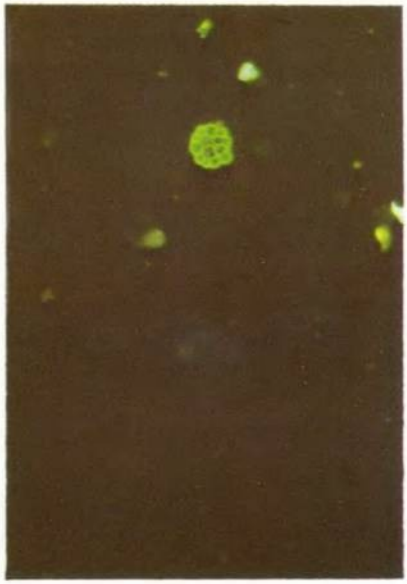
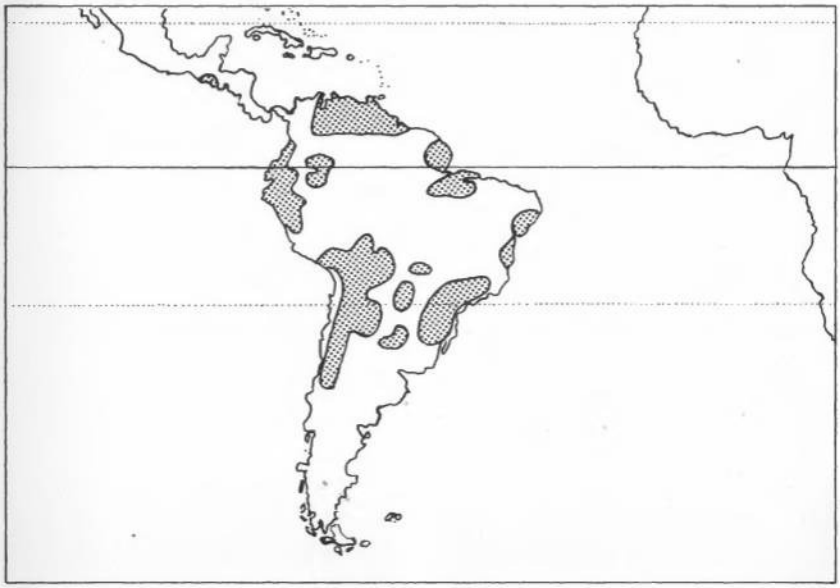


Chagas' Disease

144 Distribution Chagas' disease exists in localised endemic zones in Central and South America from the Andes to the Atlantic coast as far south as the latitude of the River Plate.

144

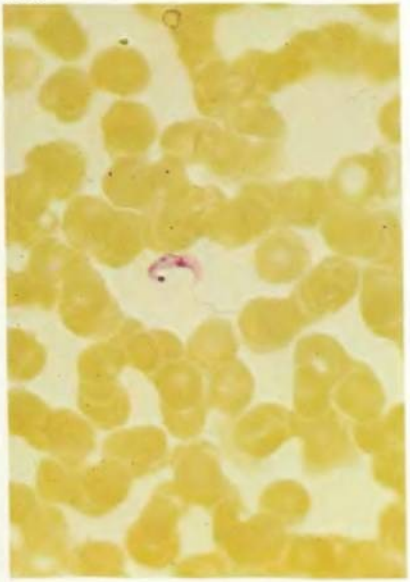


145 & 146 *Trypanosoma cruzi* in human blood film The causative agent is *T. cruzi*. It occurs characteristically in blood films as short 'C' or 'S' shaped trypomastigotes with a prominent kinetoplast. It is otherwise monomorphic. *(× 900)*

147 Typical vector biting Reduviid bugs (also known as 'assassin' or 'kissing' bugs), particularly in the genera *Triatoma, Rhodnius* and *Panstrongylus*, transmit *T. cruzi* while feeding, not by inoculation but by faecal contamination. *(× 1½)*

148 & 149 Life cycle in vector Trypomastigotes picked up in the blood meal transform to epimastigotes in the midgut of the bug. They reproduce as epimastigotes, then pass to the hindgut where they transform again to the infective stages (metacyclic trypomastigotes). The figures show epimastigote stages in the vector. *(× 900) (see also* **766***)*

145



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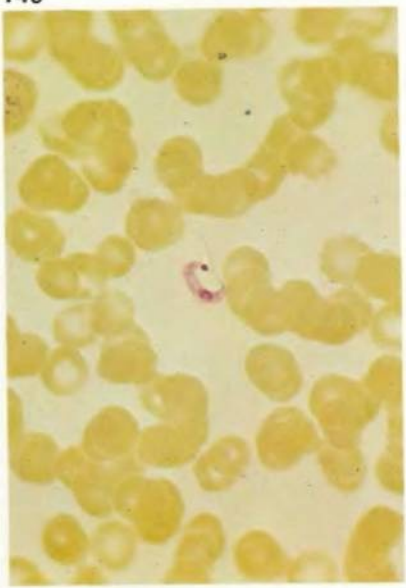

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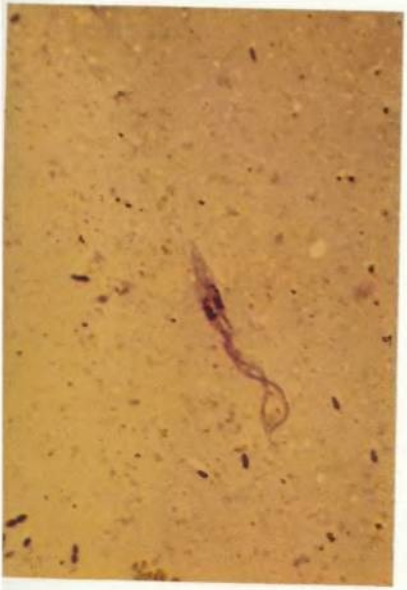

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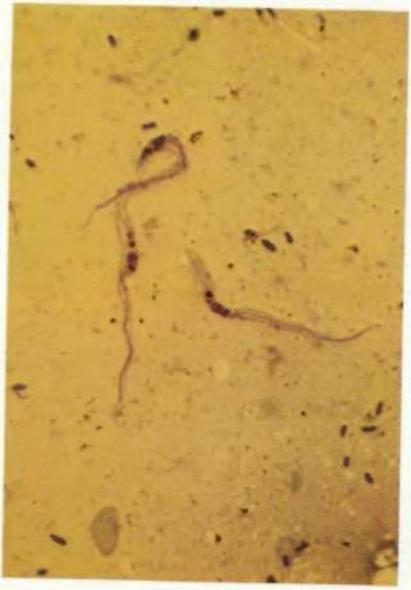

150 Metacyclic stage in faeces Infection is through contamination by parasites in bug faeces produced on the skin. These may invade the site of the bite or adjacent mucosa (eg the conjunctiva). *(× 900)*

151 Ecology of vectors The favourite habitats of the reduviid bugs are cracks in the walls of mud huts in poor rural areas. Here the insects shelter and breed. Transmission occurs predominantly at night.

152 Armadillo, reservoir host Chagas' disease is a zoonosis. It has an extensive mammalian reservoir both in wild hosts (especially armadillos and opossums), as well as domestic animals.

153 Romaña's sign The infection often begins with a local lesion, the chagoma. It causes marked local oedema which, should it occur in the region of the eye or within the conjunctival sac, is accompanied by swelling of the lids and chemosis. These unilateral periorbital changes constitute Romaña's sign.

154 Amastigotes in heart muscle After a stage of initial parasitaemia associated with fever (often unrecognised), trypomastigotes pass to the cardiac muscle and smooth muscle lining the intestinal tract. Here they transform to the amastigote stage (Leishman-Donovan bodies) in which they multiply to form pseudocysts. In the heart this is associated with severe myocarditis especially in the early stages of the infection. *(× 350)*

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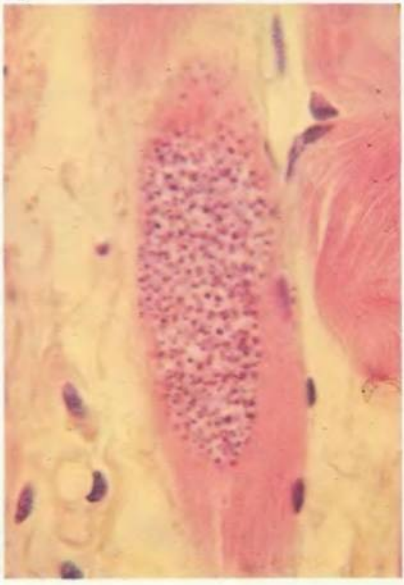

155 *T. cruzi* dividing amastigotes in tissue culture The basic structure of the amastigote is similar to that of the trypomastigote (**145 & 146**) but the flagellum is very short and the mitochondrion is poorly developed. *(× 1 250)*

156 ECG showing heart block Dysrhythmias of various types and degrees are a characteristic feature of Chagas' disease. Complete heart block with Stokes-Adams attacks can occur and may result in sudden death.

157 Cardiomegaly The heart shows gross enlargement and dilatation. The dilatation of the right atrium and both ventricles is marked in this specimen. The pathogenesis seems to be associated with a loss of autonomic control due to destruction of the ganglionic plexuses.

158 Apical aneurysm of heart Mural thrombi may be present at the apex of the left ventricle, with marked thinning of both ventricular walls. Apical aneurysmal formation is commonly seen.

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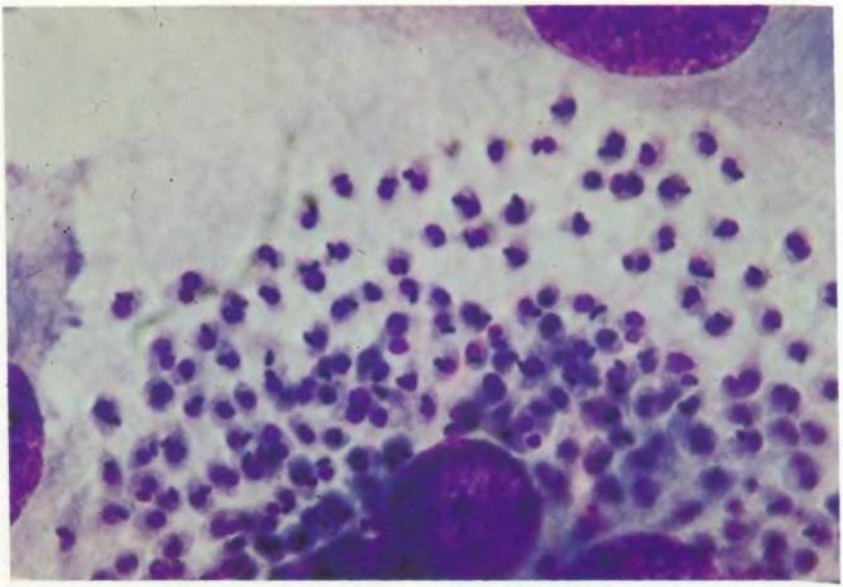

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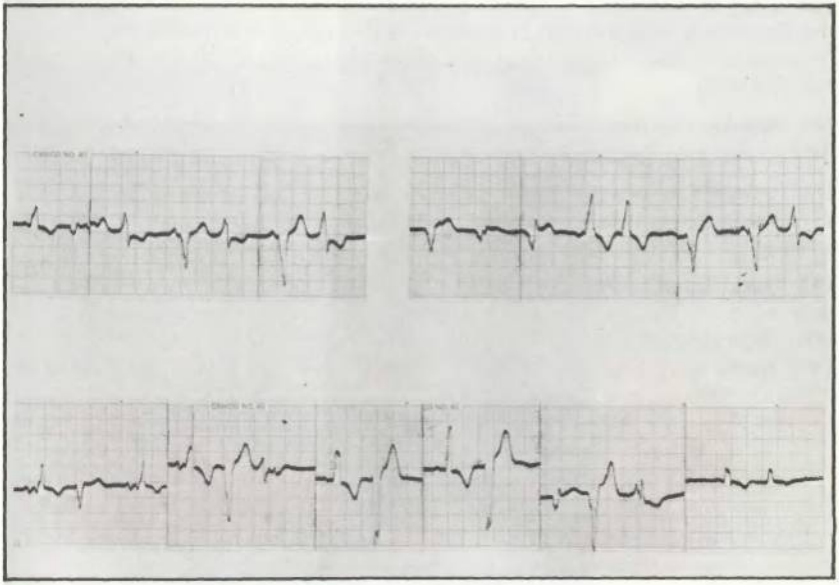

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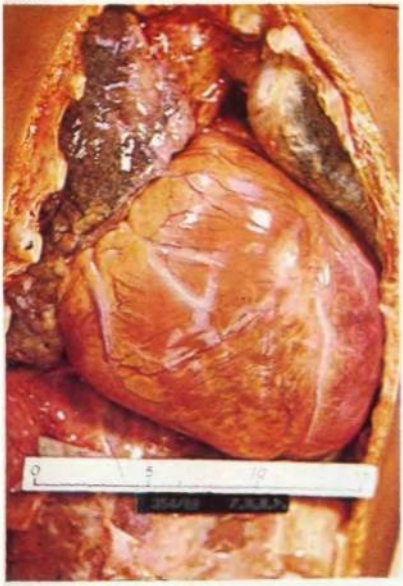

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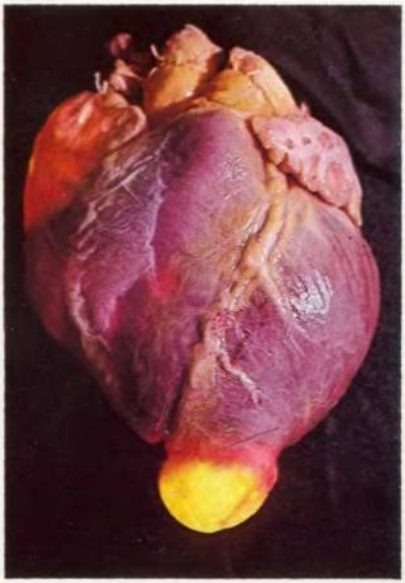


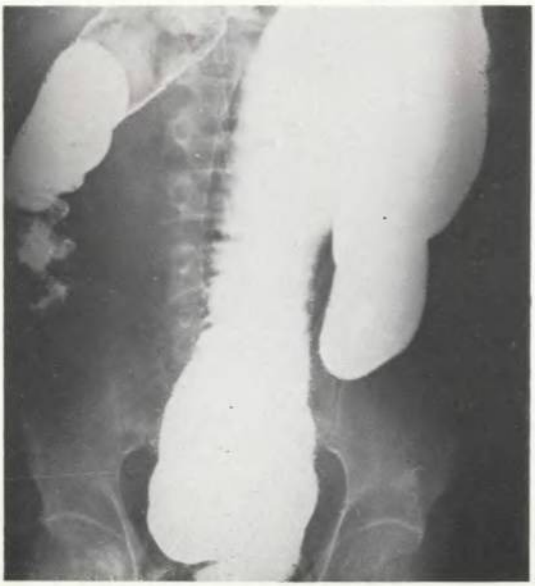
159 X-ray of megacolon Muscular degeneration and denervation of segments of the alimentary tract through destruction of the cells of Auerbach's plexus cause megaoesophagus, megastomach and megacolon, etc, which can be detected radiologically.

160 Post-mortem megacolon Gross megacolon is shown here in a woman who died of chronic Chagas' disease.

161 Parasites in oesophageal muscle Pseudocysts containing amastigotes of *T. cruzi* can rarely be demonstrated in ganglion cells of the intestinal tract, although the smooth muscle is often invaded. *(× 350)*

162 Xenodiagnosis Absolute confirmation of active infection is obtained by demonstrating that the patient can infect the vector (xenodiagnosis). Laboratory-bred clean reduviid bugs are fed on patients suspected of having trypanosomiasis. Two weeks later the hindgut is dissected out and is examined for metacyclic trypanosomes.

159



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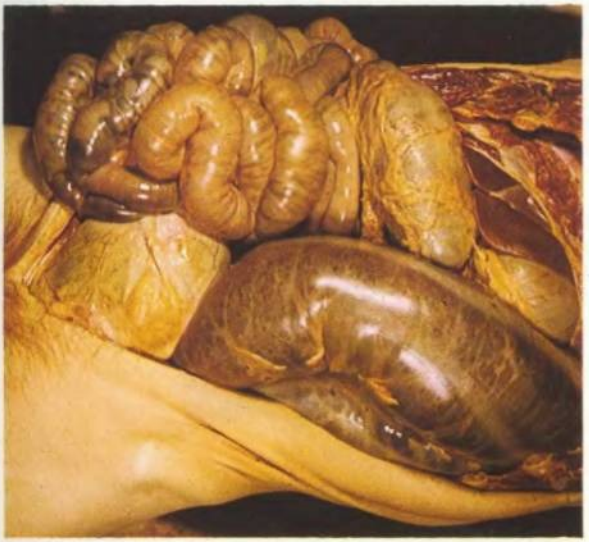

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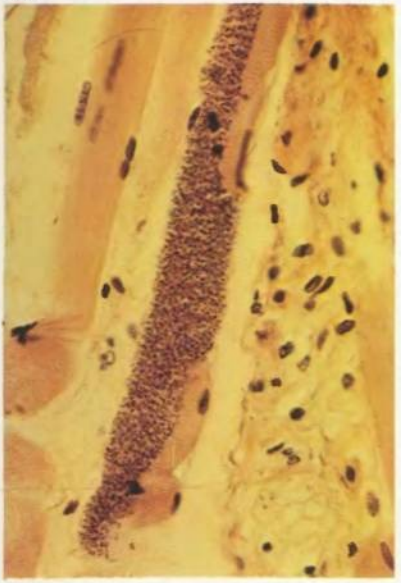


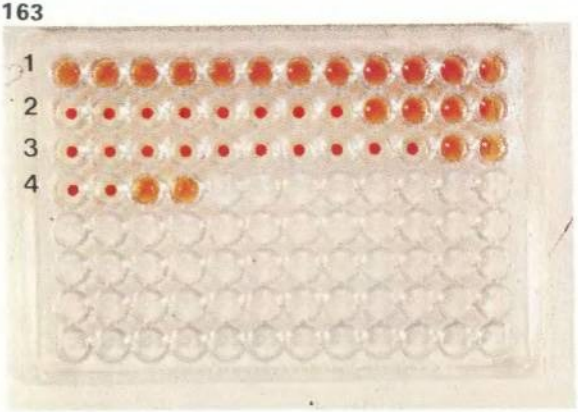
162

163 Machado-Guerreiro reaction A complement fixation test employing antigen from epimastigotes of *T. cruzi* cultivated *in vitro* is widely used, and is one of the most sensitive means of diagnosis. It does not necessarily imply the presence of active infection. (Top row – negative control; second row – positive control titre 1/256; third row – patient's serum titre 1/512; fourth row – complement control.)

164 Immunofluorescence of *T. cruzi* Fluorescent antibody tests may also be employed using whole cultured epimastigotes as the antigen. *(× 600)* (*Right:* negative control)

165 *Trypanosoma rangeli* *T. rangeli* is a long slender trypanosome also transmitted by reduviid bugs from wild animals to man. It is readily distinguished by its shape from *T. cruzi* in blood films and appears to be non-pathogenic to man. *(× 600)*

163



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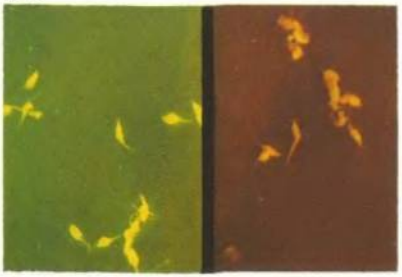

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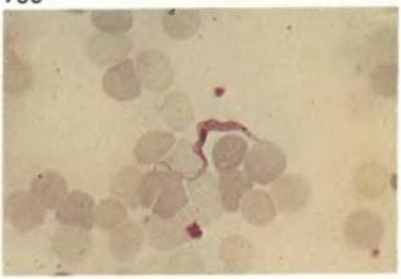
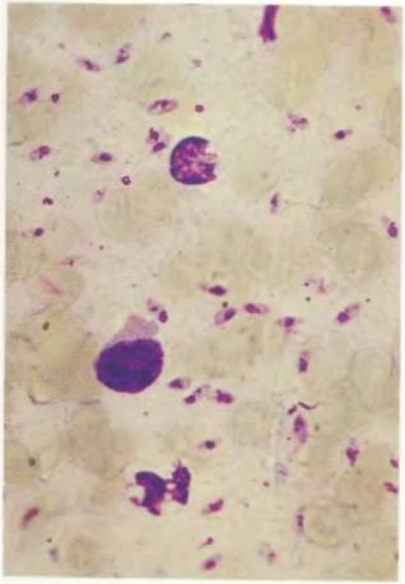


LEISHMANIASIS*

166 Life cycle of *Leishmania* in mammalian host Amastigotes (Leishman-Donovan bodies) enter phagocytes of the lymphoid-macrophage system. They reproduced by binary fission forming a mass of daughter cells in each host cell. Note the nucleus and minute flagellum in the parasites shown in this smear from a cutaneous lesion. *(× 900) (see also* **767***)*

167 Parasites in tissue culture In tissue culture (dog sarcoma) *L. mexicana* can be seen in vacuoles in the host cells. *(× 900)*

166



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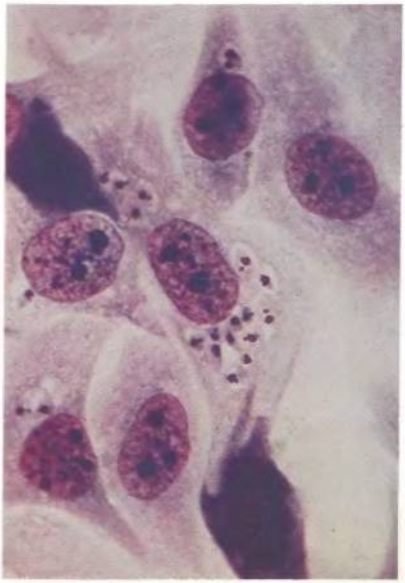


168 Ultrastructure of amastigotes Leishmanial amastigotes are seen within a parasitophorous vacuole in the host cell. *(× 30000)*

169 Sandfly larva *Leishmania* are transmitted by sandflies of the genus *Phlebotomus* in the Old World and Far East, and *Lutzomyia* in the New World. The photograph shows the larva of *P. perfiliewi* which is a vector of leishmaniasis in Southern Europe. In dry areas the larvae occupy cracks and crevices which provide a humid, cool, microclimate. Forest species possibly prefer leaf mould on the forest floor. *(× 10)*

*(*See* **Table VII**)

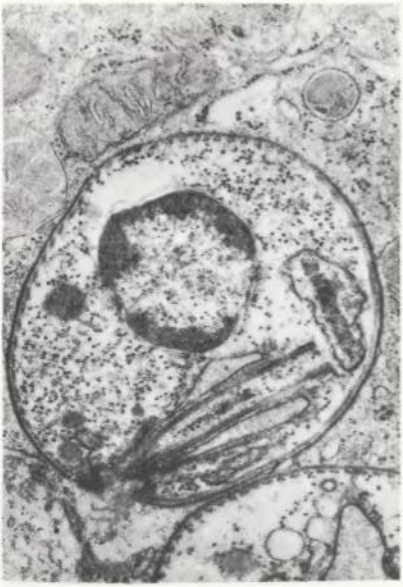
170 Pupa of *L. longipalpis* *Lutzomyia longipalpis* transmits visceral leishmaniasis in Brazil. *(× 10)*

171 Adult female *L. longipalpis* biting *(Natural size)*

172 Closeup view of *L. longipalpis* *(× 10)*

173 Promastigotes in vector In the midgut of the poikilothermic vector amastigotes transform to promastigotes which then divide asexually. The new promastigotes become attached to cuticle-lined parts of the gut such as the oesophageal valve indicated by the arrows. From here promastigotes pass into the pharynx and proboscis through which they are injected into a new host when the fly next feeds. They then transform back to amastigotes in the warm-blooded vertebrate host. *(× 1250)*

168



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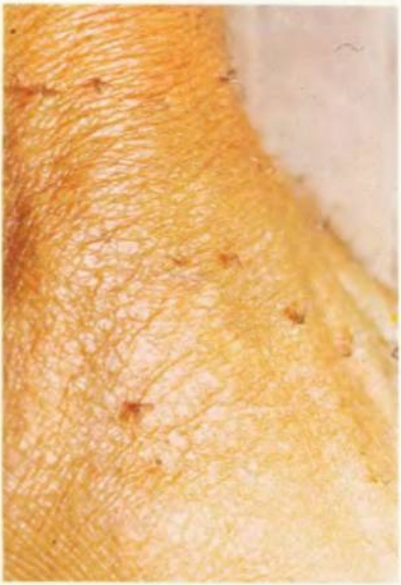

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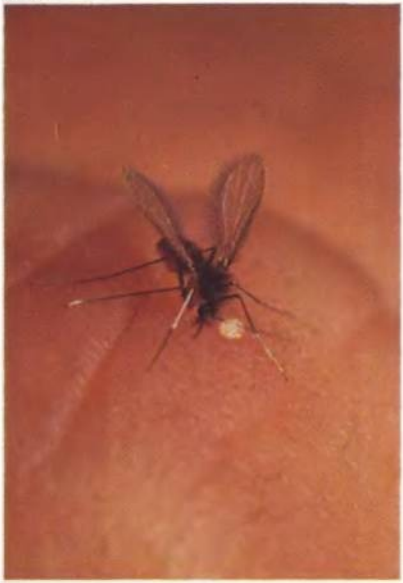

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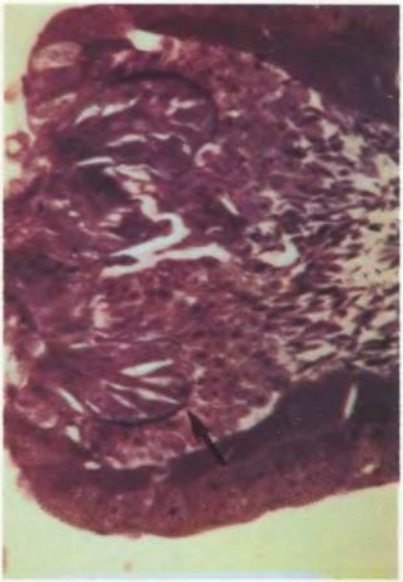

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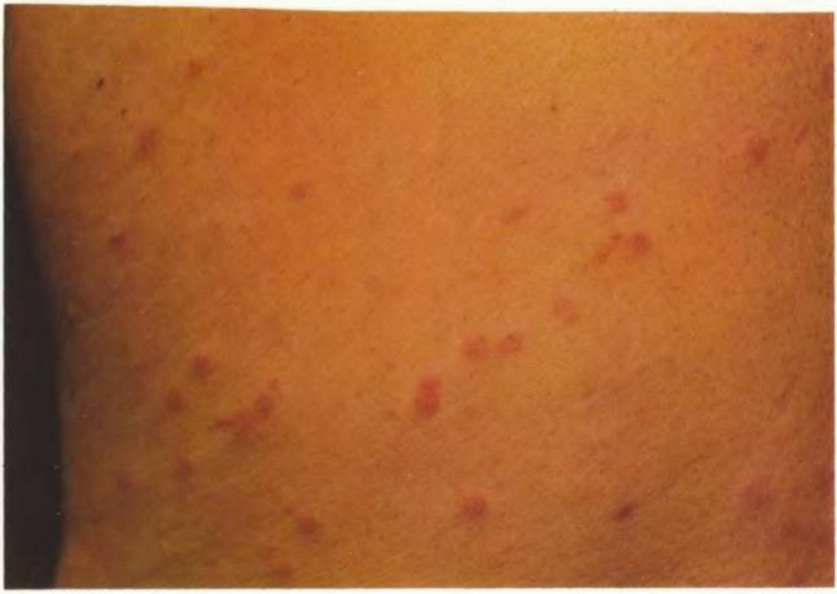


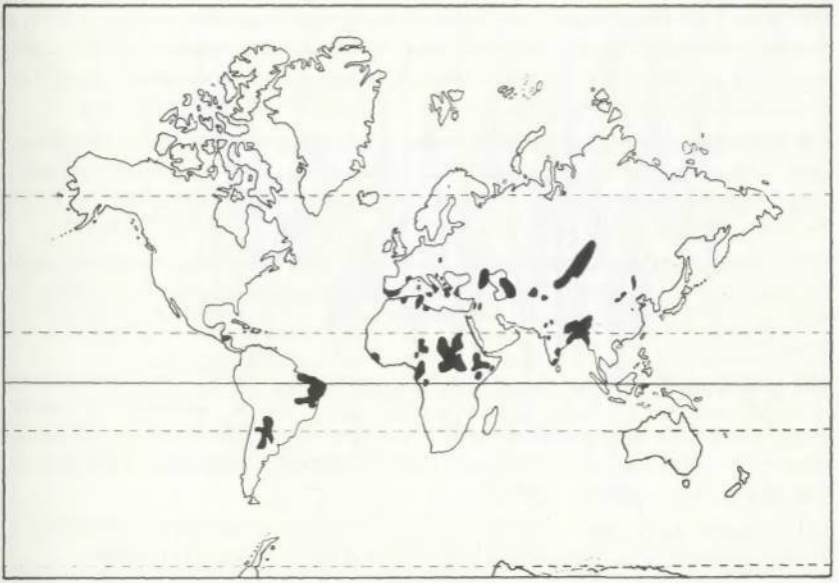
174 Reaction to sandfly bites A persistent macule appears at the site of each bite even from an uninfected fly. This may be the starting point of the lesion in simple cutaneous leishmaniasis.

Visceral Leishmaniasis (Kala-azar, Dum-Dum Fever, Black Sickness)

175 Distribution Visceral leishmaniasis caused by *L. donovani* or *L. infantum* occurs in the Mediterranean littorals, the Middle East and adjacent parts of the USSR, the Sudan, East Africa, the Indian subcontinent and China, and South America ('*L. chagasi*'). An arid warm environment provides ideal ecological conditions for the breeding of many species of sandflies. Kala-azar is commonly associated with dry, rocky hill country.

176 Termite hill association In East Africa kala-azar is associated with dwellings situated near large termite hills. The vectors become infected by biting rodents which live in the holes in the termite hills, and later transmit the disease to people living in the vicinity.

175



176

177 Reservoirs of kala-azar The photograph shows blood being collected from a dog for serological detection of kala-azar. Man is the only reservoir in India but elsewhere kala-azar is a zoonosis. Dogs, wild carnivores, and various species of rodents are commonly infected in rather focal fashion (*see* **Table VII**).

178 Clinical picture of kala-azar in Kenya Increasing enlargement of the spleen and liver is a characteristic feature, while in dark complexioned subjects deepening skin pigmentation is seen – hence the synonym kala-azar, the 'black sickness'. A generalised lymphadenopathy is common in African kala-azar.

179 Temperature chart in kala-azar The temperature chart shows a double peak every 24 hours. Despite the high temperature the patient often looks remarkably well and has a good appetite. A leucopenia with a relative lymphocytosis is often present.

180 Amastigotes of *Leishmania* in blood of experimental rodent The blood picture is highly suggestive, comprising a marked granulopenia, moderate to severe anaemia without any special features and a very high ESR. Thrombocytopenia may lead to haemorrhagic manifestations. Occasionally amastigotes are seen in circulating macrophages. *(× 900)*

181 Infantile kala-azar Children present with irregular fever, anaemia, a moderately enlarged, non-tender liver, and a greatly enlarged firm spleen.

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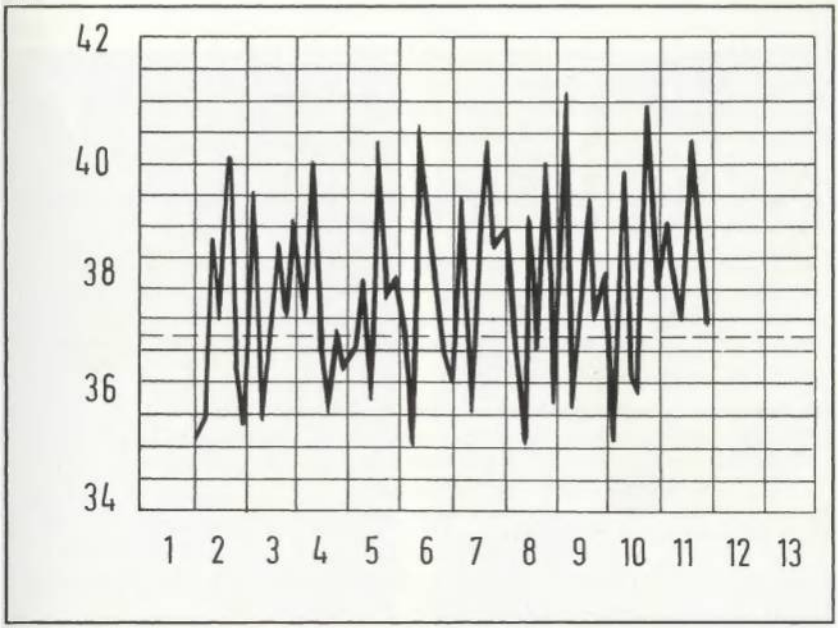

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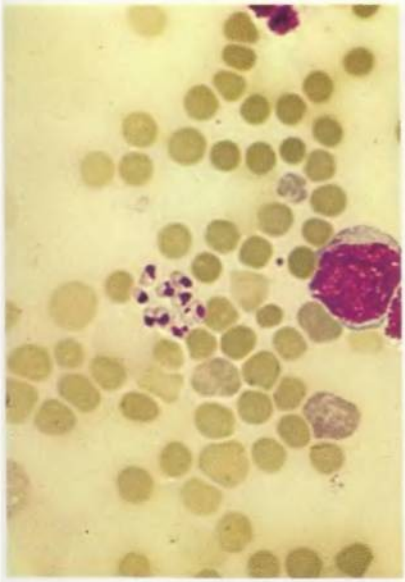

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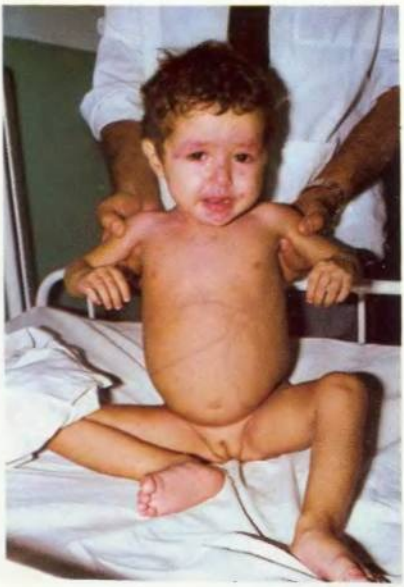


182 Post kala-azar dermal leishmanoid (PKDL) This syndrome is a sequel to visceral leishmaniasis following treatment. Dermal lesions which vary in appearance contain amastigotes in large numbers. Some start as hypopigmented macules. The lesions are highly infectious to sandflies.

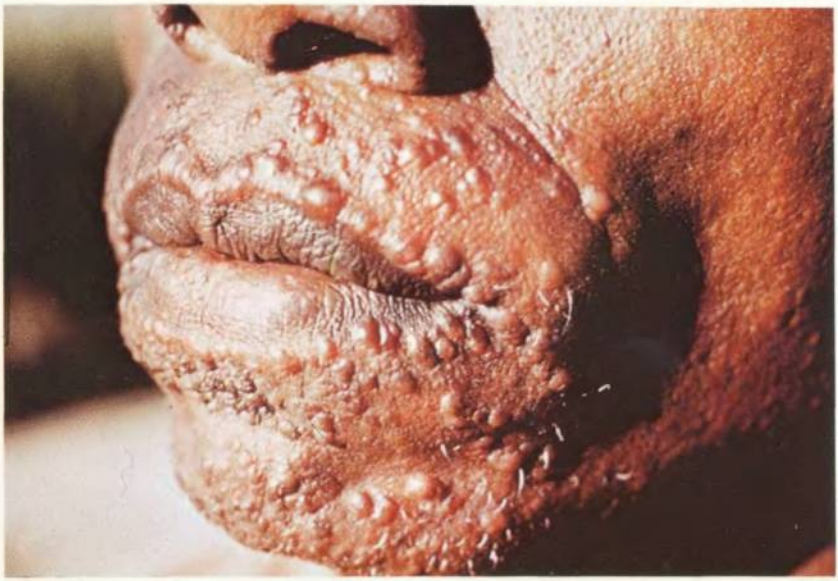
183 PKDL in a Chinese patient The patient was completely cured by chemotherapy. This response differentiates the condition from the anergic 'diffusa' type of leishmaniasis (**210–212**).

184 Smear of bone marrow The prime means of diagnosis is the detection of amastigotes in bone-marrow, spleen or blood. They are recognised in dried smears of material stained by Giemsa's method by their characteristic morphology. While typically found in macrophages, isolated extracellular amastigotes are commonly seen in such preparations. *(× 900)*

185 Diagnosis by animal inoculation The parasites may be isolated by intrasplenic inoculation into hamsters. After four to six weeks characteristic visceral lesions are seen macroscopically, and amastigotes are found in large numbers in smears of the liver and spleen. Picture taken at autopsy.

186 Promastigotes in NNN culture After inoculation of aspirated material into a special blood agar medium (NNN medium, ie Novy-Nicolle-MacNeal) and incubation at 28°C for one to four weeks promastigotes may appear. *L. donovani* is more readily isolated in culture than *L. infantum*. *(× 900)*

182



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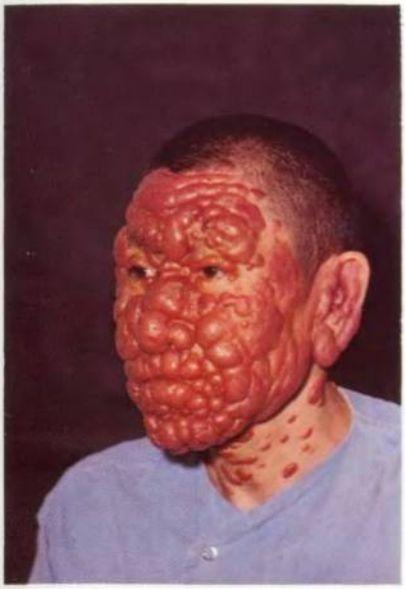

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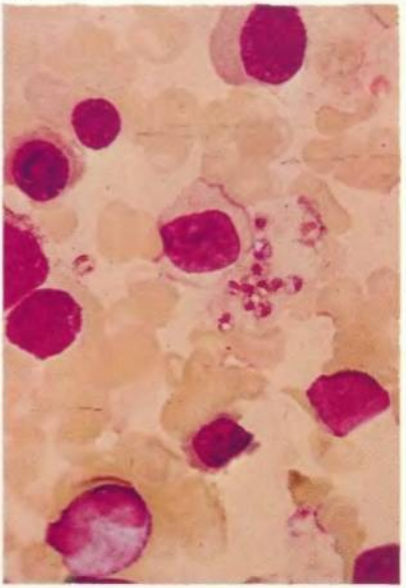

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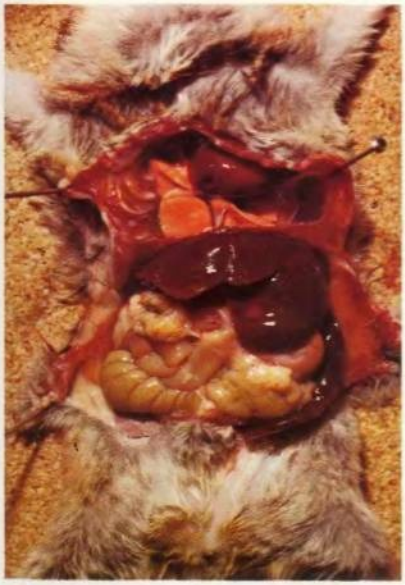

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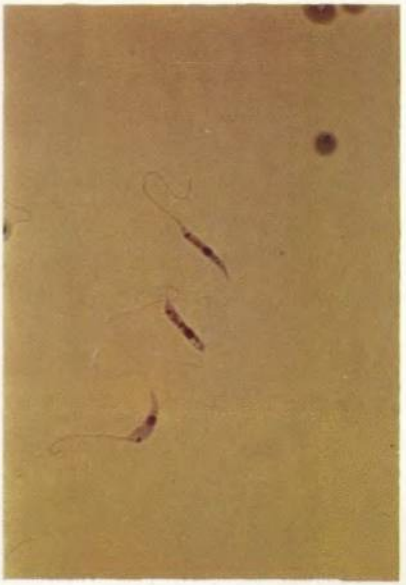
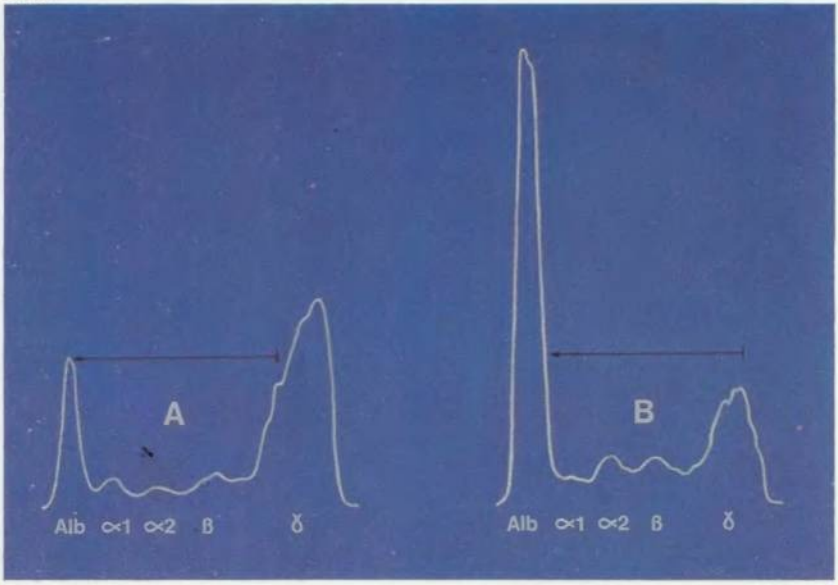


187 Serum proteins before and after treatment of kala-azar Large quantities of IgG are produced by patients with kala-azar and the A/G ratio is reversed. This may be demonstrated by electrophoresis (**187a**), or by simple tests such as the addition to serum of a drop of 30% formalin. The formation of a gel demonstrates the presence of a high proportion of globulin (formol-gel test) (**187b**).

188 Immunofluorescence of *L. donovani* amastigotes Only a small proportion of the increased IgG is specific anti-leishmanial antibody. The IgG can be demonstrated by the fluorescent antibody test using cultured promastigotes of *L. donovani* or tissue smears with amastigotes. The CFT becomes positive later than the FAT. *(× 1250)*

187a



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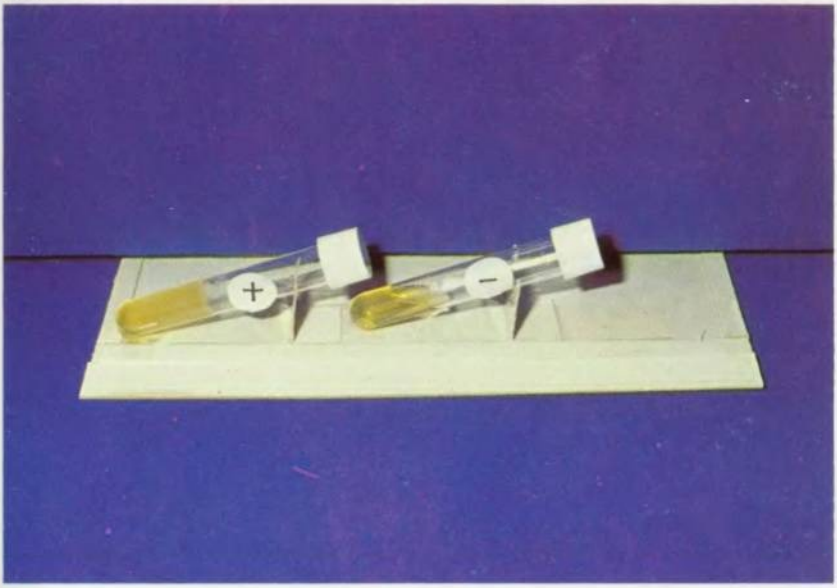

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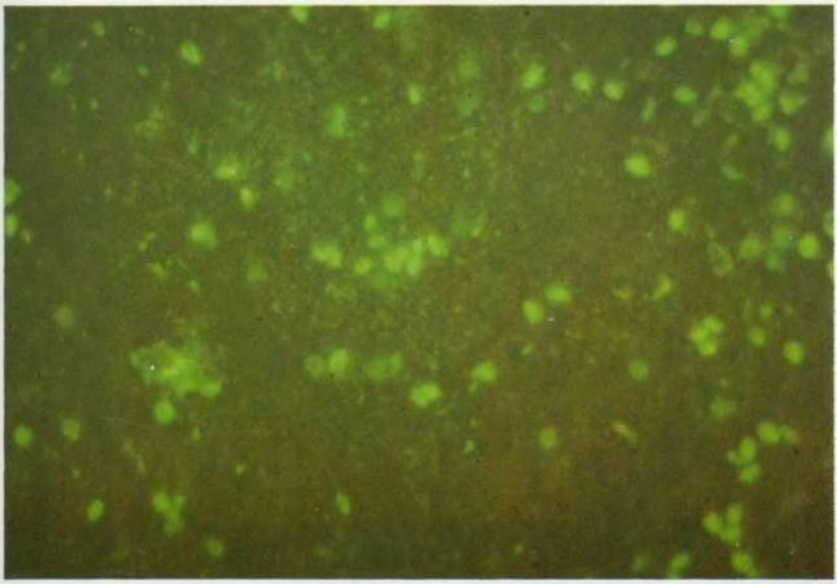


Old World Cutaneous Leishmaniasis

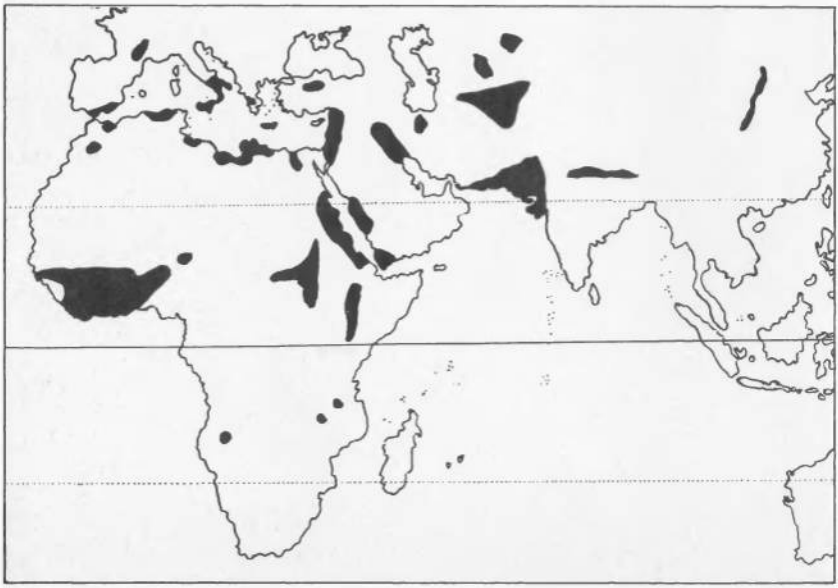
189 Distribution Dermal leishmaniasis is essentially a zoonosis which occurs in scattered foci throughout the tropical and subtropical belts. Depending upon the locality it is known as Oriental sore, Aleppo button, Baghdad boil, Delhi sore, etc. Arid or even semi-desert terrain provides ideal habitats for the vector sandflies which spend the days in cool deep crevices in the ground, between rocks, in caves, cellars, house walls, etc.

190 Hyrax The rock hyraxes shown here are a reservoir in Ethiopia of *L. aethiopica*. Various species of rodents, such as the great gerbil of Eastern Iran and neighbouring parts of the USSR (*Rhombomys opimus*) are important animal resevoirs of *L. (tropica) major*. In the Mediterranean littoral, dogs and wild carnivores may be infected with *L. tropica (minor)*.

191 Simple 'dry' lesion on cheek *L. tropica* produces dry, often self-healing lesions which are usually single. This form is commonly seen in and around towns in the Middle and Near East.

192 Dry lesion on nose of woman in Southern France The cheek is more commonly involved than the nose.

189



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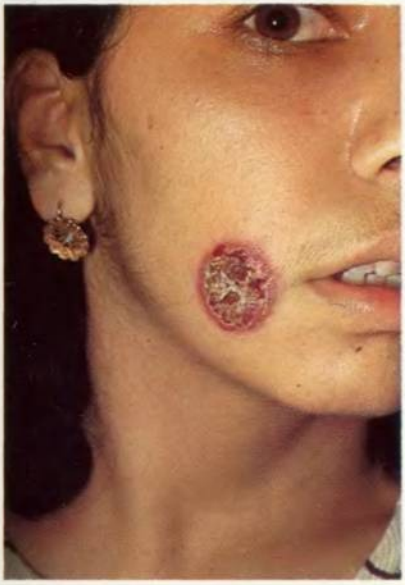

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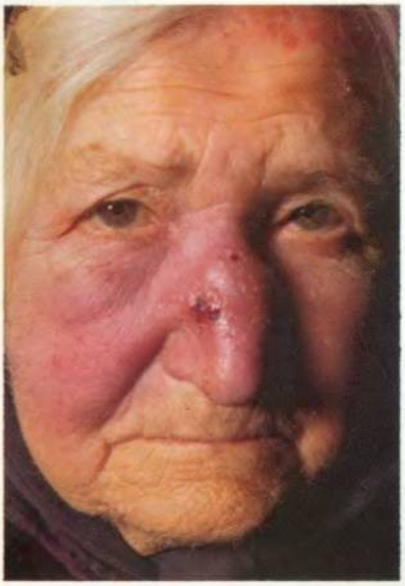

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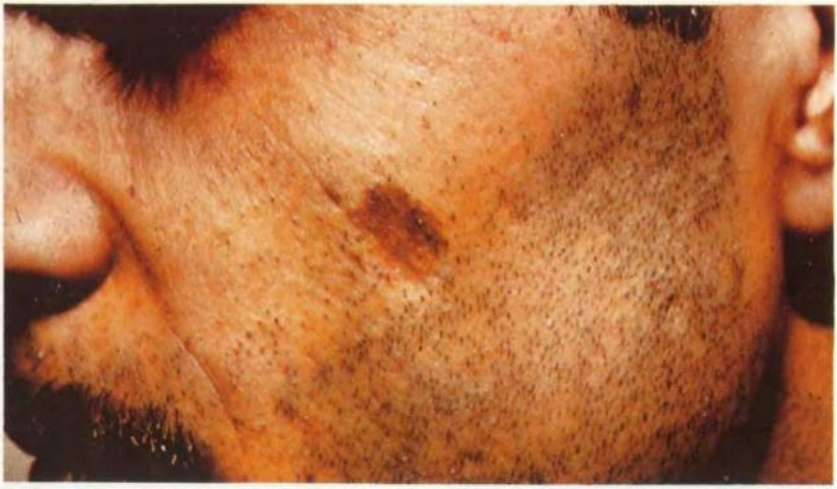

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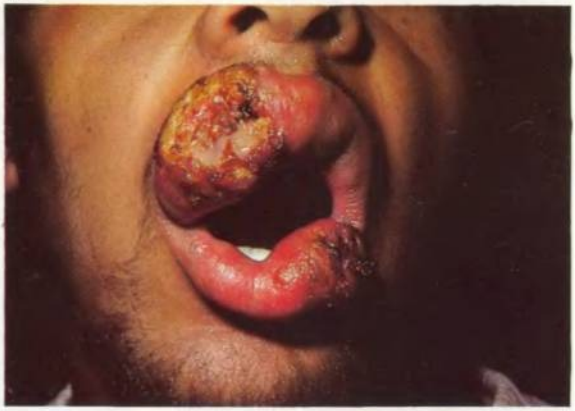


193 Typical healed oriental sore An atrophic, papery, slightly depressed scar results when healing occurs.

194 'Wet' lesion of mouth In rural areas of USSR and Iran, moist, ulcerative lesions may be caused by *L. major*. These may be extensive and sometimes involve the epithelium of lips and nose.

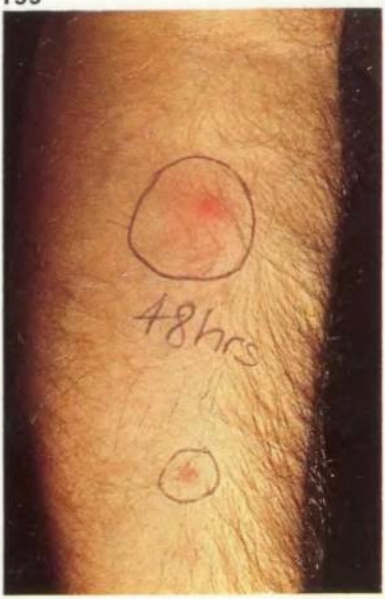
195 Montenegro test Diagnosis may be assisted by the injection of an intradermal antigen prepared from cultured promastigotes of *L. tropica* or other dermatotropic species. This produces a typical cell-mediated response (Montenegro test) in most cases of cutaneous disease. It is negative in the active stages

of kala-azar, but may become faintly positive during the course of treatment especially in East African patients. The reaction in the upper (antigen) site is maximum after 48 hours. The lower site is the control.

New World Cutaneous Leishmaniasis

196 Distribution (including Espundia) New World cutaneous and mucocutaneous leishmaniasis occur focally throughout Central America and South America as far south as São Paulo State of Brazil. The disease is limited by the Andean chain to the West except in Peru where a special form of cutaneous disease 'Uta' is found in the Western slopes of the Andes.

195



196

197 Cutaneous lesions in Panama The cutaneous disease in Panama sometimes shows evidence of lymphatic spread rather similar to the form seen in the Guyanas which is known there as 'Pian bois'. The picture illustrates lymphatic swelling proximal to a cutaneous ulcer. The causative organism is *L. b. panamensis*.

198 Healed simple cutaneous lesions Simple, single ulcers similar to those of Oriental sore caused by organisms of the *L. mexicana* group usually heal readily without complications.

199 Residual infection in 'healed' lesions Occasionally an apparently healed ulcer such as this may be found on biopsy to contain living organisms. Such cases are associated with a constant positive FAT titre. It is suspected that mucocutaneous disease may sometimes arise after many years as a result of metastasis from lesions of this type.

200 Chiclero's ulcer Forest workers collecting gum from wild chicle trees commonly sleep near the forest floor and are bitten on exposed parts of the head by vectors that normally maintain transmission of *L. mexicana* among the forest rodents. Ulcers leading to erosion of the auricular cartilage are known as 'chiclero's ulcers'.

201 Metastatic type of simple sore Occasionally multiple ulcers, each of the typical Oriental sore type, and all of the same age, appear to develop as a result of direct metastasis from an infected macrophage that becomes disrupted while in the peripheral circulation.

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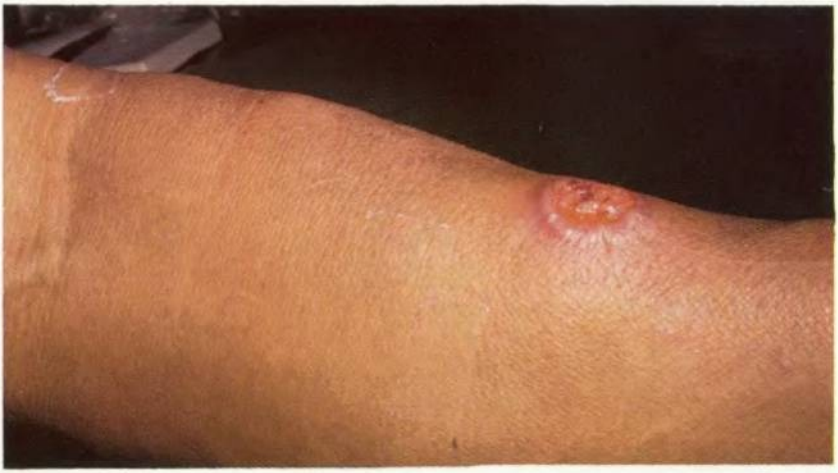

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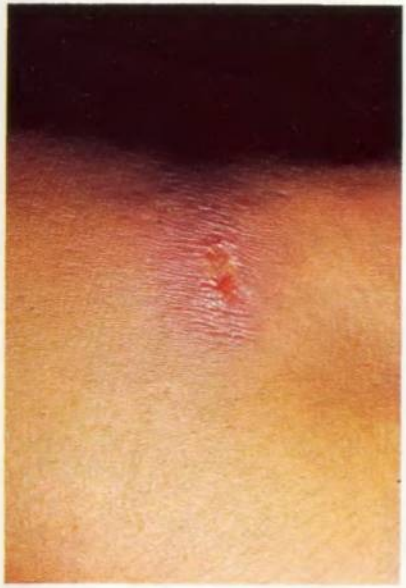

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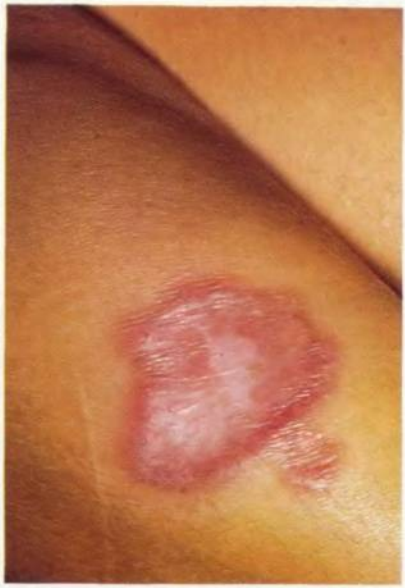

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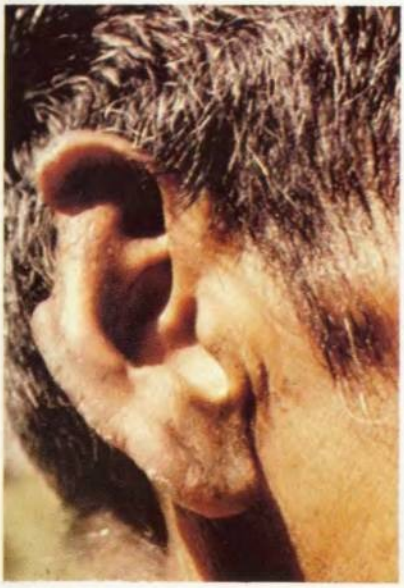

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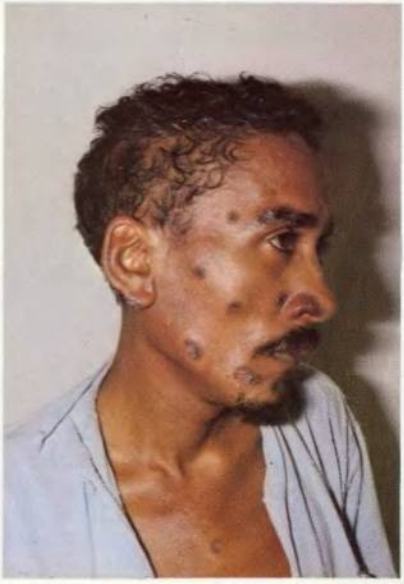


202 **Rodent reservoirs of Brazilian rain forest** Most cutaneous leishmaniasis in the New World is a zoonosis associated with rodents of the rain forest. Rodents such as *Proechimys guyanensis* are typical animal reservoirs for forest leishmaniasis in both Brazil and Central America. *L. mexicana amazonensis* has been isolated from this species. The Brazilian rain forest is the typical habitat of *L. m. amazonensis*.

202

203

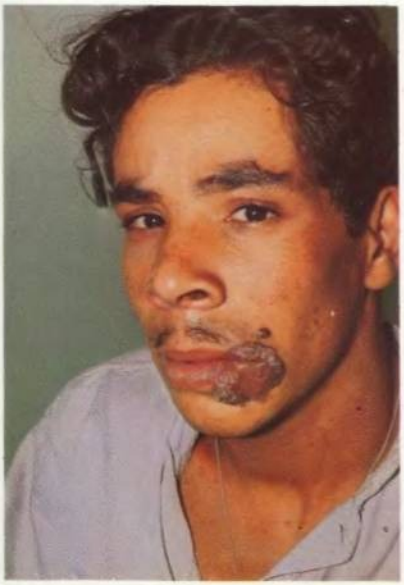
203 Animal reservoirs in Panama In Panama a wide variety of animals serve as reservoirs for *L. b. panamensis*. The hosts include species of monkeys, marmosets, and even such bizarre animals as the three-toed sloth shown here.

Mucocutaneous Leishmaniasis

204 Early lesion of Espundia The lesions of mucocutaneous leishmaniasis are first evident as ulcers involving the mucocutaneous junctions of the mouth and nose.

205 Pharyngeal involvement Ulceration often extends to the pharynx and soft palate, and the first symptoms may be related to tissue destruction in this area. This man had the scar of a large ulcer which apparently healed on his leg some 30 years before.

204



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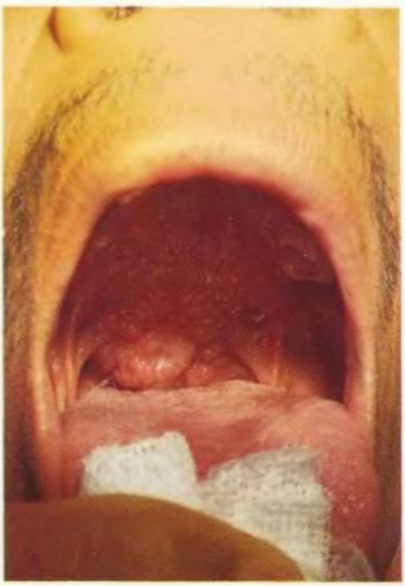
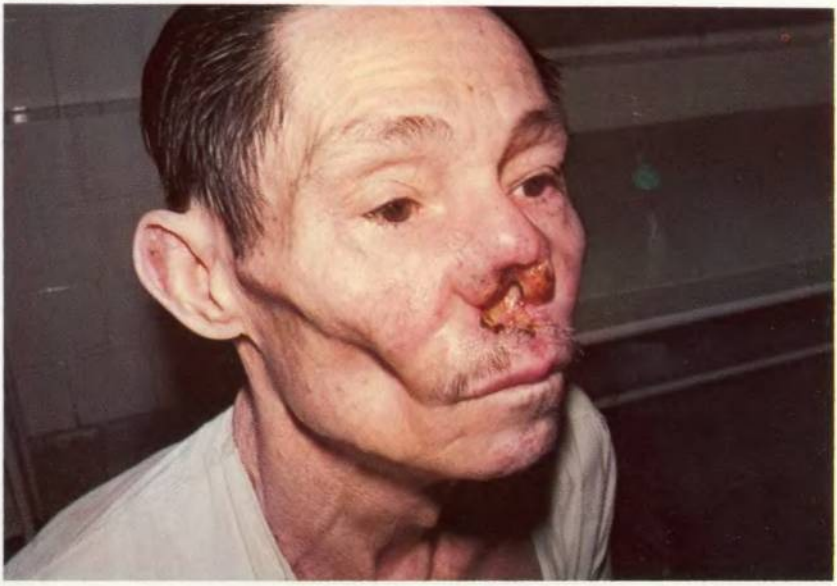


206 Destructive Espundia Gross destruction of the nose, including the septum and palate may follow inadequate treatment. In any case patients with this disease respond very poorly to any form of therapy.

207 'Slow' lesion in hamster three months after inoculation *L. braziliensis*, the causative organism of Espundia, is difficult to demonstrate in the lesions. Inoculation into the nose and feet of hamsters may result in the development of small nodules at the inoculation sites nine months or more later. Culture *in vitro* is very difficult. This is termed a 'slow strain' of parasite.

208 *L. mexicana* in hamster six weeks after inoculation In comparison to *L. braziliensis*, *L. mexicana* and allied organisms rapidly produce large histiocytomas at the sites of inoculation within a few weeks. These parasites also usually grow very readily in NNN medium.

206



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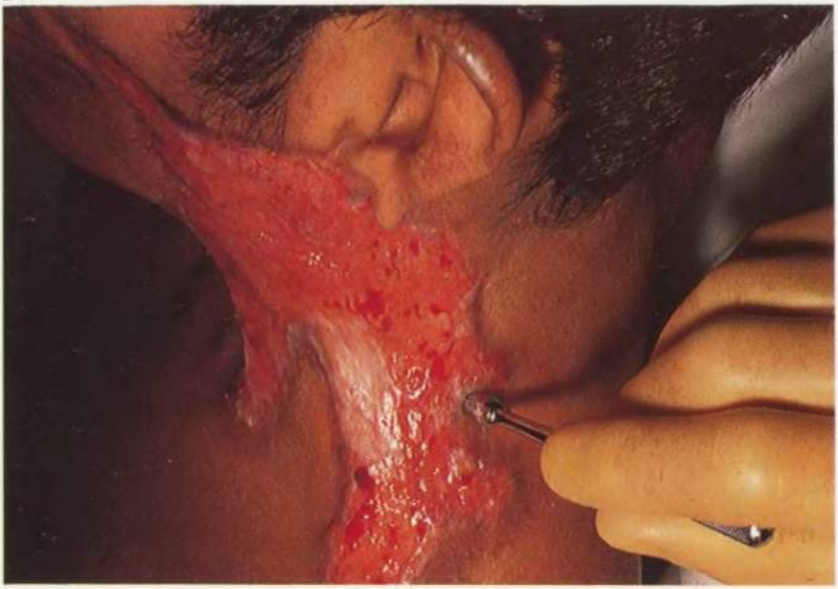


209 Skin biopsy technique In doubtful cases skin biopsy can be taken using a skin punch. The material can then be examined histologically and an attempt made to culture the organisms in hamsters or in NNN medium.

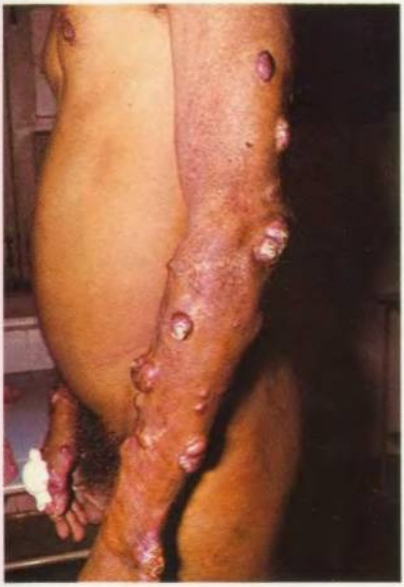
Diffuse Cutaneous Leishmaniasis (DCL)

210 DCL in the New World In rare individuals, a highly specific failure of cell mediated immunity may result in the development of chronic disseminated disease resembling lepromatous leprosy. This patient from Brazil was infected with *L. mexicana amazonensis*.

211 DCL on the foot of an Ethiopian A similar syndrome occurs in other parts of the world such as Ethiopia where this patient was infected with *L. aethiopica*. DCL never affects mucosae, and is thus readily distinguished from Espundia.

212 Skin smear This smear from a skin biopsy from an Ethiopian patient with DCL shows large numbers of amastigotes. Parasites are rarely so numerous in biopsies from patients with Oriental sore. *(× 500)*

210



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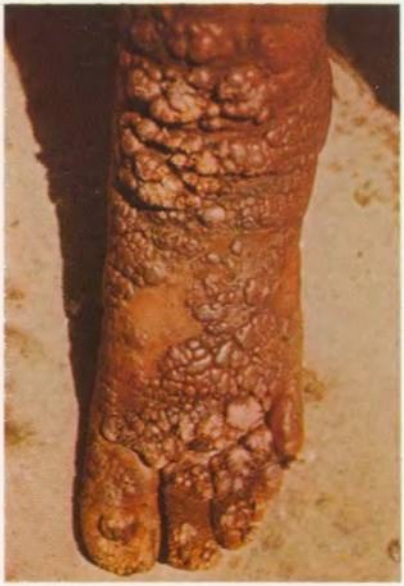

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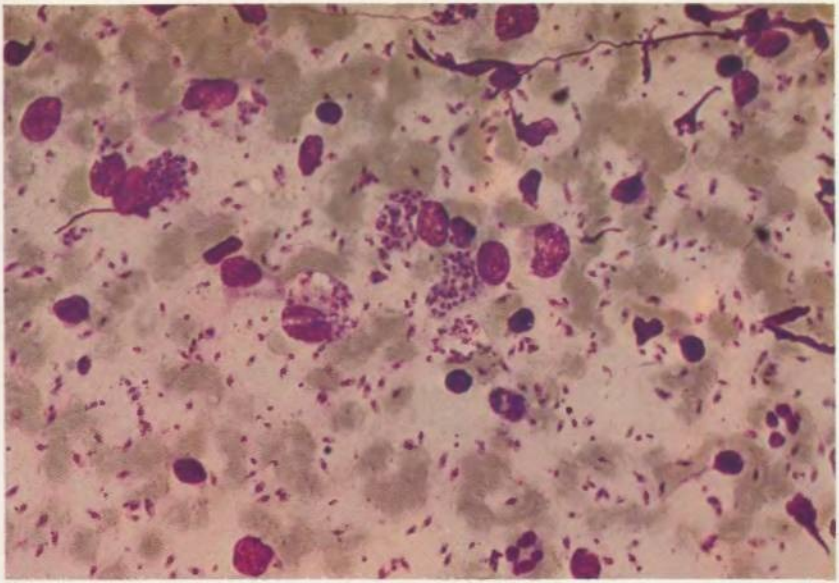


Wuchereria bancrofti and *Brugia malayi*

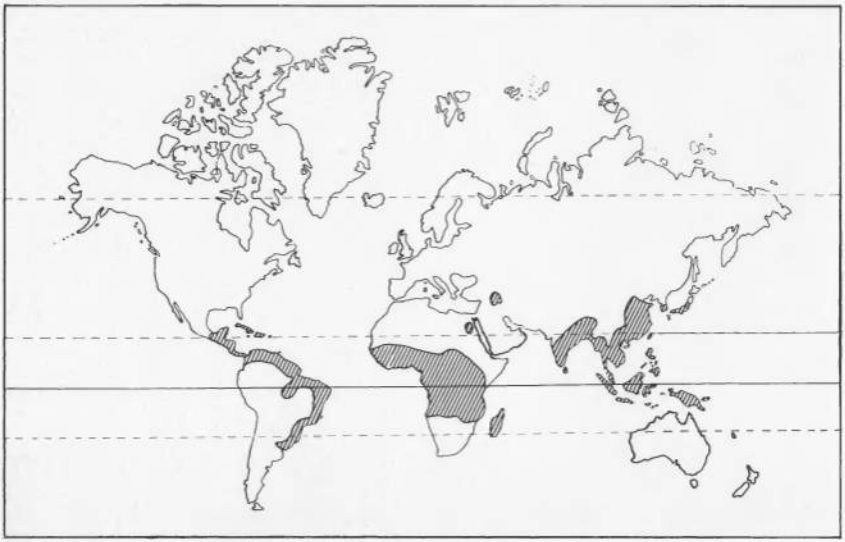
213 Distribution *W. bancrofti* is widely distributed throughout the tropics. Although normally nocturnal, a subperiodic diurnal form occurs in the Eastern Pacific. *B. malayi* has only been recognised in Asia. The periodic form occurs in India, SE Asia and Japan. The subperiodic form occurs only in Malaysia, Borneo and the Philippines.

214 Larvae in mosquito thorax Microfilariae in the peripheral circulation are ingested by mosquitoes when they suck blood. Microfilariae exsheath, migrate from the midgut, and penetrate the thoracic muscles where they mature to sausage-shaped first and second stage larvae. *(× 100)* Differential characters of some of the mosquito vectors, *Mansonia*, *Culex* and *Aedes*, are shown in **217–223** (*see also* **2–15; 768**).

215 Infective larvae After about two weeks they develop into third stage, filariform larvae which enter the proboscis. These infective larvae later penetrate the skin of a new host through the puncture wound made when the mosquito bites. *(× 20)*

*(*See* **Tables VIII & IX**)

213



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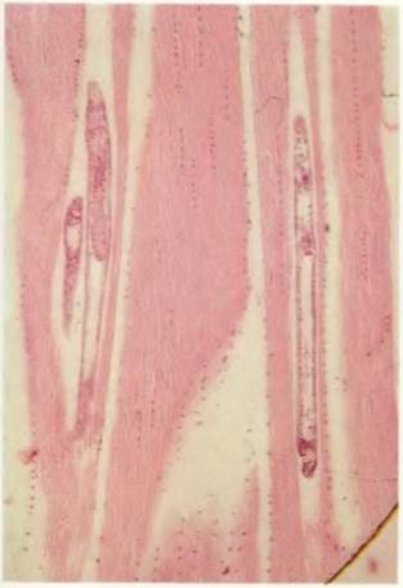

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216 Female *Aedes (Stegomyia)* polynesiensis Night biting *Culex pipiens fatigans* and various species of *Anopheles* are the main vectors of the nocturnal periodic form of *W. bancrofti*. Day biting *Aedes polynesiensis* transmits the subperiodic form of *W. bancrofti* in various Pacific islands. Species of *Mansonia* are the main vectors of *B. malayi*. Compare the hump-backed stance with that of Anopheles (**62**). *(× 5)*

217–219 Aquatic stages of *Culex* and *Mansonia* Unlike Anopheles mosquitoes (*see* **61**) or those of *Culex* (**217**) *Mansonia* larvae (**218**) and pupae (**219**) are attached by their breathing tubes (siphons) to underwater roots, stems and leaves of aquatic plants. Note the saw-edged tips of the siphons. Their ideal breeding habitats are open swamps with *Pistia*, water lilies and other aquatic plants. *(× 60)*

217

218

219

220 & 221 Tarsal claws of *Aedes* and *Culex* The tarsal claws of *Aedes* (**220**) have strong hooks and a simple pulvillus. *(× 1 000)*. *Culex* (**221**) has fleshy pulvilli and no hooks. *(× 1 300)* (In these scanning electron micrographs the hooks are not seen in the *Aedes*. The contrast between the simple pulvillus of *Aedes* and the fleshy pulvillus of *Culex* is clearly demonstrated.)

220

221

222 & 223 Wing scales of *Culex* and *Mansonia* The adults are distinguished from other culicines by the typical large wing scales. *Culex* (**222**); *Mansonia* (**223**). *(× 370)*

224 Peridomestic culicine breeding site near Delhi Septic pits and drains containing stagnant water are ideal breeding grounds for *Culex pipiens fatigans* which is a peridomestic vector of bancroftian filariasis.

225 & 226 Male and female *W. bancrofti* adults On maturation the infective larvae copulate and the adult filariae become localised in lymph glands, eg in the groin. Adult male *W. bancrofti* (**225**) are about four cm long, females eight to 10 cm (**226**). *(× 8)*

222

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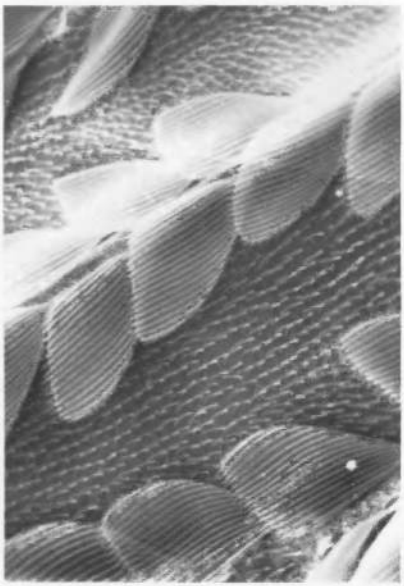

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226

227 Lymphangitis Acute involvement of lymphatic vessels is common, especially in the extremities. In association with lymphangitis there is almost always some local lymphadenitis and fever.

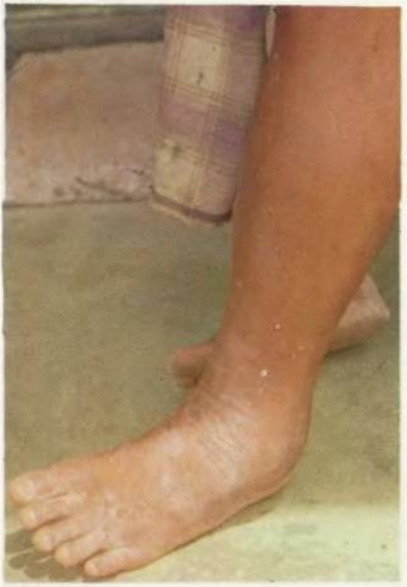
228 Hydrocele In the acute stages orchitis may occur. It is commonly associated with hydrocele and microfilariae may be found in the hydrocele fluid.

229 Elephantiasis due to *B. malayi* In regions of high endemicity, lymphatic obstruction may occur, especially in the leg; progressing in chronic cases to the grotesque extreme called 'elephantiasis'. This may also arise in the arm, breast or scrotum. By this stage microfilariae are rarely found in blood films.

230 Elephantiasis of right epitrochlear gland in a Fijian A feature which is unusually frequent in the South Pacific and is also due to *W. bancrofti* is gross enlargement of the epitrochlear lymph node.

231 Elephantiasis of the scrotum due to *W. bancrofti* in Tahiti Severe elephantiasis of the scrotum may produce gross and incapacitating deformity that requires radical surgery to remove the surplus tissue.

227



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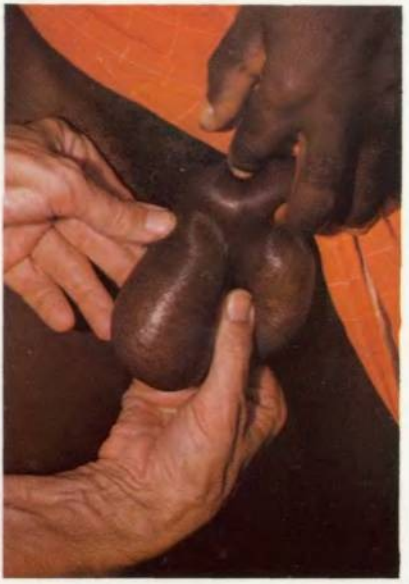

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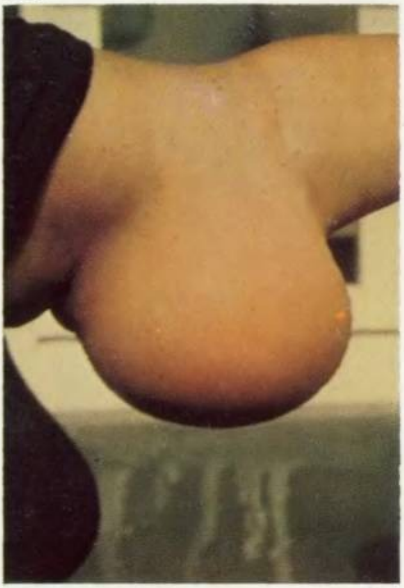
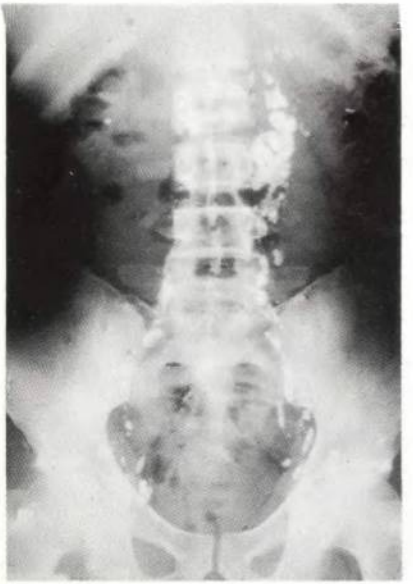


231

232 & 233 Lymphogram in patient with chyluria Obstruction of the cisterna chyli or its tributaries may occur. The dilated lymph vessels rupture and discharge chyle into the urinary tract, thus producing the milky appearance known as chyluria (**233**).

232

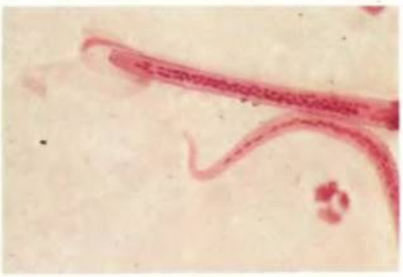


233

234–244 Microfilariae in blood films and skin snips If microfilariae are present in the peripheral circulation they can usually be found by examining a fresh preparation of blood taken between 10 pm and midnight. The worms may be seen in a wet preparation but morphological differentiation is only possible after suitable staining with a Romanowsky stain (Leishman or Giemsa), or haematoxylin. *W. bancrofti* (**234**); *B. malayi* (**235 & 236**); *L. loa* (**237 & 238**); *M. ozzardi* (**239**); *T. perstans* (**240**); *O. volvulus* (**241 & 242**); *T. streptocerca* (**243 & 244**)*. *(× 600)*

* See **283–285** for skin snip technique for these species.

234



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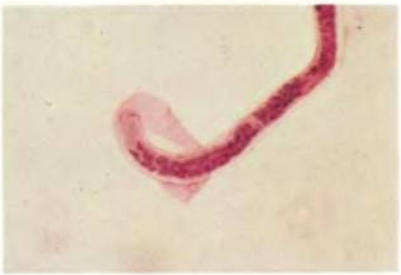

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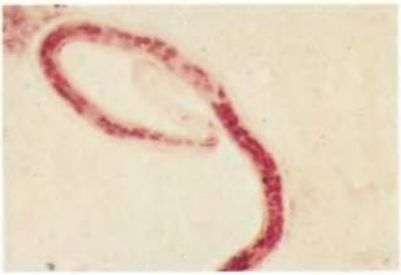

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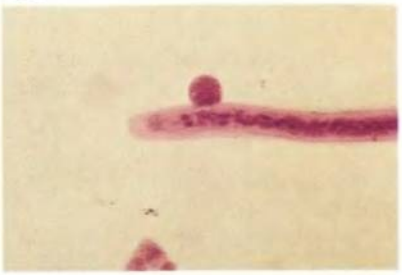

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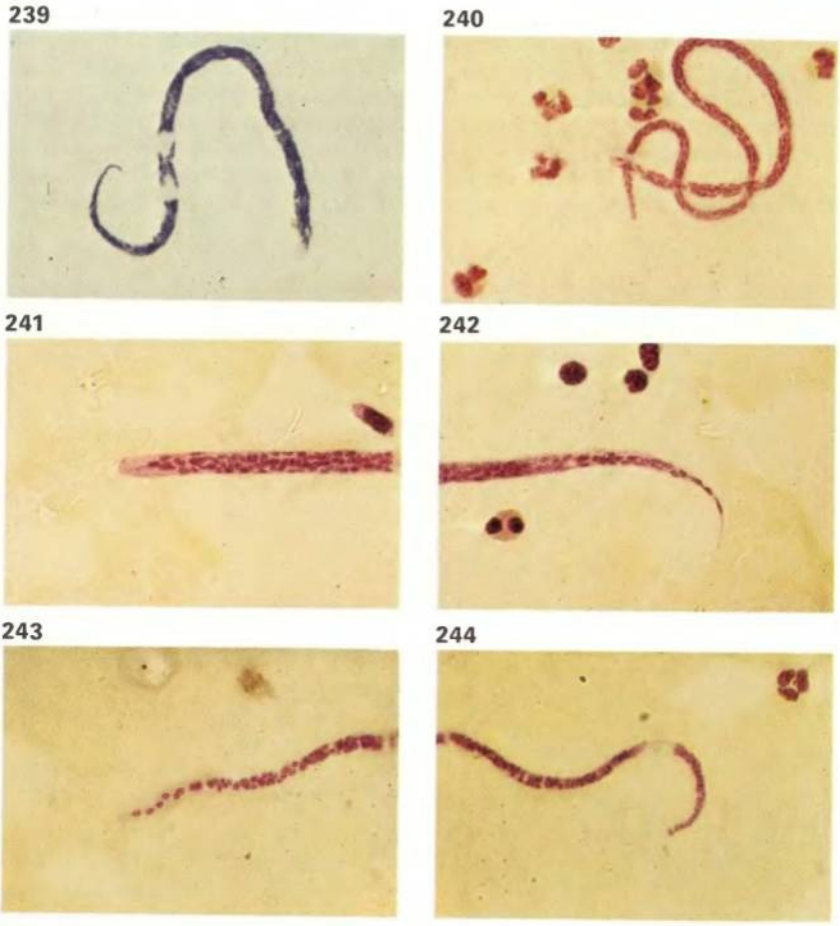
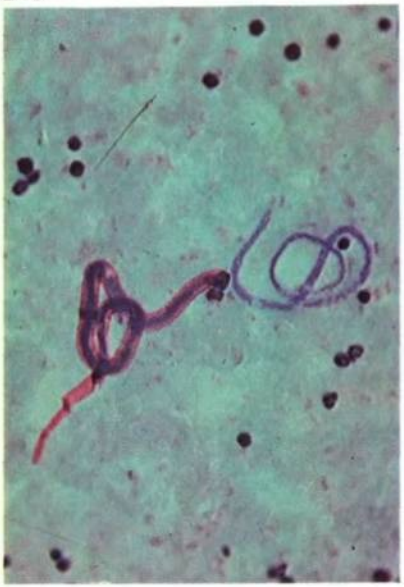


245 Giemsa-stained microfilaria The sheathed microfilaria of *W. bancrofti* (left) is easily distinguished from that of *B. malayi* (right) by its size, the staining characteristics of the sheath in Giemsa stain, and the distribution of the nuclei in the posterior end. (cf **234–236**) *(× 350)*

246 Microfilarial counts Counts of microfilariae per unit quantity of blood are necessary for the epidemiological evaluation of filariasis (although not for individual diagnosis). 20 cu mm pipettes are commonly used to make a thick blood film of specified size.

245



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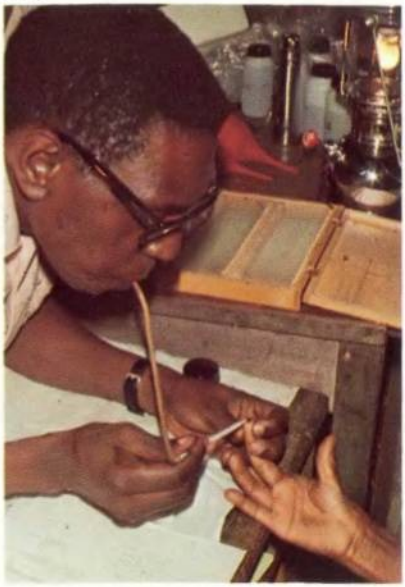

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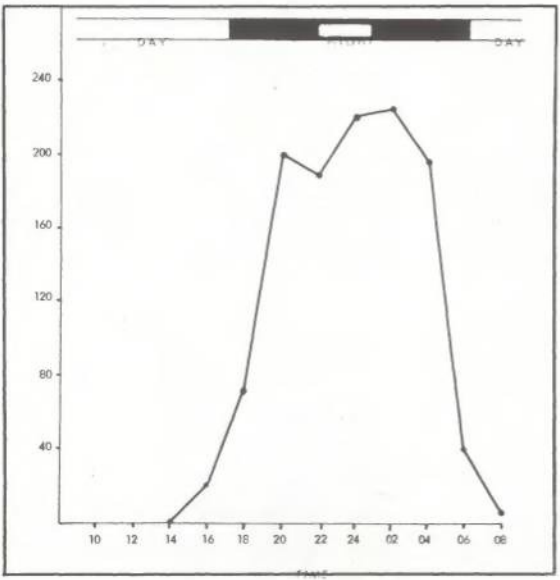
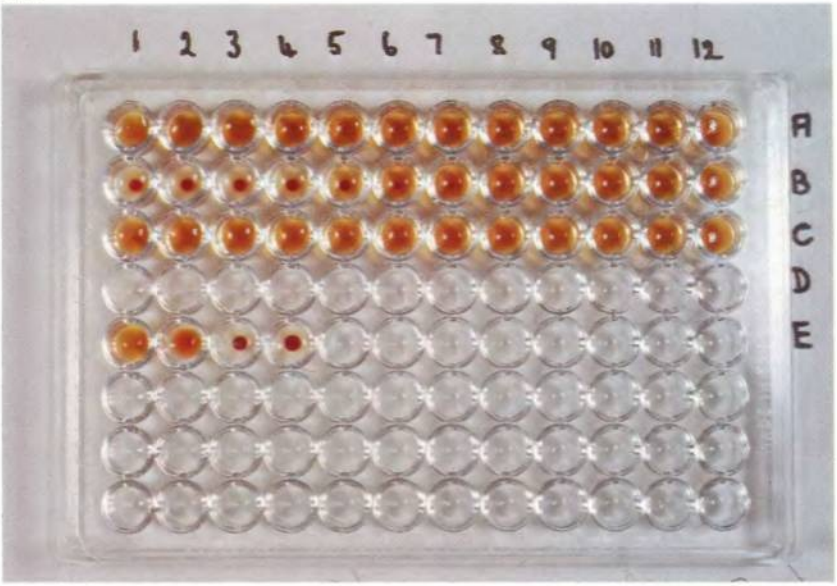


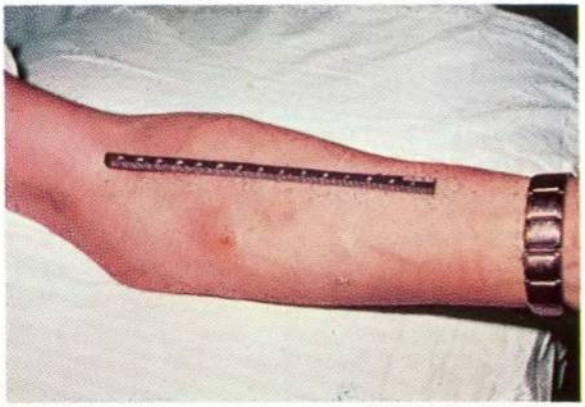
247 Circadian rhythm In a typical *W. bancrofti* infection parasite counts reveal a marked nocturnal periodicity. In the diurnal subperiodic type microfilariae are readily seen in blood taken during the day.

248



248 Serological diagnosis of filariasis The Complement Fixation Test using *Dirofilaria immitis* of the dog as antigen will confirm a diagnosis of filariasis, but it does not distinguish between the different species of filaria. (Row A negative control serum; row B shows a positive titre of 1/64; row C is a negative reaction from another patient; E is complement control.)

249



249 Positive skin test with *Dirofilaria* antigen A similar antigen may also be used to demonstrate delayed cutaneous hypersensitivity.

Tetrapetalonema perstans

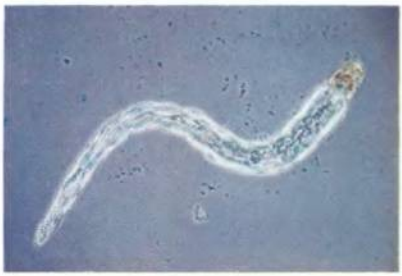
250 Larvae of *Culicoides* *T. perstans* is found in tropical Africa and coastal regions of Central and South America. The vectors of *T. perstans* are small speckled wing flies of the genus *Culicoides* of which *C. austeni* and *C. grahami* appear to be the main vectors in West Africa. The aquatic stages are commonly found in tree holes, leaf axils and other small natural water containers. *(× 60)*

251 Pupa of *Culicoides* *(× 6)*

252 Adult *Culicoides* biting *(× 5)*

253 Wing of *C. grahami* *(× 15)*

250



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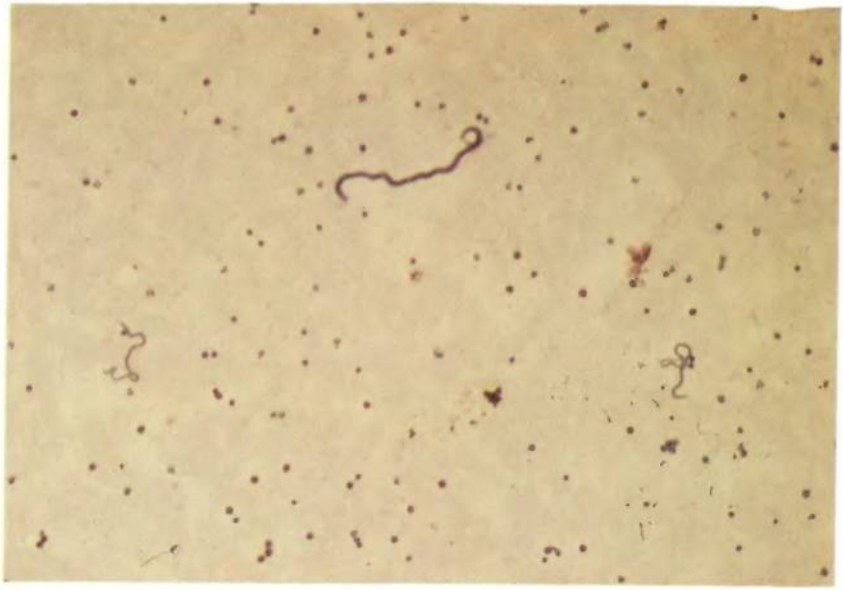


254 *T. perstans* and *Loa loa* in blood film The unsheathed microfilaria of *T. perstans* is readily distinguished from the sheathed microfilaria of *Loa loa* (or that of *W. bancrofti*) in blood films by its smaller size, even at a fairly low magnification. Multiple infections with several species of blood-dwelling microfilariae are common. *T. perstans* infection is usually asymptomatic and may last many years. The adults live in body cavities, the peritoneal cavity being the commonest site. Males measure about 45 mm in length, females about 70 to 80 mm. *(× 125)*

Loaiasis

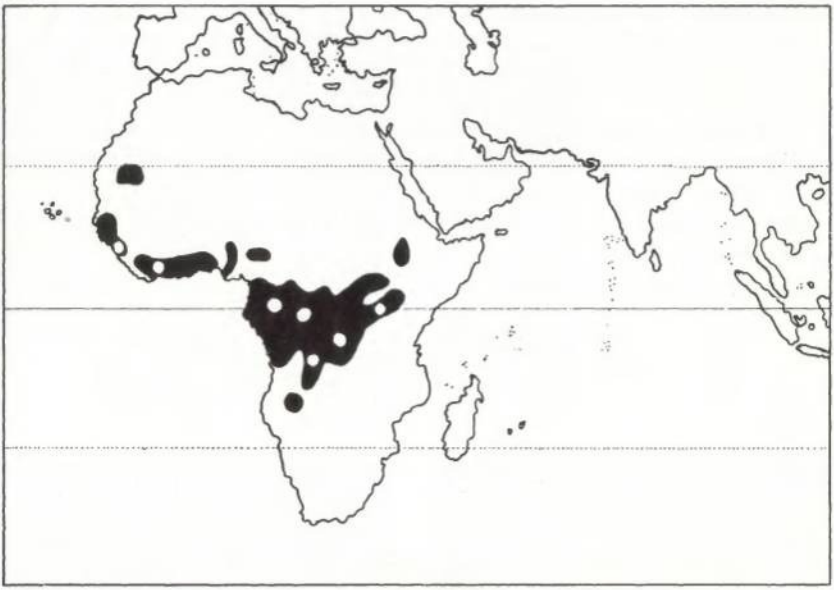
255 Distribution of loaiasis Loaiasis is confined to Africa, extending from the Gulf of Guinea in the West to the Great Lakes in the East. Infection with an almost identical parasite is common in these areas in certain monkeys such as the mandrill.

Tropical rain forest

Loaiasis recorded

256a & b Female *Chrysops dimidiata* biting Tabanid flies of the genus *Chrysops*, particularly *C. dimidiata* and *C. silacea*, transmit loaiasis. The flies **(256a)** *(× 4)* live in the canopy of primary rain forests **(256b)**. (see also **769**)

255



256a

256b

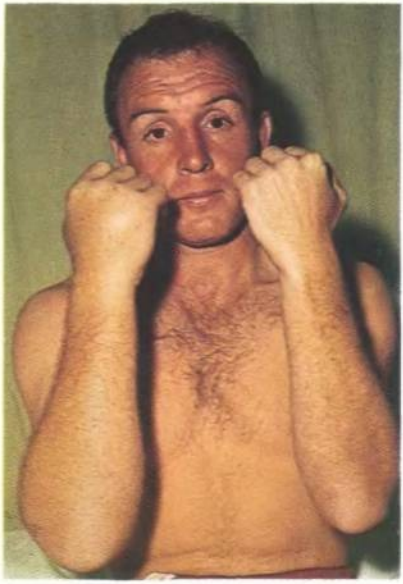
257 Calabar swelling Recurrent large swellings lasting about three days are characteristic and indicate the tracks of the migrating adults in the connective tissue. They are most frequently seen in the hand, wrists and forearm. A marked eosinophilia (60 to 90%) accompanies this phase of the infection.

258 Adult *Loa loa* in the eye The movement of the adult worm under the conjunctiva gives rise to considerable irritation and congestion. The adult is arrowed in this figure.

259 Extraction of worm The adult worm can be extracted with fine forceps after anaesthetising the conjunctiva.

260 Tail of male *Loa loa* *(× 90)*

257



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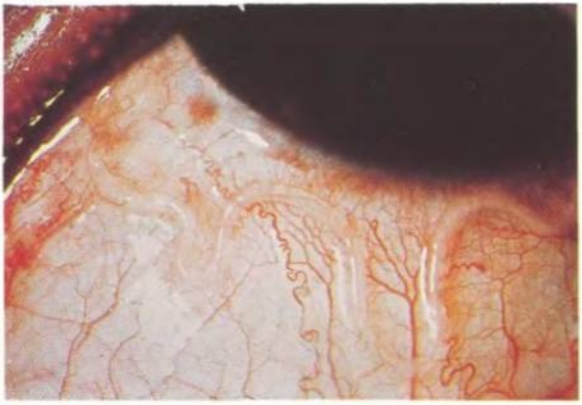

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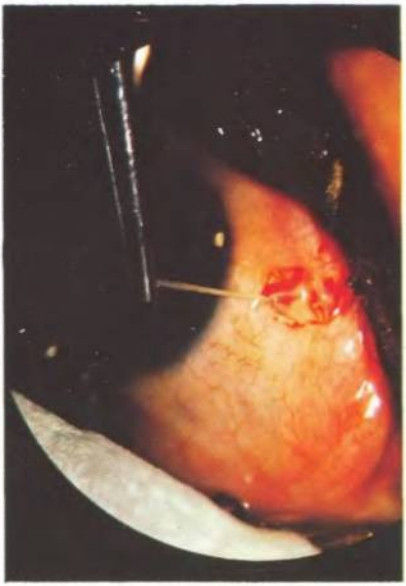

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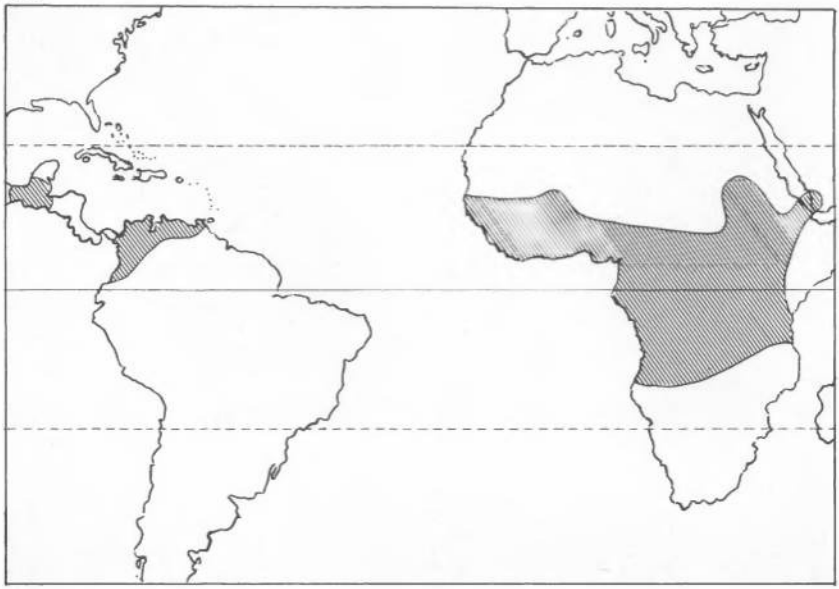
Onchocerciasis

261 Distribution of onchocerciasis *Onchocerca volvulus* is a tissue dwelling nematode, the microfilariae of which are found predominantly in the skin and eye. Onchocerciasis has a focal distribution in Africa and South America. It is endemic in West Africa, equatorial and East Africa and in the Sudan. A small focus is known also in Yemen. It occurs in Central America and in parts of Venezuela and Colombia.

262 & 263 Aquatic stages of the vectors The filarial worm is transmitted by *Simulium* or 'Buffalo flies'. Species of the *S. damnosum* complex are the vectors in West Africa, *S. naevei* in East Africa, and *S. ochraceum* and *S. metallicum* in Central and South America. The larvae **262** and the pupae **263** are attached to submerged objects in fast running water from which they extract oxygen through head filaments (*see also* **267**). *(× 6)* (*see also* **770**)

264 Adult *Simulium damnosum* *(× 10)*

261



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265 Typical locality of *Simulium* in West Africa Fast moving, highly oxygenated water in streams, rivers, waterfalls, etc provides the essential ecological environment. The figure shows a branch of the Upper Volta river in Ghana during the rainy season. In some hyperendemic villages in West Africa a third of all adults may be blinded by onchocerciasis.

266 *Simulium pupae* attached to rocks Similar river in the dry season showing stone boulders in the bed of the stream which, by causing eddies in the water, provide enough oxygenation to permit the *Simulium* to survive while the river level is low.

267 *Simulium pupae* on a crab In East Africa larvae and pupae of the *S. neavei* complex are attached to fresh-water crabs.

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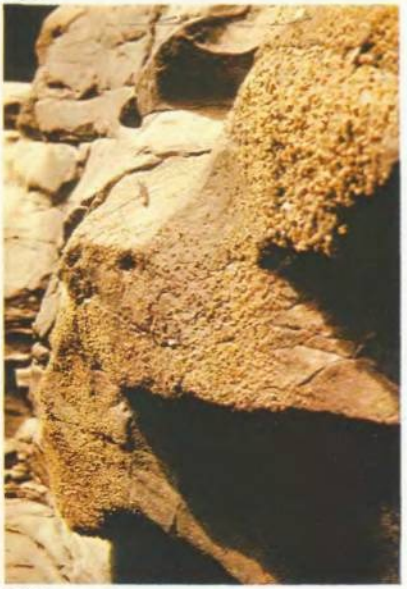

267

268 ***Onchocerca*** **nodules on iliac crests** The adult filariae become encapsulated in fibrous material which forms nodules in the subcutaneous tissues. They are found predominantly in the lower part of the body in Africa, while in South and Central America they are more commonly found on the head and upper trunk.

269 Macroscopic section of nodule In this gross section of a nodule the adult worms are seen entwined. *(× 2)*

270 Transverse section of nodule A microscope section through an onchocercal nodule, showing adult worms and microfilariae. *(× 20)*

271 Microfilaria in skin biopsy The microfilariae migrate to the skin and the eye. *(× 350)*

272 Onchocercal changes in skin Onchocerciasis is characterised by lesions in two main sites, the skin and the eye. A pruriginous condition commonly called 'craw craw' in Africa involves irregular, broad areas of the skin where small papules form around the microfilariae.

268

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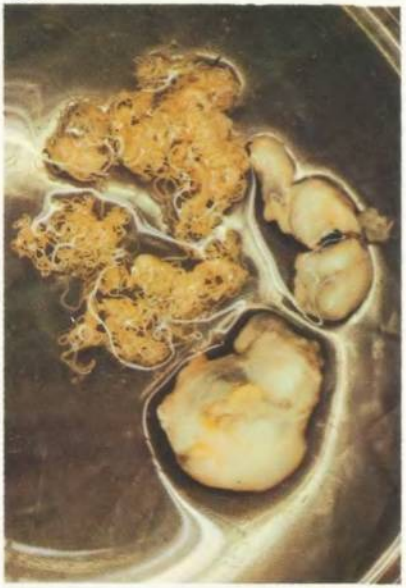

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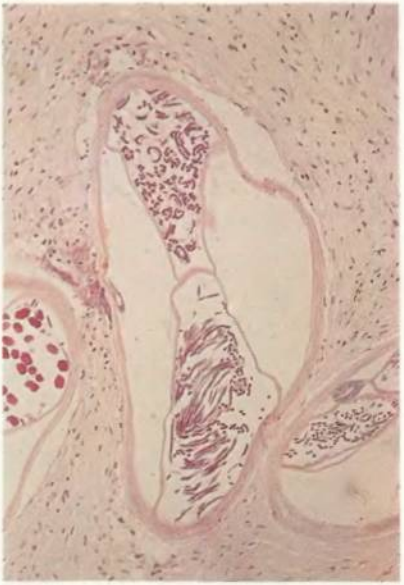

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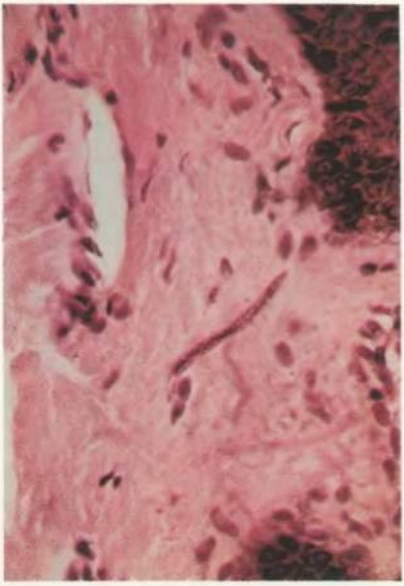


272

273 'Elephant' skin Thickening and wrinkling of the skin give rise to the 'lizard' or 'elephant' skin appearance.

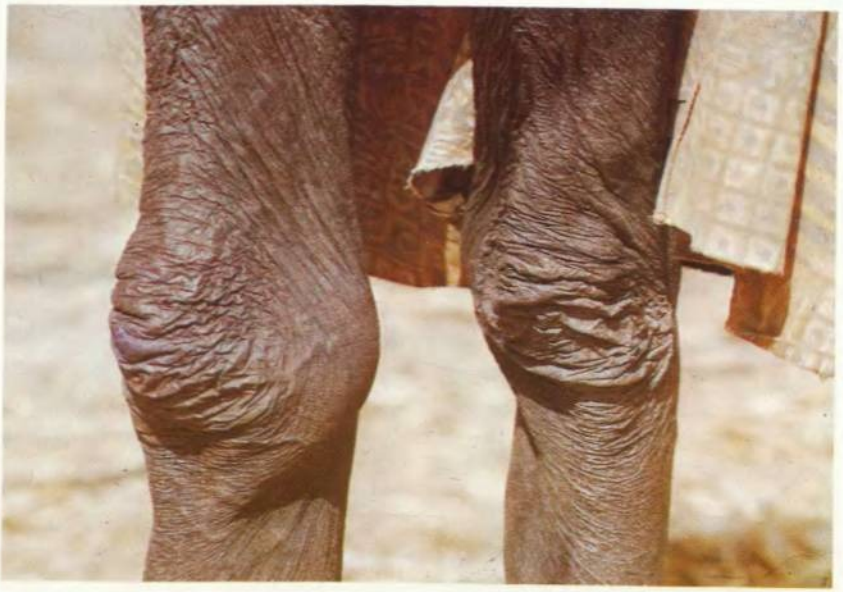
274 Chronic dermatitis of knee In long standing lesions, a chronic onchodermatitis with gross convoluted appearance of the skin and lichenification may occur.

275 Depigmentation Pretibial atrophy and depigmentation in a patient with late (burnt-out) onchocerciasis. This condition is sometimes called 'leopard skin'.

276 'Tissue paper' skin In chronic infections atrophy of the skin may occur resulting in a 'tissue paper' appearance.

277 'Hanging groin' Involvement of the inguinocrural glands can result in an appearance described as 'hanging groin'.

273



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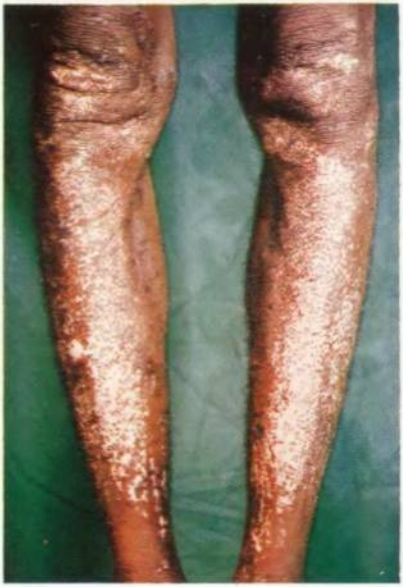

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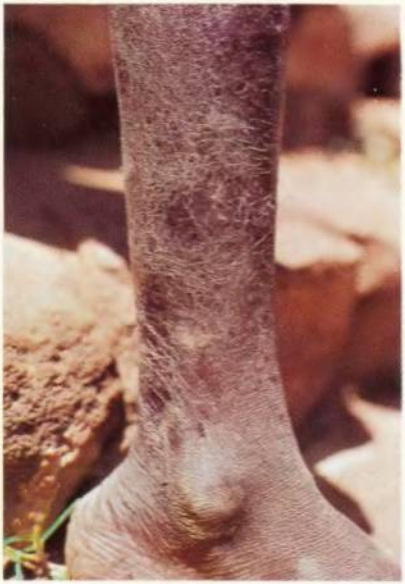


277

278 Hanging groin and scrotal elephantiasis.

279 Sowda A peculiar feature ot onchocerciasis in the Yemen is a condition known as Sowda which is characterised by hyperpigmentation, usually of one of the lower limbs, often accompanied by inguinocrural lymphadenopathy.

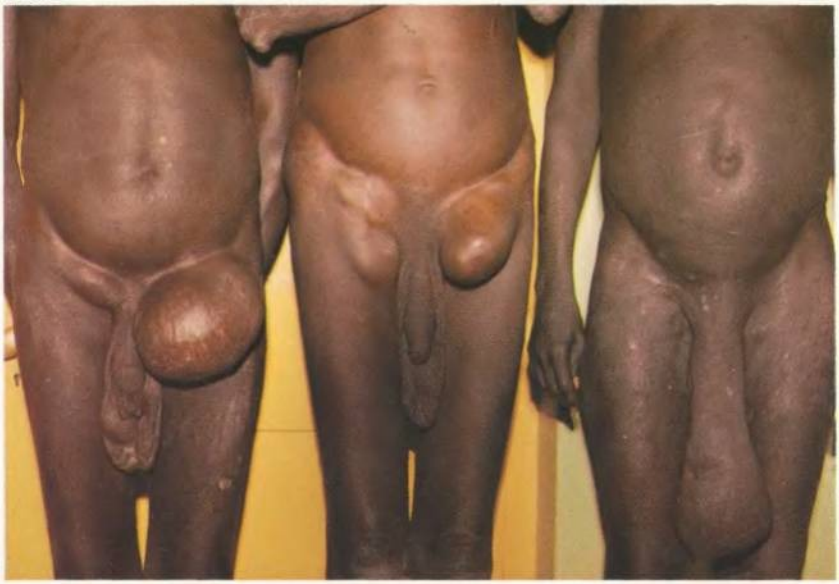
In Central America 'Erisipela de la costa' is characterised by an erythematous appearance of the face or upper trunk. It occurs in heavily infected young people, usually under 20 years of age. Purplish tinged plaques or papules may be observed in Central America in patients usually of an older age group. This condition is known as 'mal morado'.

280 Early corneal involvement The tissue reaction associated with dead microfilariae in the cornea gives rise to a number of 'snowflake'-like opacities as seen in the figure. This punctate keratitis may clear with time.

281 Sclerosing keratitis Heavy microfilarial infection of the cornea leads to the development of progressive, sclerosing keratitis which commonly produces blindness.

282 Optic atrophy A variety of choroidoretinal lesions may follow damage by microfilariae to the anterior segments of the eye, and finally optic atrophy may develop as seen in this eye.

278



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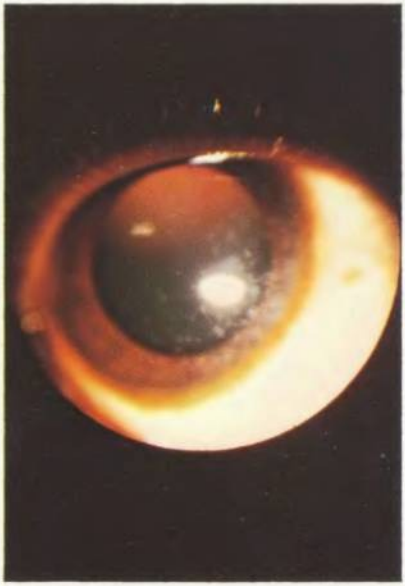

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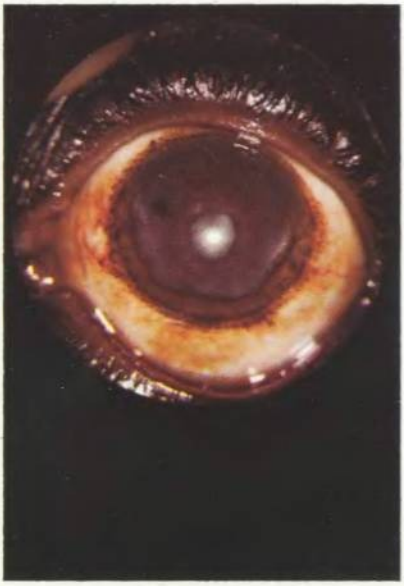

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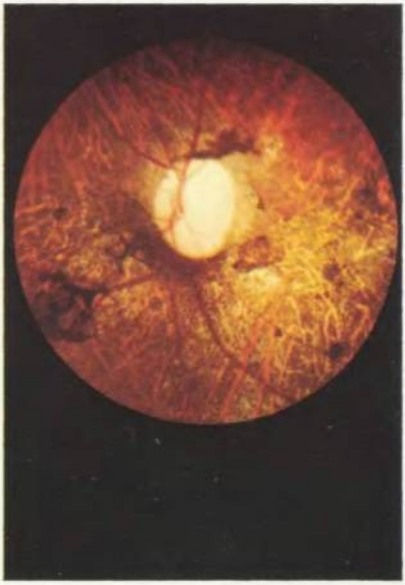
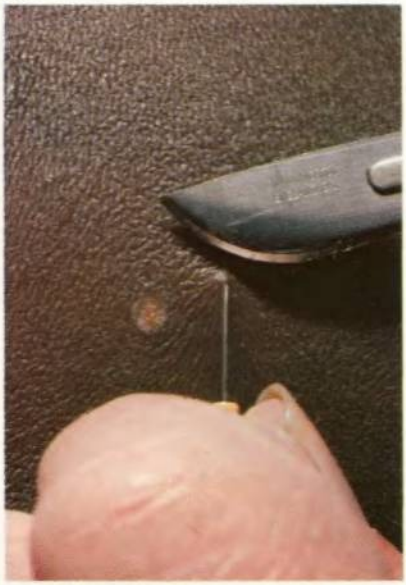


283 & 284 Skin snip technique With a razor blade or scalpel, a tiny piece of skin, often from the back of the shoulders, iliac regions or calf, is snipped off and placed in a drop of saline on a microscope slide under a cover slip.

285 Living microfilariae of *O. volvulus* After some minutes actively moving microfilariae emerge from the skin into the surrounding saline. *(× 600)*

286 Slit lamp examination Slit lamp examination often reveals numerous microfilariae in the anterior chamber of the eye. They are best looked for in the inferior medial quadrant.

283



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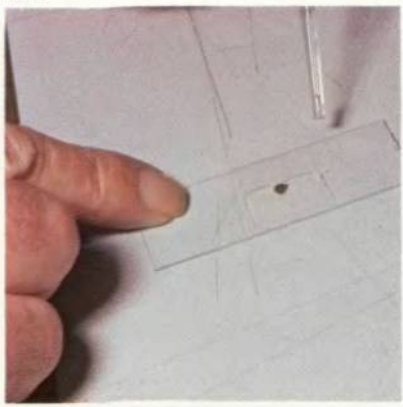

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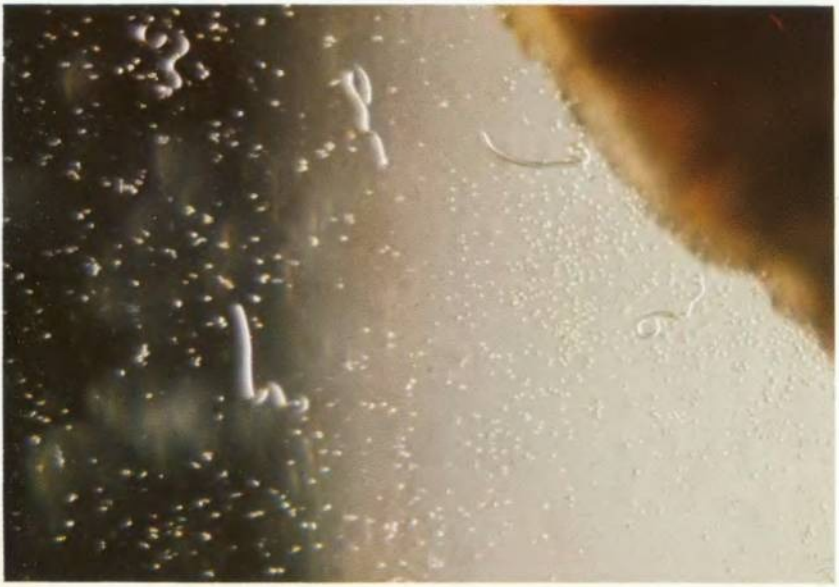

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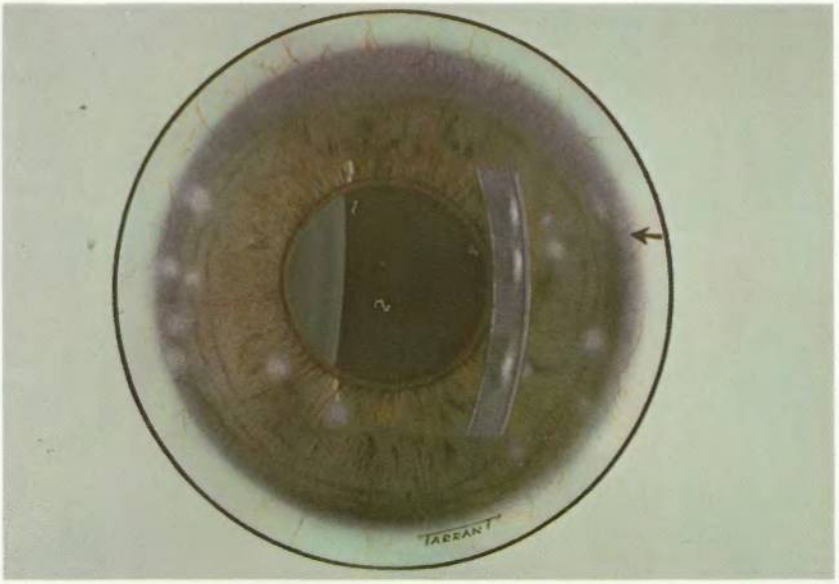


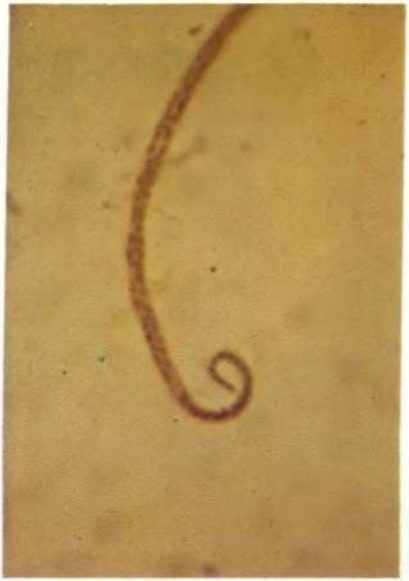
287 Nodulectomy Serious eye changes can be prevented in some early cases by excising the nodules containing the adult worms, thus preventing the continuing production of the microfilariae which are the actual pathogenic agents in this disease. Nodulectomy has been widely employed in Central and South America.

287

Other Filariases

288 & 289 Tails of microfilariae *T. streptocerca* and *O. volvulus* *T. streptocerca* (**288**), another skin dwelling unsheathed microfilaria must be distinguished from that of *O. volvulus* (**289**) in African patients. For this a stained preparation should be examined, eg with haematoxylin. *T. streptocerca* produces few pathogenic effects. It is transmitted by *Culicoides grahami*, also infects chimpanzees, and is only known in Africa. *(× 900)*

288



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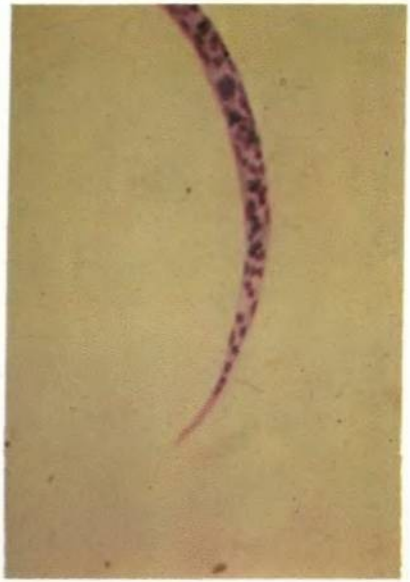
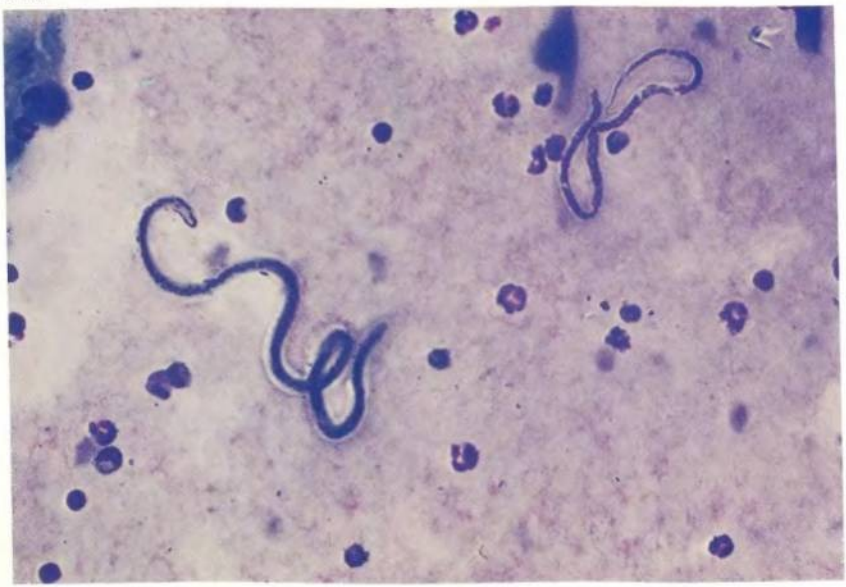


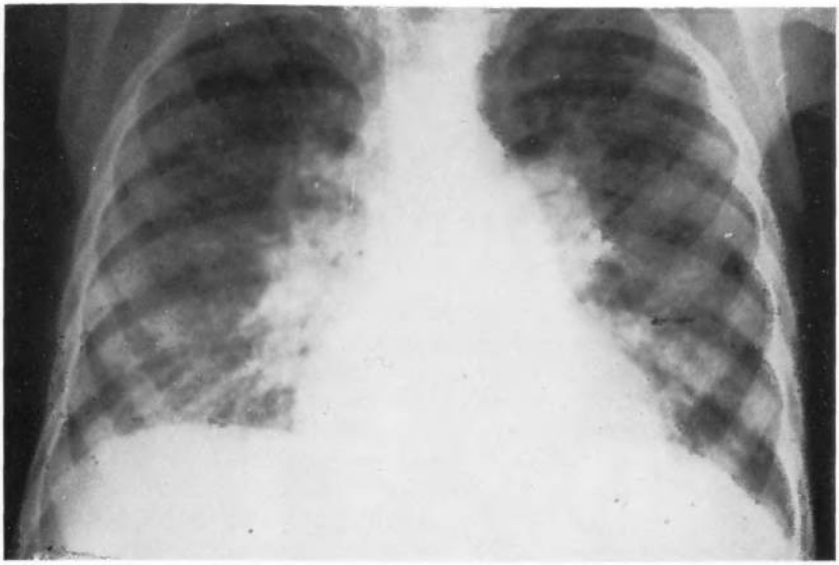
290 Microfilaria of *Mansonella ozzardi* This unsheathed microfilaria (right) is found in the blood in parts of South America and the Caribbean. The microfilaria is readily distinguished from the larger and sheathed microfilaria of *W. bancrofti* (left) which also occurs in parts of South America. Vague symptoms of various types have been attributed to infection with the parasite which is also transmitted by *Culicoides* spp. *(× 500)*

291 Eosinophilic lung This is a peculiar allergic reaction to filarial infections of animal origin. It occurs principally in Southeast Asia and particularly affects Indians. The condition is characterised by nocturnal cough and bronchospasm, with transient shadows in the lungs.

292 Eosinophilia Eosinophilia is very marked and the condition responds well to specific filaricides. A typical leucocyte response to healing with diethylcarbamazine is shown. H = Diethylcarbamazine (Hetrazan); E = eosinophils/cu.mm; D = days.

290





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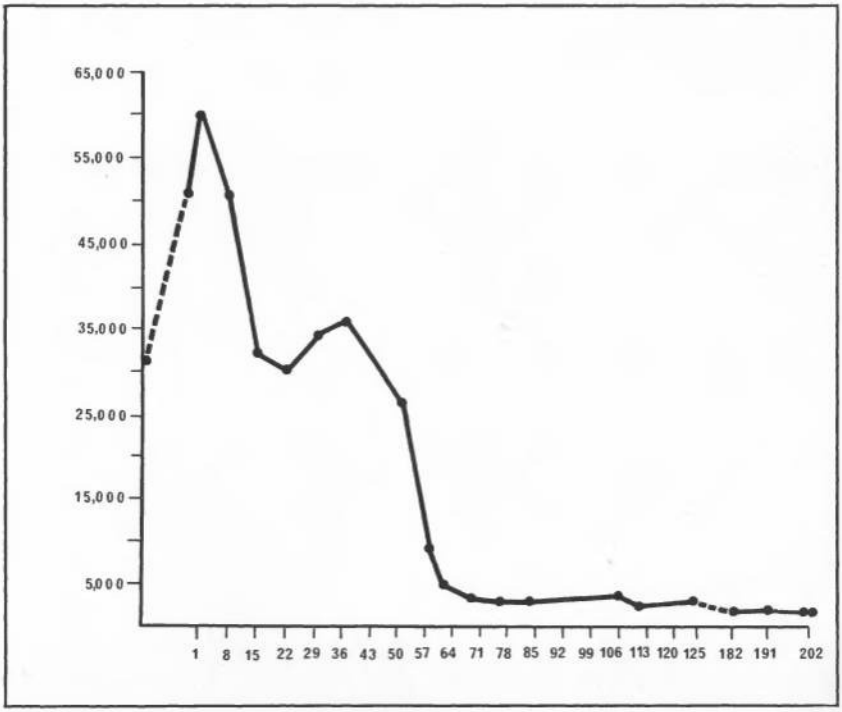
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Part II
Soil-mediated Helminthiases

Soil-transmitted helminthic infections are of two types; the hookworms which undergo a cycle of development in the soil, the larvae being infective, and a second group of nematodes which merely survive in the soil as eggs that have to be ingested in order for the cycle to continue. The geographical distribution of the hookworms is limited by the requirements of the developing larvae for warmth and humidity. Generally speaking, the second type can occur not only in the tropics and subtropics, but also in temperate regions. All these helminthiases provide an index of the level of hygiene and sanitation in a community since they depend for their dispersal on the indiscriminate deposition of faecal material on the ground, the use of untreated night soil as an agricultural fertiliser and similar unsophisticated human habits. In temperate as in other areas, those infections that are spread directly, ie through the ingestion of eggs, are common in microenvironments which favour such spread, notably homes for mentally subnormal people, refugee camps, orphanages, etc. The provision of adequate sewage disposal facilities virtually excludes these diseases.

The hookworm infections are transmitted through soil-dwelling infective larvae that penetrate the skin. Faecal contaminated soil in the neighbourhood of human habitations or on farmland is the source of infection for the barefooted inhabitants. Conversely, the use of footwear greatly reduces the prevalence of hookworm infection. Barefooted rubber tappers in Malaya commonly acquire infection from contaminated soil. Larvae of a number of animal hookworm species do not mature in man but the invasive larvae produce a transitory skin eruption as they migrate (cutaneous larva migrans). Visceral larva migrans may be due to infections with eggs of the dog or cat roundworm (*Toxocara canis* etc). Here too the larvae do not mature in man but may set up inflammatory reactions in the viscera, especially the liver, or in the eye.

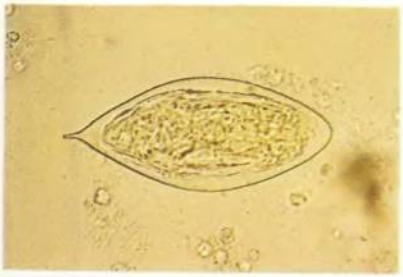
Generally speaking, the degree of harm done to the host is related to the worm burden in these infections. Hookworm disease results when large numbers of adult worms are present and the loss of blood due to the worms cannot be balanced because the host's diet is deficient in iron, etc. Moreover multiple intestinal helminthic infection is the rule in many areas. Heavy infections with *Ascaris* may result in intestinal obstruction. *Trichuris* infection of the large bowel can lead to rectal prolapse in infants.

THE HOOKWORM INFECTIONS

Ankylostoma duodenale and *Necator americanus*

293–307 The eggs of helminths *Schistosoma haematobium* (**293**), *S. mansoni* (**294**), *S. japonicum* (**295**), *Fasciola hepatica* (**296**), *Ascaris lumbricoides* (**297**), *Ascaris* (infertile) (**298**), *Paragonimus westermani* (**299**), *Diphyllobothrium latum* (**300**), Hookworm (**301**), *Trichuris trichiura* (**302**), *Enterobius vermicularis* (**303**), *Hymenolepis nana* (**304**), *H. diminuta* (**305**), *Taenia* (**306**), *Clonorchis sinensis*, *Opisthorchis felineus*, *Heterophyes heterophyes* (**307**). (*See* **Table VIII** for classification. The eggs of the last three species are very difficult to distinguish) *(× 250)*

293



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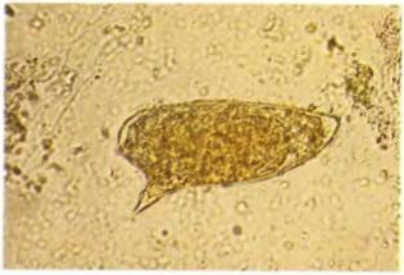

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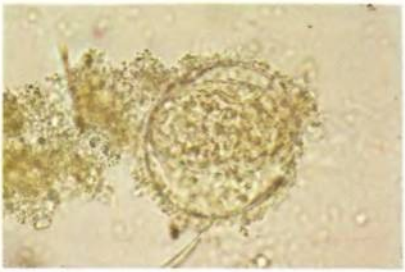

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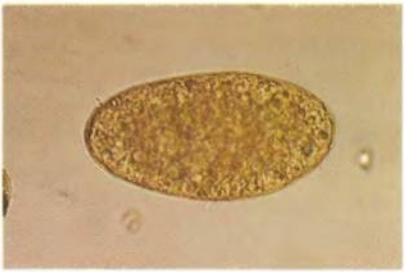

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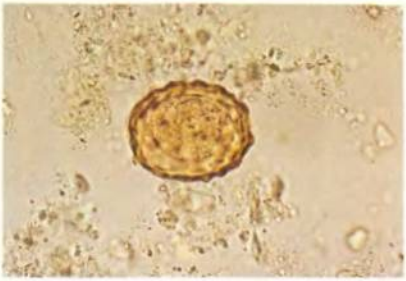

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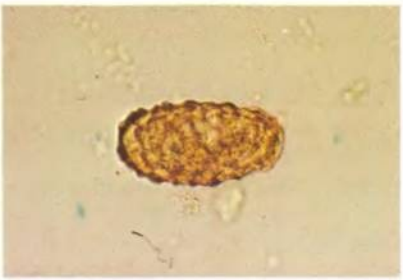

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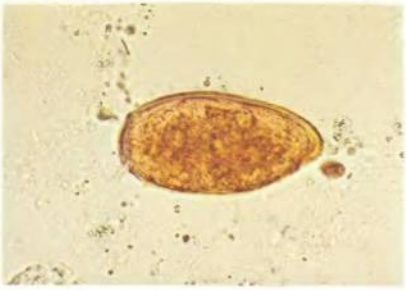

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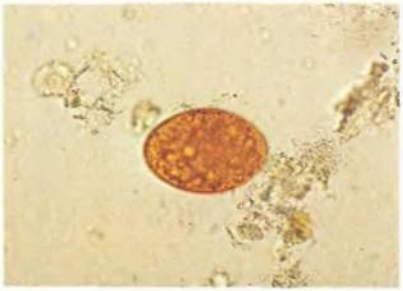

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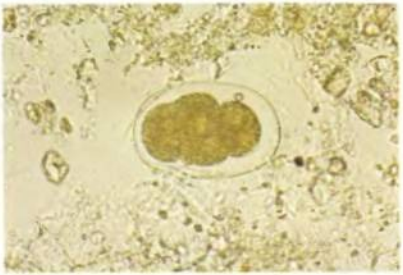

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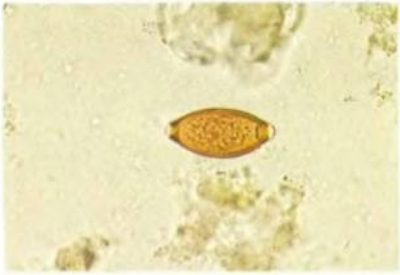

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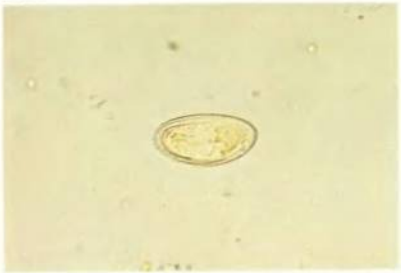

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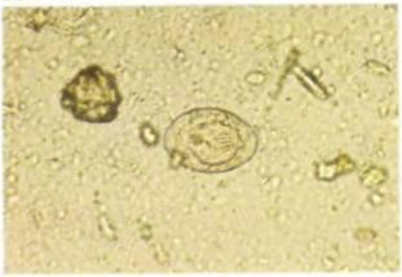

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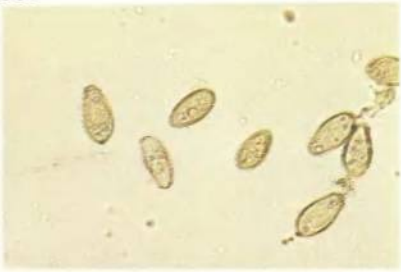


308 Distribution map of hookworm infection The common hookworms of man are *A. duodenale* and *N. americanus*. It is estimated that 1000 million persons are infected with hookworm (about a quarter of the world's population). Usually one species predominates in any one locality. The map shows the approximate areas in which one or other species dominates. Since the larvae can only develop in warm moist soil the distribution of the parasite is limited by climatic conditions.

Ancylostoma duodenale

Necator americanus

309 Comparative size of nematodes

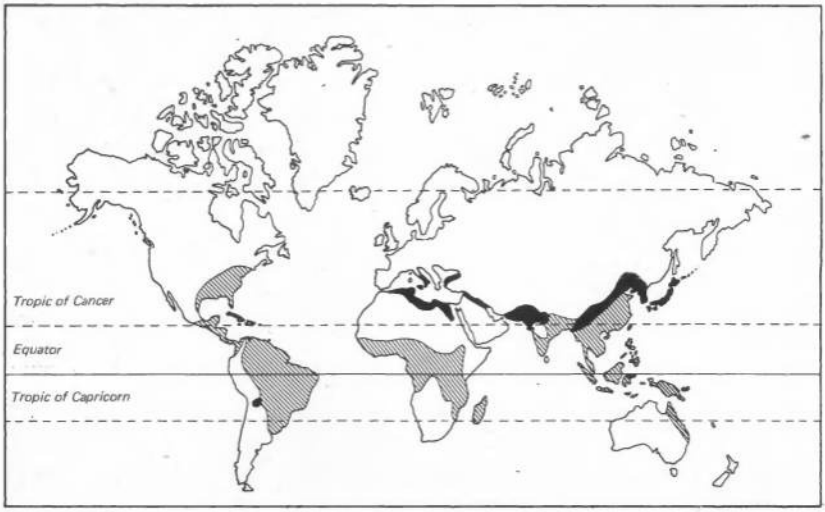
	A	B
First row	*N. americanus* ♀ ♂	*A. duodenale* ♀ ♂
Second row	*E. vermicularis* ♀	*T. spiralis* ♀ and ♂
Third row	*T. trichiura* ♀	*T. trichiura* ♂

310 Filariform larva of hookworm Eggs (**301**) passed in the faeces hatch into rhabditiform larvae in damp soil; they feed and undergo two moults to produce an infective sheathed filariform larva. *(× 350)* (*see also* **771**)

311 Larvae of hookworm in lung of dog These penetrate bare skin, usually of feet or legs and enter the blood stream, to reach the lungs. The larvae then penetrate into the bronchioles, pass into the pharynx and are swallowed. They become attached to the small intestine and mature to adults. *(× 100)*

312 Adult hookworms *in situ* The worms are about one cm long and characteristically curved. They are attached by their buccal capsules to the villi of the small intestine. *(Natural size)*

308



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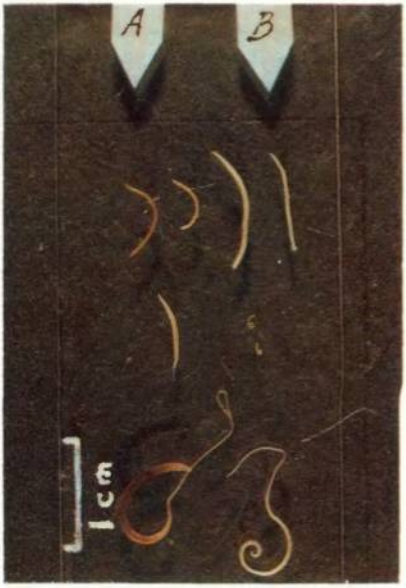

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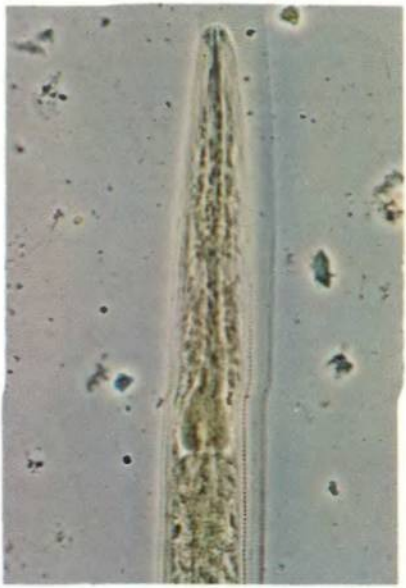

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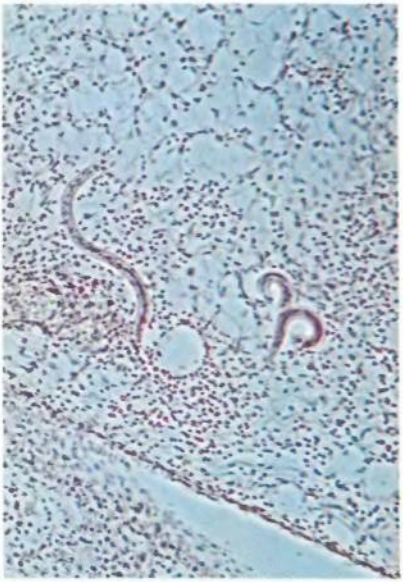
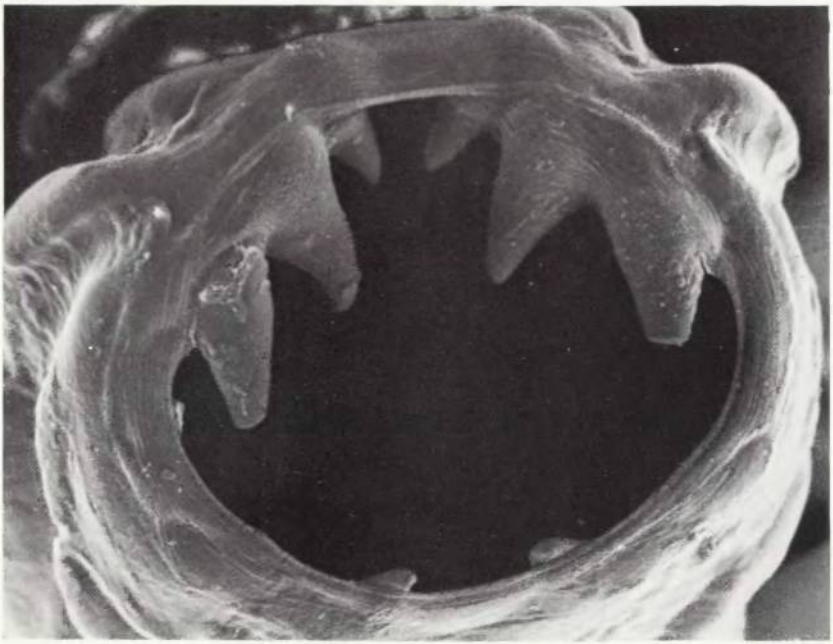


312

313–317 Identification of adult hookworms *A. caninum* **(313)** *(×470)*, *A. duodenale* **(314)** *(×630)*, *A. ceylanicum* **(315)** *(×670)*, *N. americanus* **(316)** *(×470)*, *A. duodenale* **(317)** *(×470)*. The different species may be distinguished by the characteristic morphology of the head capsule **(313–316)** and male bursa **(317)**, seen here in scanning electromicrographs. The male bursae are distinguished by the numbers and pattern of the 'rays'.

313



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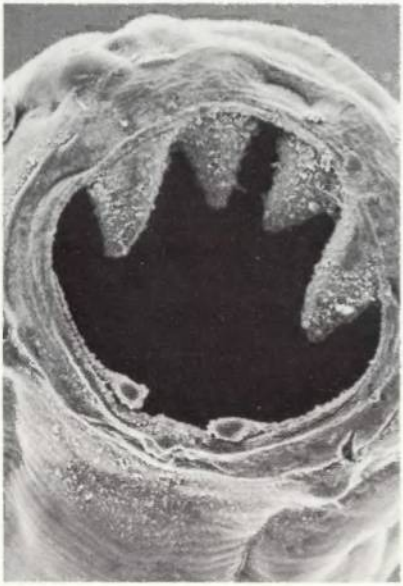

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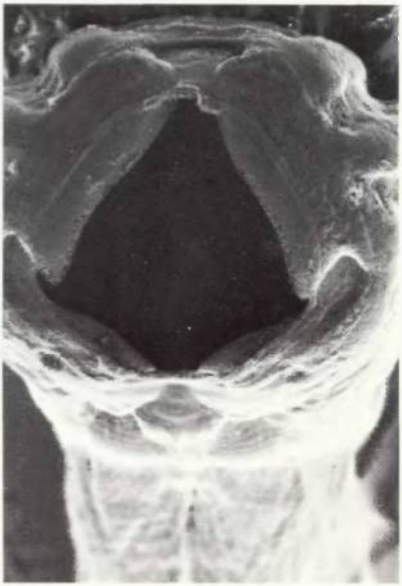

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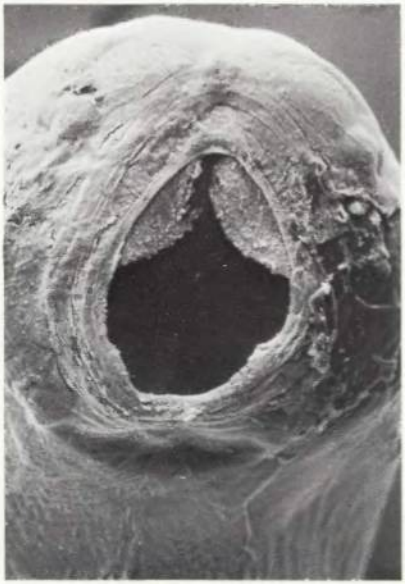


317

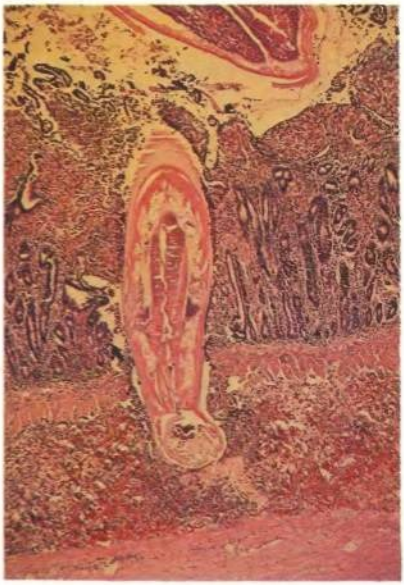
318 Section of adult *A. duodenale in situ* The hookworm feeds by sucking blood from the intestinal mucosa. It has been estimated that a single *A. duodenale* can withdraw as much as 0.2 ml a day while *N. americanus* withdraws 0.05 ml. *(× 20)*

319 Clinical picture of gross hookworm disease Severe anaemia is the classical feature of hookworm disease. This results from high hookworm loads and low daily iron intake. The patients usually complain of lassitude, shortness of breath, while oedema and ascites also occur.

320 Blood film from a patient with hookworm anaemia The typical anaemia resulting from severe hookworm infection is of the iron deficiency type with a low MCHC and low serum iron. *(× 900)*

321 'Creeping eruption' due to larvae of dog hookworms Infective larvae of various species of animal hookworms (eg *A. braziliense*, *A. caninum*, *A. ceylanicum*) frequently fail to penetrate the dermis. They migrate through the epidermis leaving typical serpiginous tracks known also as 'creeping eruption'.

318



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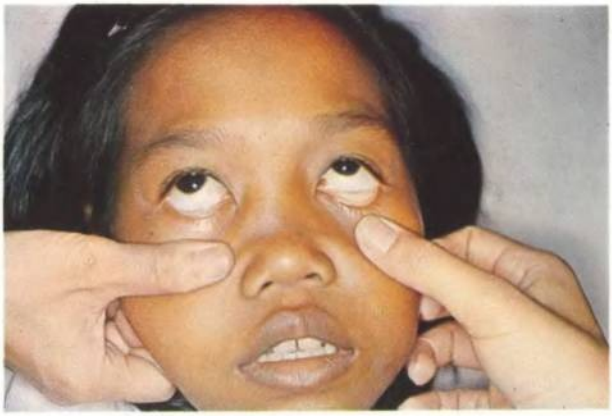

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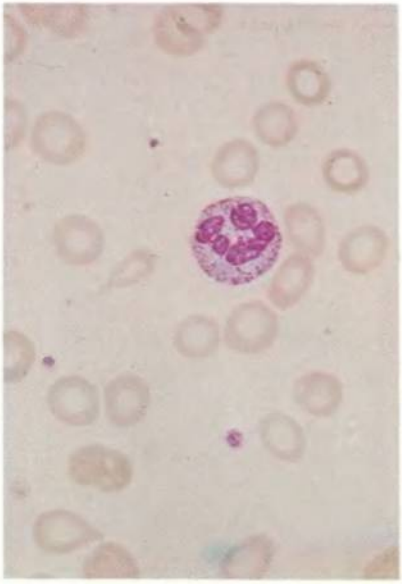

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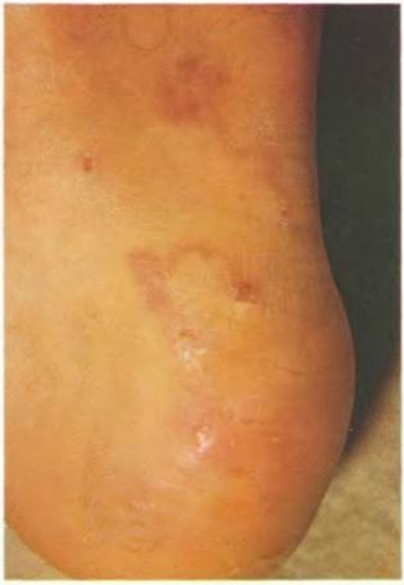


Strongyloidiasis

322 Rhabditiform larvae of *S. stercoralis* in faeces The life cycle of *Strongyloides stercoralis* can follow one of several patterns: (1) Virtually identical to that of the hookworms, except that the rhabditiform larvae hatch in the colon and are passed as such in the faeces. (2) Involving a free-living adult stage in moist soil. Rhabditiform larvae developing from these also grow into infective filariform larvae which penetrate the skin of a new host. (3) Direct auto-infection of the original host by rhabditiform larvae which develop to the infective filariform stage before leaving the intestine. After invading the circulation through the intestinal mucosa the cycle continues in a normal fashion. The picture shows rhabditiform larvae in the faeces. The egg which is rarely seen in the faeces is very similar to that of hookworm. *(× 40)* *(see also* **772**)

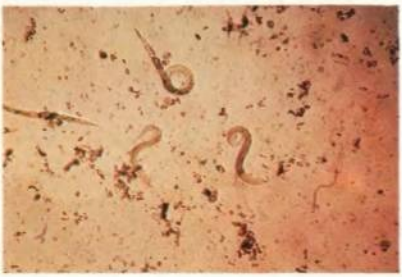
323 Adult male and free-living larva in soil *(× 40)*

324 Migrating larvae of *S. stercoralis* in skin Auto-infection can lead to severe 'creeping eruption' usually in the back. This may occur many years (30 or more) after initial infection. Deep migration of the larvae may be associated with an 'eosinophilic lung' type of syndrome.

325 Sections of parasitic female and eggs embedded in jejunal mucosa Free living females (about one mm long) are smaller than the forms found in the intestine (up to 2.2 mm). Males are normally only found in the soil and are about 0.7 mm long. The parasitic females lying in the intestinal mucosa may reproduce parthenogenetically, the eggs filtering through to the intestinal lumen or first hatching in the mucosa. *(× 90)*

326 Post-mortem appearance of colon The incidental administration of steroids and immuno-suppressive agents may greatly enhance infection with *S. stercoralis* which may even be fatal as in this case. Note the multiple ulcerations and thickening of the wall of the colon.

322



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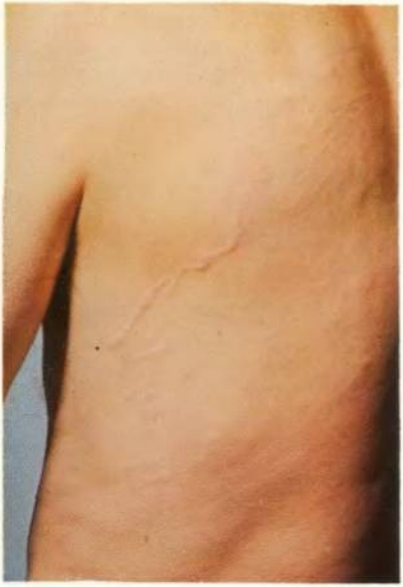

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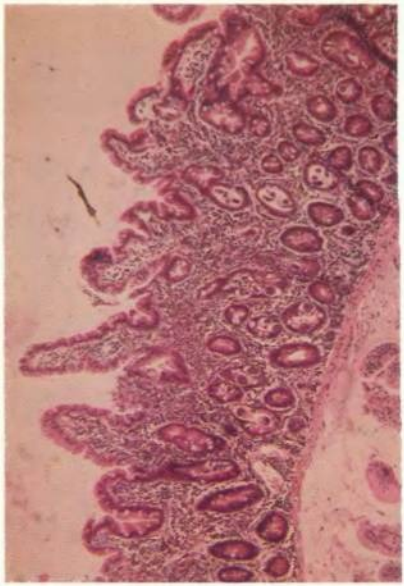


326

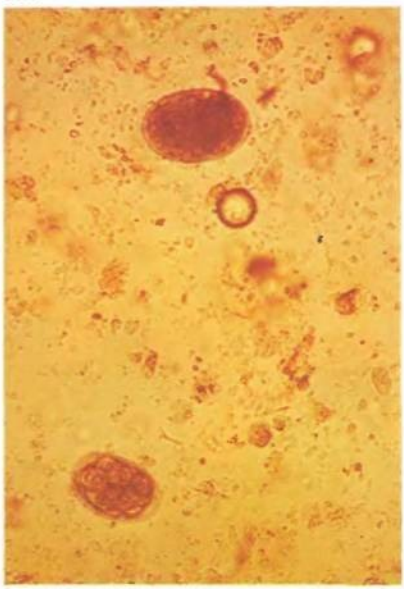
Ternidens infection

327 Head capsule of adult *Ternidens deminutus* In some areas such as East Africa and Rhodesia infection commonly occurs with a hookworm-like parasite of monkeys, *Ternidens deminutus* which infects also monkeys in Asia. The parasite is found at any level in the small and large intestine and normally infection is asymptomatic. *(× 60)*

328 Eggs of *T. deminutus* and hookworm compared The eggs are usually mistaken for those of hookworm, but the adults passed accidentally during hookworm therapy are distinctive (*see also* **301**) (*see* **455–461** for *Angiostrongylus*). *(× 125)*

327

328



INFECTION WITH *ASCARIS* & RELATED NEMATODES

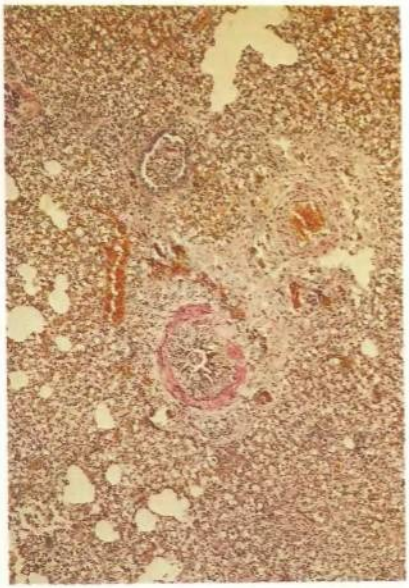
Ascariasis

329 ***Ascaris lumbricoides*** **larvae migrating in lung** Eggs (*see* **297**, **298**) passed in the faeces mature in the soil and are ingested together with contaminated food. Indiscriminate defaecation and the consequent distribution of eggs on the soil ensure the continuity of transmission of ascariasis. The prevalence of this worm in the community is a good indication of the standard of personal hygiene and sanitation. The eggs can survive even in a cold climate, and the infection is therefore global in distribution.

The eggs hatch in the human duodenum. The rhabditiform larvae penetrate the mucosa to enter the blood stream, and hence reach the lungs. Here they grow through two larval moults then, like hookworms, they break through into the bronchioles, make their way to the pharynx and are swallowed. The figure shows a section of larva in a bronchiole. This stage is associated with eosinophilia, and pneumonitis may accompany heavy infestations. (*see also* **773**)

330 Head of adult This scanning electron micrograph shows the typical head structures. *(× 110)*

329



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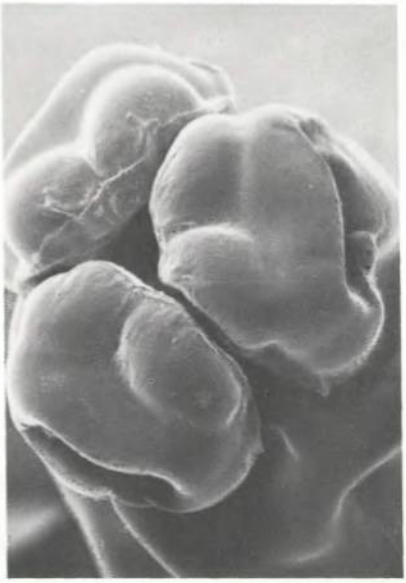


331 Adult *Ascaris* They mature into adult roundworms in the small intestine. The adult males are about 15 to 30 cm long, and females 20 to 35 cm.

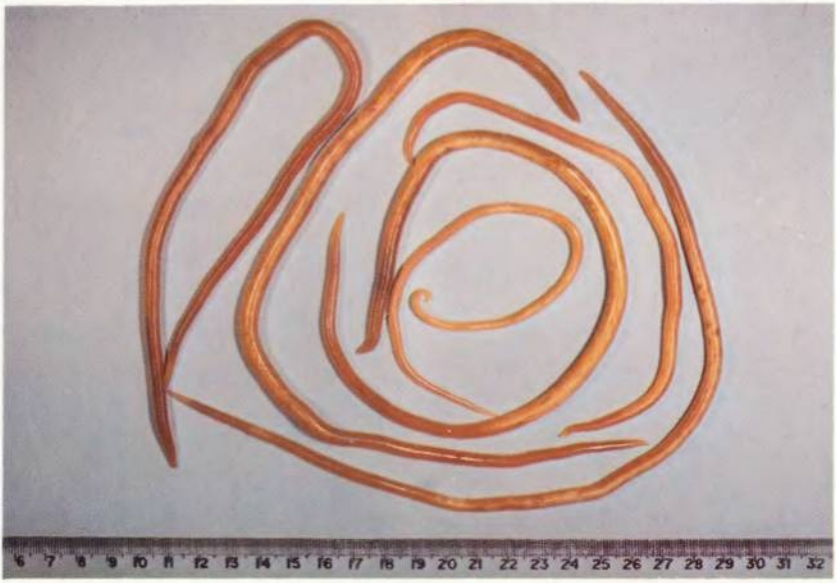
332 *A. lumbricoides* seen in X-ray The adults may be seen as filling defects in patients having barium meals for investigation of intestinal symptoms.

333 Obstruction due to roundworms Heavy infections, especially in children, may lead to intestinal obstruction. Volvulus is an additional complication in this intestine from a two-year-old child.

334 Adult roundworms migrating in liver The adult worms have a marked tendency to penetrate any available hole in their vicinity and may escape through abdominal fistulae following operations such as appendicectomy. They may also block such organs as the common bile duct, and the appendix itself.

335 Massive *Ascaris* infection in child A large bolus of roundworms expelled following anthelminthic treatment.

331



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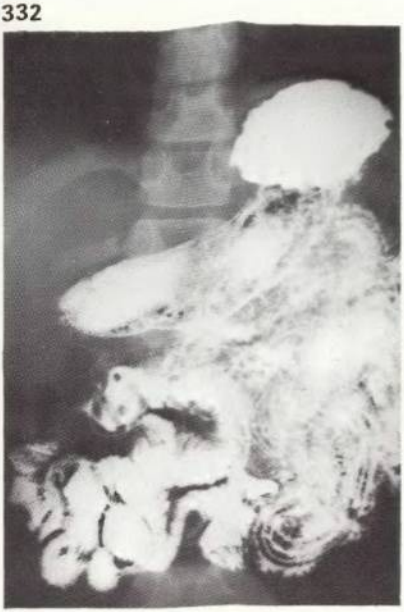

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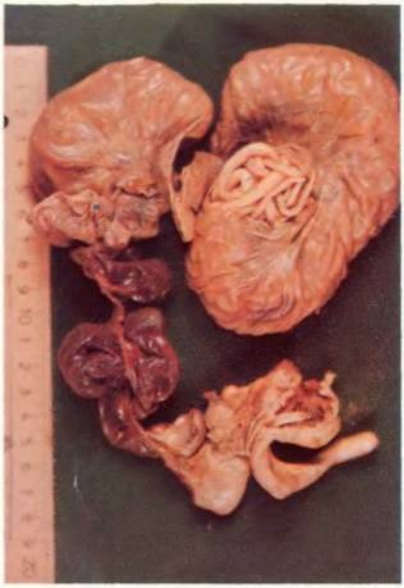

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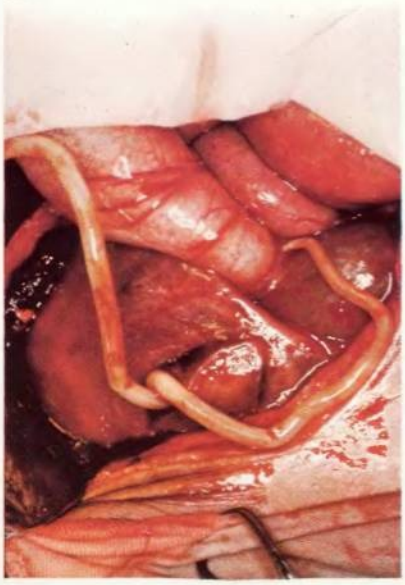

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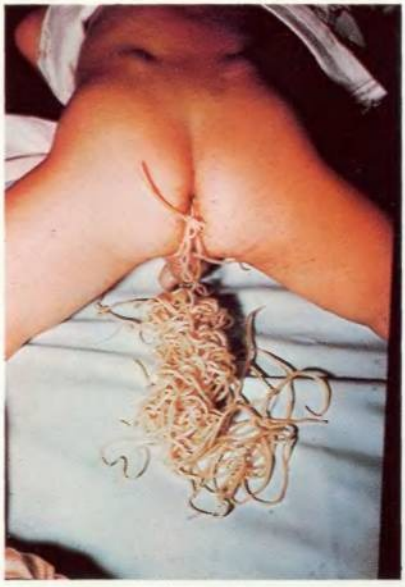


Toxocariasis

336 Adult *Toxocara canis* Visceral larva migrans results from accidental infection of man with eggs of the ascarid roundworm of the dog *Toxocara canis*, and cat *T. cati*. The adults are about 10 cm long and similar in general appearance to *A. lumbricoides*. *(× ⅓)*. Uncontrolled contamination of soil by dogs and cats is common everywhere. Like those of *Ascaris* the eggs of *Toxocara canis* and *T. cati* can survive even in a cold climate.

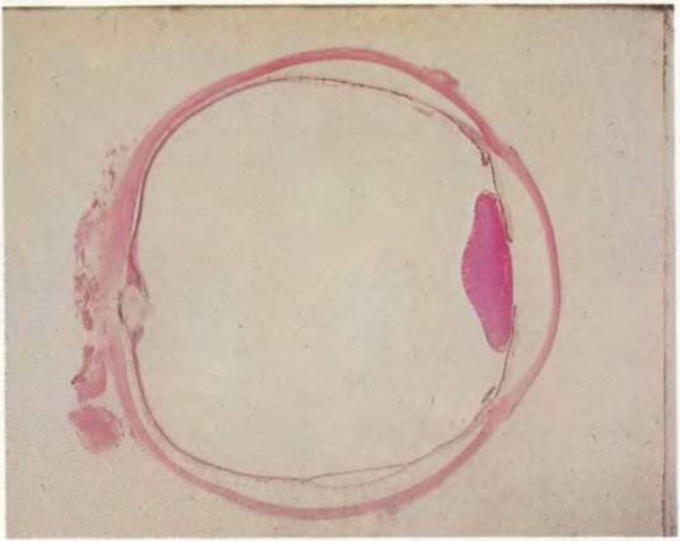
337 Longitudinal section of human eye The life cycle in the animal host is the same as that of *Ascaris* but the invasive larvae in man become arrested in various tissues where they are gradually phagocytosed. In the process they induce marked eosinophilia and local tissue reaction. Invasion of the eye produces a retinoblastoma-like appearance, and may lead to blindness. Sometimes the eye is mistakenly enucleated.

338 Section of *T. canis* larvae in human eye The larva is seen in the centre of the tumour-like mass at the posterior part of the eye (*see* **337**). *(× 20)*

339 Migrating larvae in liver Indefinite symptoms of hepatic involvement associated with marked eosinophilia should lead to a suspicion of visceral larva migrans as the causative agent. *(× 150)*

336

337



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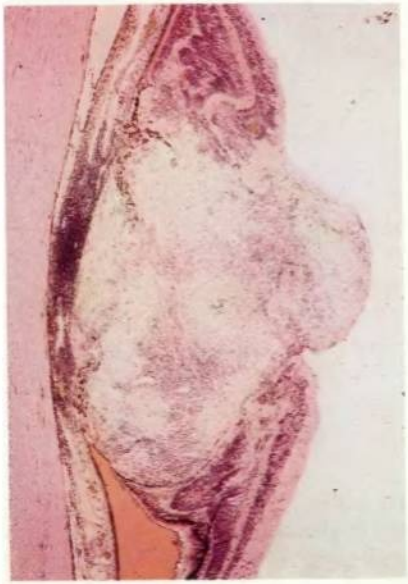

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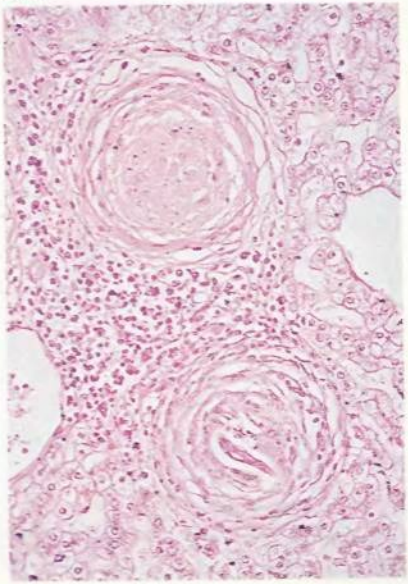

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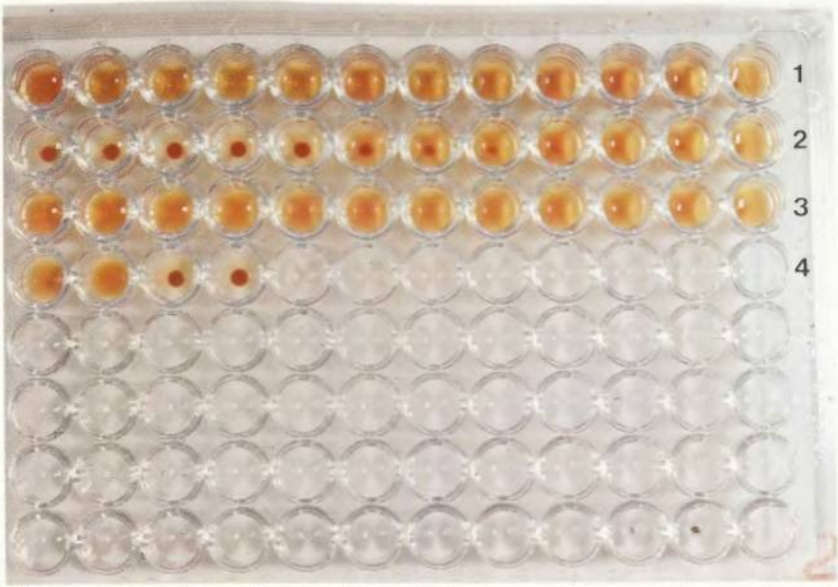

340 Serological diagnosis The diagnosis is supported by a positive serological response to *Toxocara* antigen such as the CFT. (Top row is a negative control serum; second row – positive response at 1/64 from a patient with visceral larva migrans; third row – negative serum from another patient with suggestive symptoms, fourth row – complement control.)

TRICHURIASIS

Trichuris trichiura has a direct life cycle. Eggs passed in the faeces are swallowed with soil-contaminated food. The eggs hatch into larvae which penetrate the villi of the small intestine. After three to 10 days the young worms pass down to the caecum where the whip-like anterior portion becomes entwined in the mucosa.

341 & 342 Adult morphology, females and males The adult worms are about three to five cm long, the females being slightly larger than the males which are coiled. (*see also* **774**)

343 Whipworms *in situ* The adult worms are readily seen in this figure of the caecal mucosa.

344 Rectal prolapse Heavy infections in infants and young children may cause rectal prolapse following chronic diarrhoea with abdominal pain.

(For capillariasis *see* **546–550**.)

341

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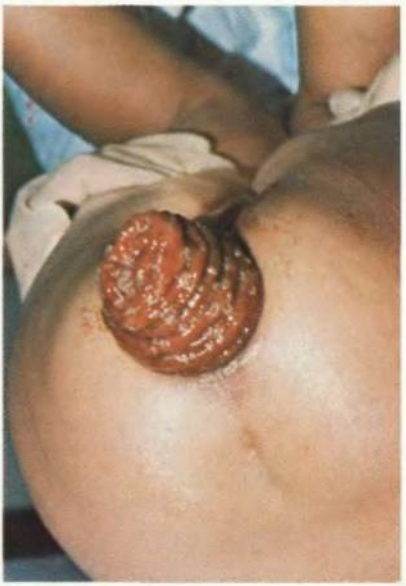

Part III
Snail-mediated Helminthiases

With the exception of angiostrongyliasis which is a nematode infection transmitted accidentally to man in the course of its complex life cycle in rodents, the important group of snail-transmitted helminths that infect man are all trematodes (flukes) which undergo a complicated cycle involving various species of land or aquatic snails. The three common schistosome infections of man are responsible for a vast amount of general ill health and contribute in certain areas to the heavy mortality rate found among adolescents and young adults. Since infection occurs by penetration of the skin by water-dwelling infective stages (cercariae) transmission tends to increase in parallel with the increase of land utilisation by irrigation. Hence schistosomiasis is a positive obstacle in the way of agricultural and economic development in many parts of the developing world.

The intestinal liver and lung flukes have a somewhat more complicated life cycle in that the cercariae develop a resting stage (the metacercaria) which, in turn, must be ingested by man in order to infect him. As the metacercariae are found usually on various water plants, on fish or on crustaceans, infection with these worms tends to be localised to areas where particular local eating habits bring man and the metacercaria together. Thus clonorchiasis is restricted to parts of Southeast Asia where raw fish is commonly ingested, fasciolopsiasis where, for example, water caltrops and water chestnuts are eaten uncooked, and paragonimiasis where the appropriate crustaceans form part of man's diet in one culinary delicacy or another. Many of these flukes are primarily parasites of animals other than man, eg *Fasciola hepatica* is essentially a parasite of sheep that infects man when he eats watercress or other aquatic plants from contaminated, wet pastures. They are thus zoonoses and, like many zoonoses, are relatively uncommon with notable, highly localised exceptions. In certain villages in northeast Thailand, for example, *Fasciolopsis buski* has been found to infect nearly 100% of the population and *Opisthorchis viverrini* 90%. An idea of the numbers of human infections with these trematodes is given in Table X.

SCHISTOSOMIASIS

The schistosomes provide a classical example of the life cycle of trematodes. Eggs passed in the faeces or urine hatch in water and the emerging miracidia invade susceptible snails. After reproduction through two generations of sporocysts, cercariae develop. These emerge from the snails and penetrate the skin of new human hosts who pass through the water. With the loss of the cercarial tail during penetration the parasite becomes a schistosomule which migrates actively through the body until it reaches the portal venous system of the liver. After copulating, the female lays eggs either in the vessels of the mesenteric plexus surrounding the large intestine (*S. mansoni*, *S. japonicum* and *S. intercalatum*), or the venous plexus of the bladder (*S. haematobium*). Spiny eggs penetrate the mucosa of these organs and can thus reach the exterior in faeces or urine. The use of molluscicides to control snail breeding, combined with mass chemotherapy is one of the best available means of combating schistosomiasis.

345 Distribution of *S. mansoni* The distribution of schistosomiasis is regulated both by the presence of susceptible snail intermediate hosts and human sanitary habits. *S. mansoni* occurs in Africa, the Middle East and in parts of South America. It was introduced from the Old World into the New World where potentially susceptible species of *Biomphalaria* were already present.

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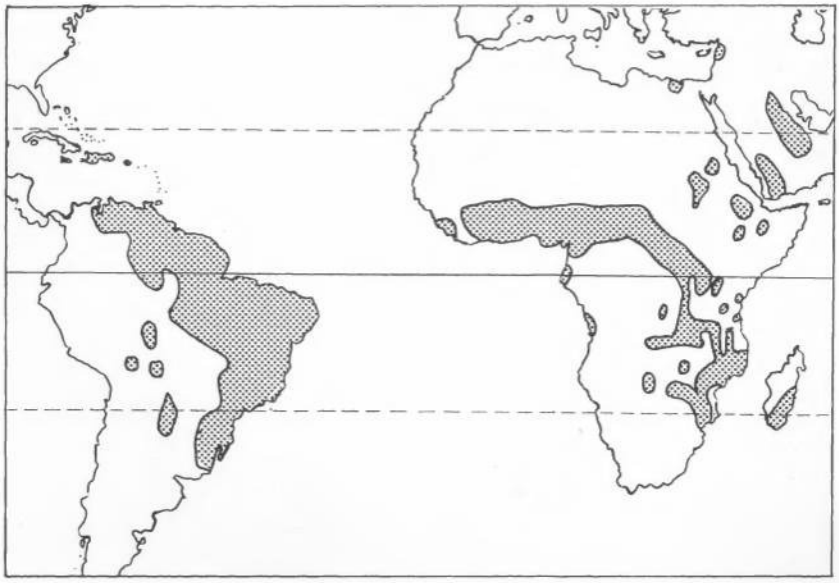

346 Distribution of *S. haematobium* and *S. japonicum*. *S. haematobium* is found in Africa and the Middle East. *S. japonicum* is endemic in the Far East, South East Asia and the Philippines.

S. haematobium

S. japonicum

347 Miracidium in egg of *S. mansoni* The three common species of schistosomes infecting man have easily recognisable eggs although those of *S. haematobium* may be confused with *S. intercalatum* (**397**). Miracidia can be seen inside mature eggs. *(× 600)* (*see also* **775**)

348 Living miracidium of *S. mansoni* hatching These may readily be seen by immersing eggs separated from faeces in a large quantity of fresh water in which they hatch. *(× 600)*

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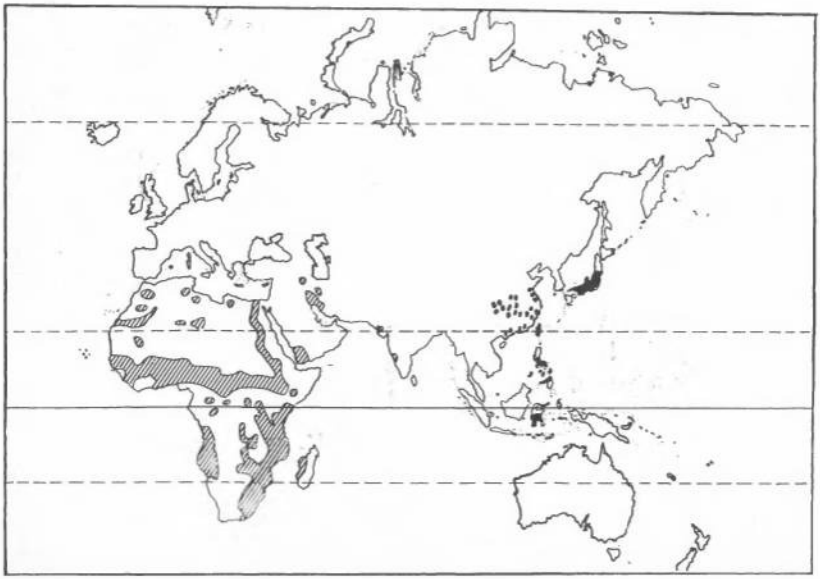

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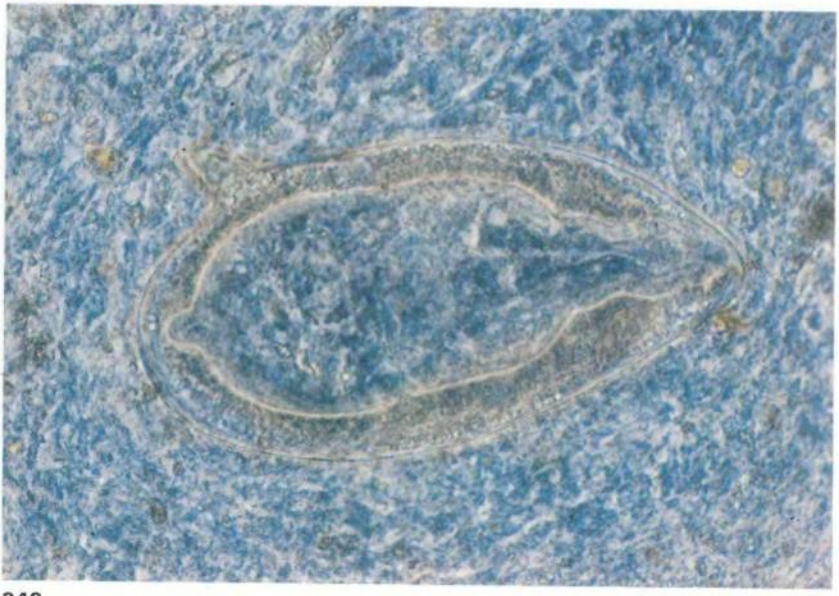

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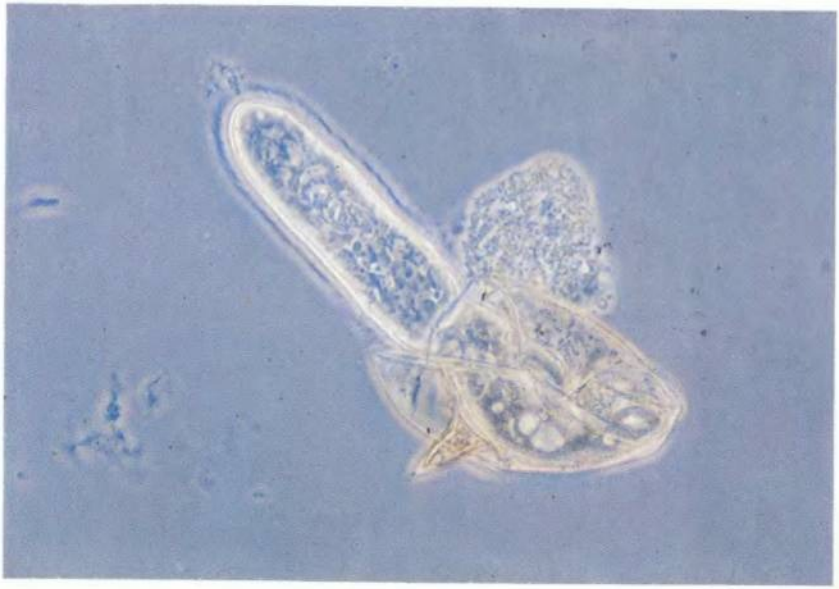

349 Miracidium free in water. *(× 600)*

350 Apical papilla of miracidium This scanning electron micrograph shows the surface locomotor cilia and apical papilla with its 'mini-sucker' which helps it attach to a snail. *(× 3300)*

351 'Mother' sporocyst in the hepatopancreas of a snail The cycle in the snail is of variable duration depending on the species of parasite, host and environmental conditions, but it is usually only one month. Cercaria develop in the second generation ('daughter') sporocysts. The figure shows on the top several coils of the sporocyst dissected from the hepatopancreas. One of the cercaria has broken out and is seen at the bottom of the figure. *(× 60)*

352 Living bifurcate cercaria of *S. mansoni* Once snails start to 'shed' cercariae they continue to do so during daylight hours for up to as much as 200 days. A heavily infected snail may shed 1500 to 2000 cercariae a day. *(× 350)*

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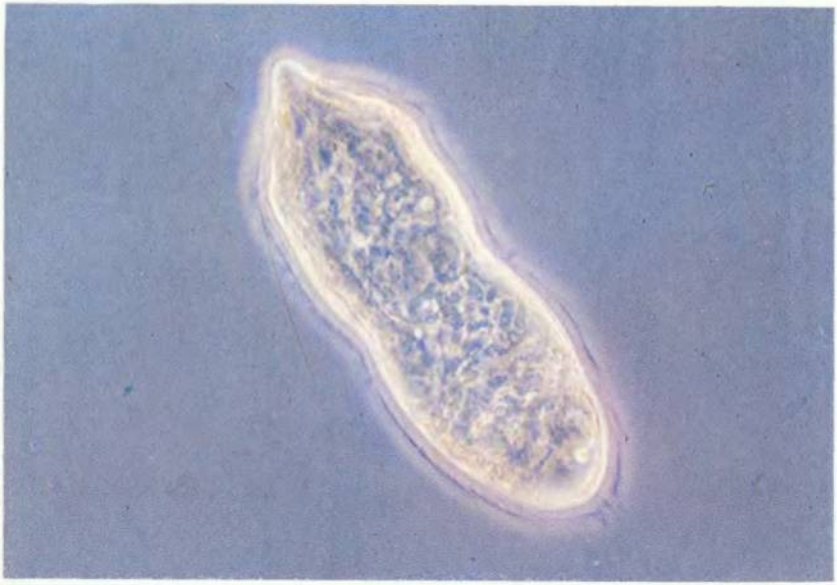

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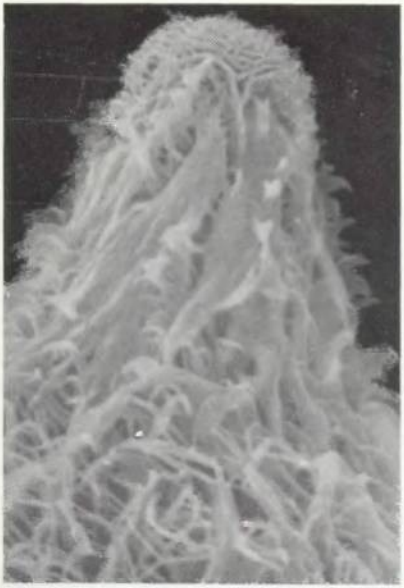

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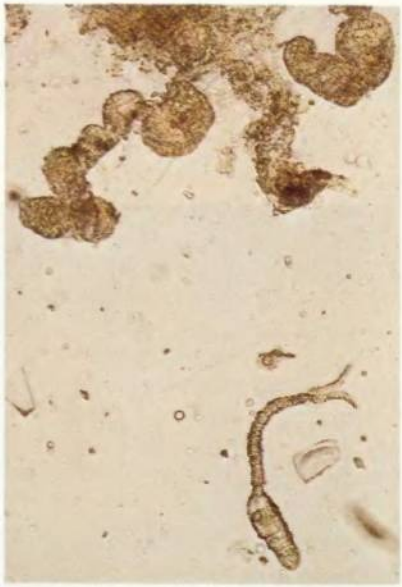

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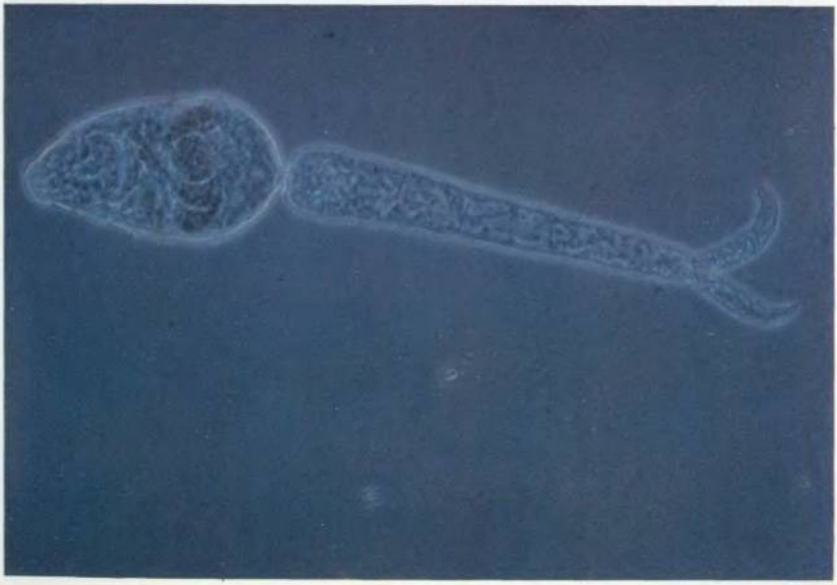
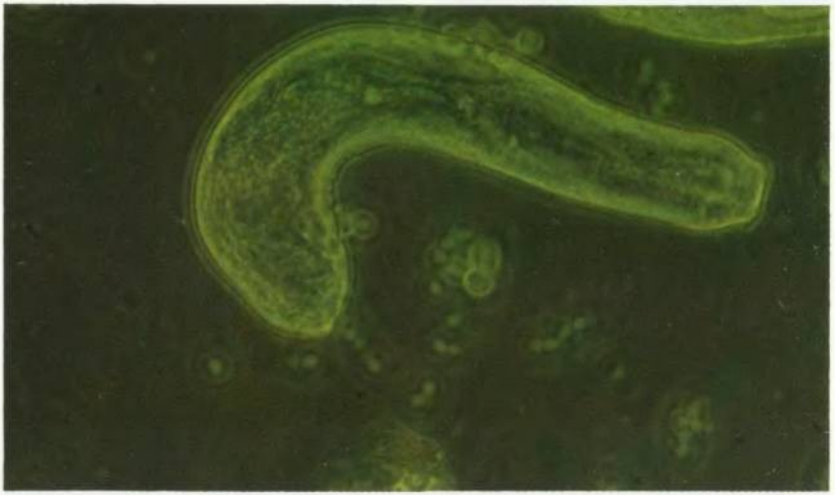


353 Schistosomule of *S. mansoni* *(× 500)*

354 Living male and female *S. mansoni* in mesenteric vein of mouse The slender female is seen within the gynecophoral groove of the male. *(× 4½)*

353



354

355–360 Morphology of adults Mature males and females live in copulating pairs. The common species are recognised by the characters shown in the figures. Testes of males: *S. haematobium* (**355**); *S. mansoni* (**356**); *S. japonicum* (**357**). Ovaries of females: *S. haematobium* (**358**); *S. mansoni* (**359**); *S. japonicum* (**360**). *(× 15)*

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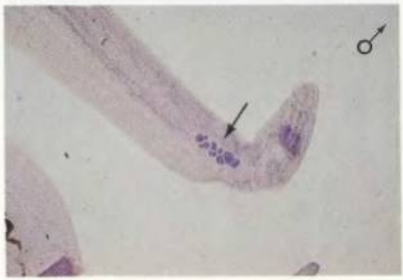

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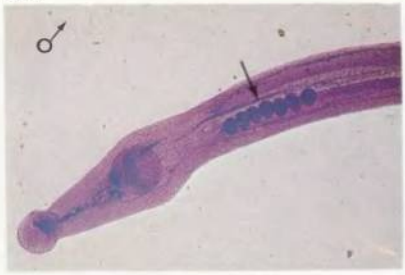

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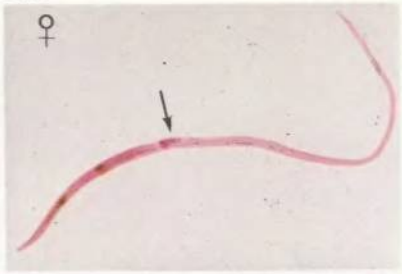

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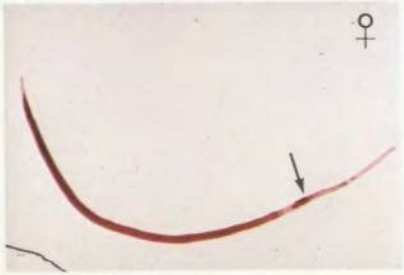

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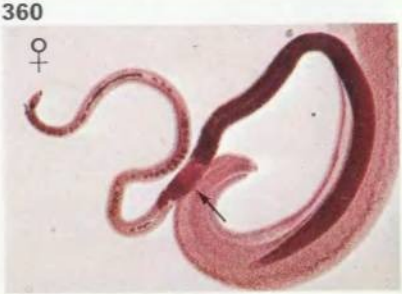

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361–363 Living intermediate hosts of schistosomes The snail intermediate hosts of *S. mansoni* are the species of *Biomphalaria* (**361**). Various species of *Bulinus* (**362**) are hosts of *S. haematobium* through its range. *Oncomelania* species (**363**) are hosts for *S. japonicum* in the Far East. Three examples are shown in the figure (*see also* **Table XI**). *(× 4)*

364 Sampling snail populations A scoopful of *Biomphalaria sudanica*, a host for *S. mansoni* in Lake Victoria.

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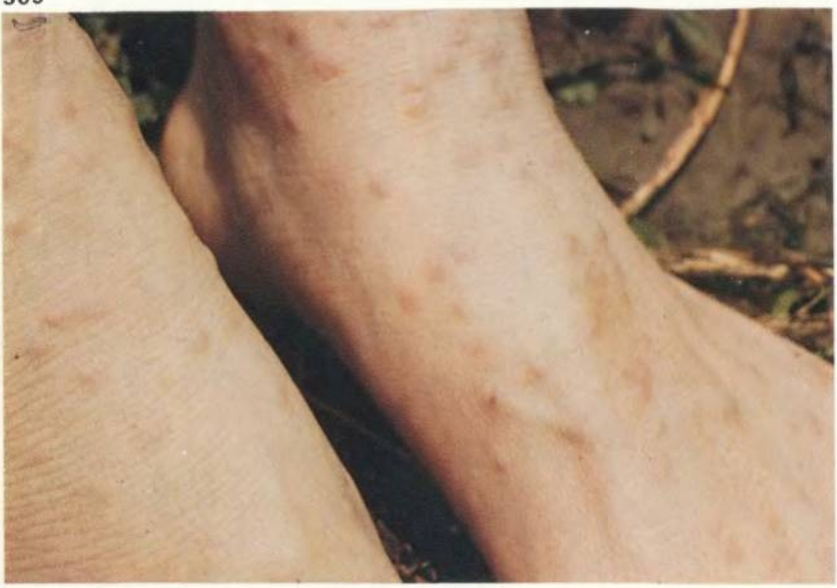

365 Dermatitis from avian cercariae in a Japanese patient Penetration of the skin by cercariae may give rise to an itchy rash known as 'cercarial dermatitis'. This is occasionally seen in countries free of human schistosomiasis due to invasion by the cercariae of avian schistosomes which are otherwise non-pathogenic to man.

Schistosoma mansoni

366 Adult *S. mansoni* in portal tract Male and female schistosomes lodge *in copula* in the portal tract, mesenteric or vesical plexuses. The figure shows a cross-section of a male and female *S. mansoni* in a branch of the portal vein. *(× 40)*

367 Granuloma replacing egg of *S. mansoni* in liver Eggs **(294)** may lodge ectopically in any tissues, where they cause characteristic granulomas. It has been suggested that toxic substances associated with the ova trigger the fibrotic process. In histological sections the ova are seen in the portal and periportal regions. All types of reaction may be present from acute eosinophilic cellular infiltration to the dense collagenous deposition which leads to periportal fibrosis. *(× 40)*

368 Periportal fibrosis of the liver Periportal fibrosis ('pipestem fibrosis') is the classical pathological hepatic lesion. The white areas which may be round, oval, or stellate, are due to the terminal fibrotic reaction originally caused by the presence of the ova in and around the portal venous radicles.

369 Egyptian splenomegaly The combination of enlarged, irregularly fibrosed liver, and greatly enlarged spleen is commonly called 'Egyptian hepatosplenomegaly'.

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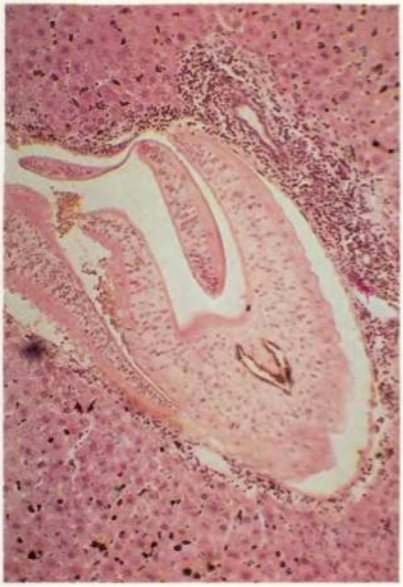

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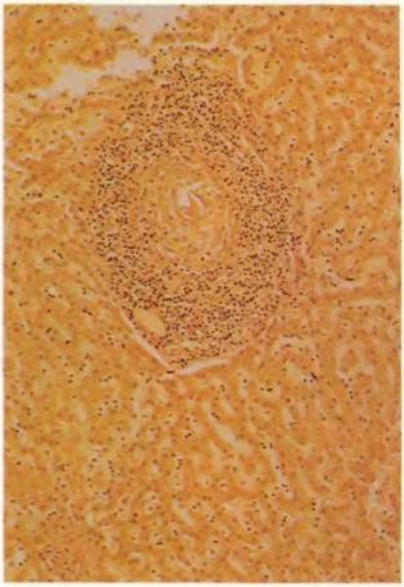

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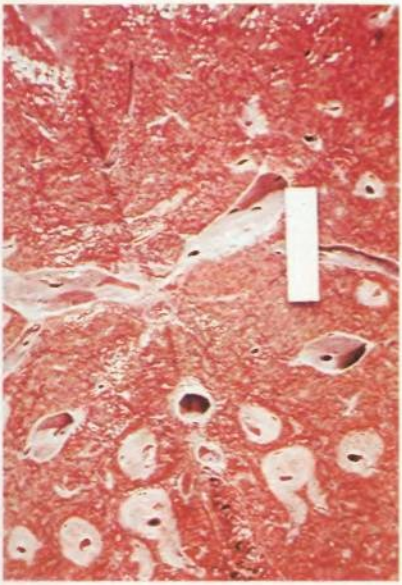

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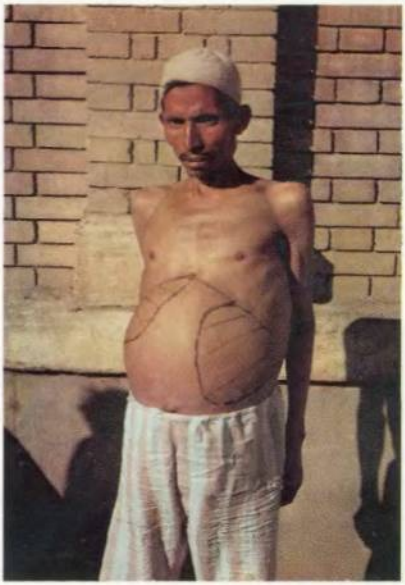

370 Ascites secondary to chronic portal hypertension in a Brazilian The classical clinical feature of chronic *S. mansoni* infection is portal hypertension. The opening up of a secondary, circulatory shunt leads to the development of varices in the oesophageal and gastric veins, ascites and gross splenomegaly.

371 Petechial haemorrhages In children portal venous obstruction secondary to schistosomiasis may present as telangiectasis-like spots, as in this Brazilian child.

372 X-ray of oesophageal varices Oesophageal varices like those shown on this X-ray can rupture, leading to a fatal haematemesis.

373 X-ray of colonic polyposis In the early stages of *S. mansoni* infection diarrhoea is a common complaint. Extensive polyposis of the colon sometimes occurs. This lesion is reversible with antischistosomal drug therapy.

374 & 375 Sigmoidoscopic view of colonic polyps Four views of polyps in the descending colon.

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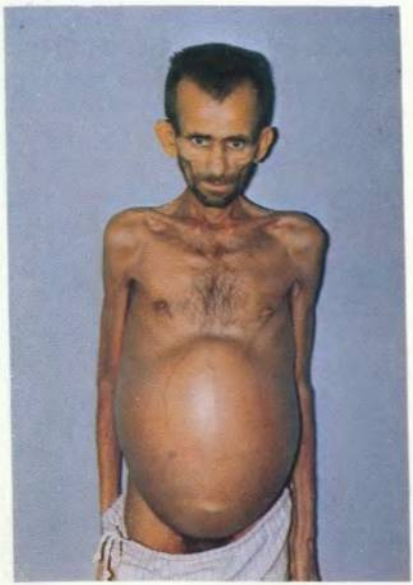

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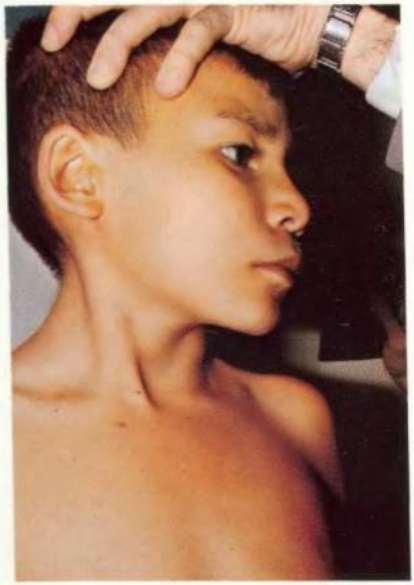

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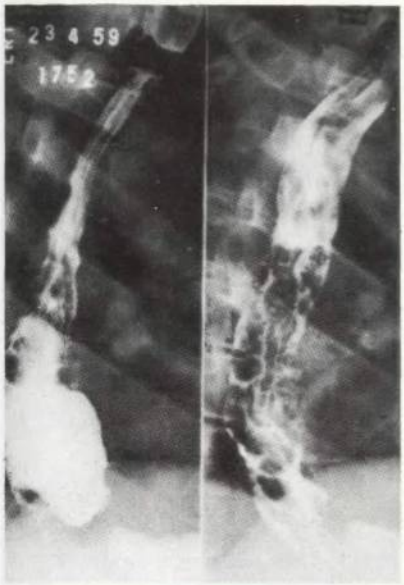

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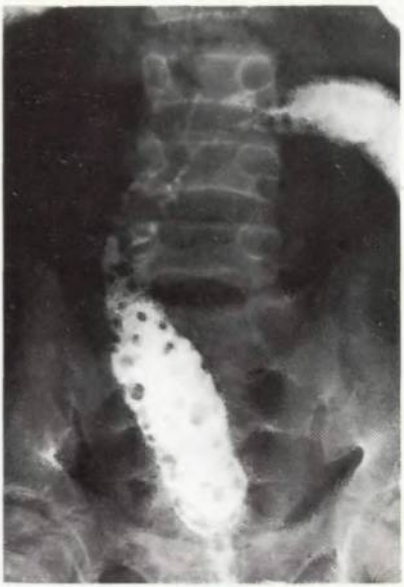

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376 Polyposis of colon at post mortem Massive polyposis of the colon with fatal intestinal haemorrhage occurred in this Egyptian farmer.

377 Biopsy showing *S. mansoni* eggs in polyposis of colon Diagnosis is usually confirmed by demonstrating eggs of *S. mansoni* in the stool. However intestinal biopsy through a proctoscope or sigmoidoscope is also an effective means of finding eggs and establishing a definitive diagnosis of *S. mansoni* infection. It may also be positive in patients with *S. haematobium* or *S. japonicum*. *(× 60)*

378 Ectopic infection – *S. mansoni* infection of lung When the lungs are affected typical eggs may appear in the sputum. Ectopic lesions in the spinal cord result in a transverse myelitis and paralysis. The lesions usually contain large numbers of ova in necrotic material surrounded by eosinophils, and multi-nucleated giant cells. The CSF shows an increase in protein and cells, especially eosinophils. *(× 60)*

379 Cor pulmonale Eggs may reach the lungs and other ectopic sites by metastatic blood spread. An end result of the periarteritis caused by the presence of eggs in the lungs is fibrosis of the pulmonary arterioles with pulmonary hypertension. This results in enlargement of the right heart, ie cor pulmonale.

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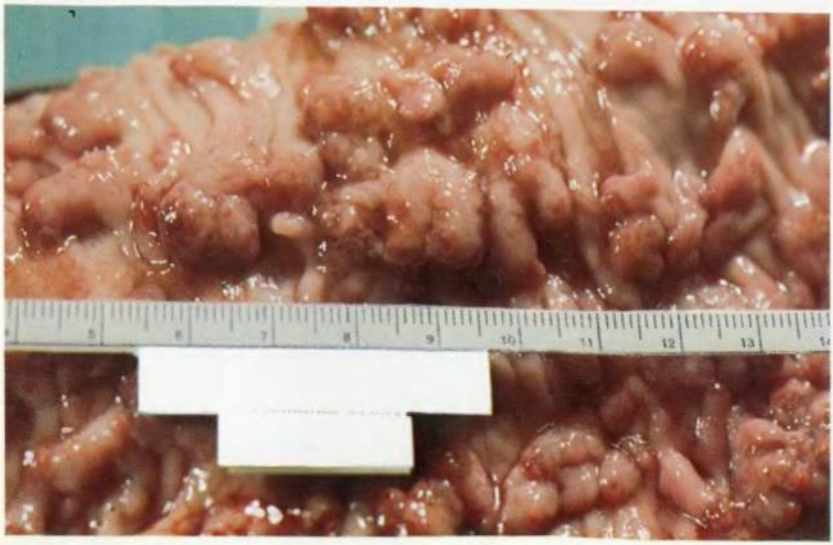

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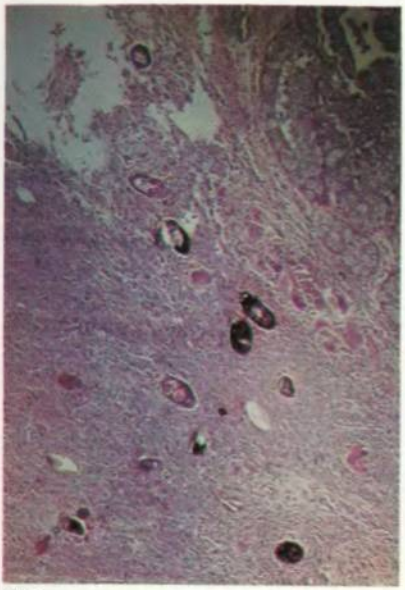

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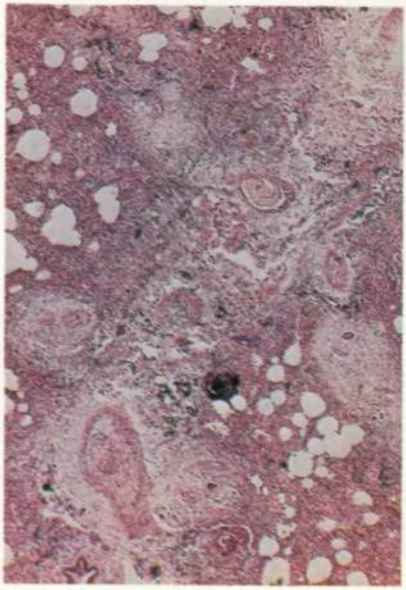

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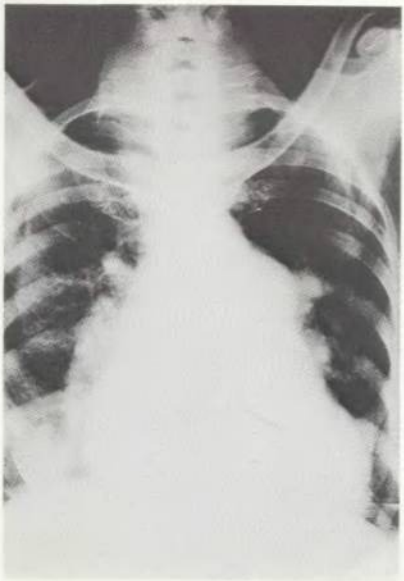

380 Ecology of *S. haematobium* infection Increased land use through the development of irrigation projects as in Egypt and the Sudan may result in an increasing incidence of *S. haematobium* transmission through *Bulinus* snails breeding in the irrigation canals. In this village 62% of children from two to six years were infected.

381 Haematuria Haematuria, often at the end of urination, is a characteristic early clinical feature of infection with this parasite. Typical terminal spined eggs of *S. haematobium* (*see* **293**) may be found in the centrifuge deposit.

382 Eggs in section of bladder Schistosome ova laid by female worms in the vesical plexus are retained in the vesical tissues and later become calcified. *(× 20)*

383 X-ray of bladder showing filling defects due to large nodules Active proliferating papillomatous or granulomatous lesions are responsible for the bladder-filling defects seen radiologically in the early stages of the infection.

380

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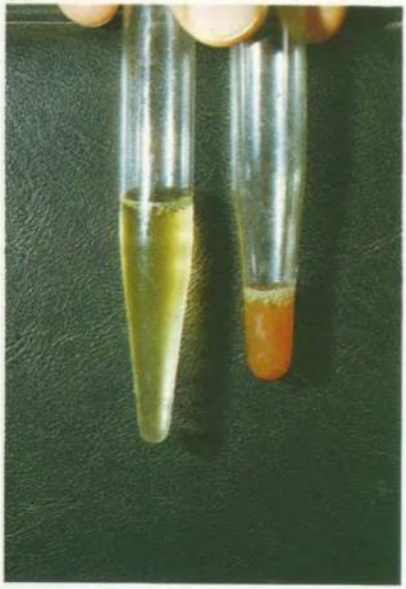

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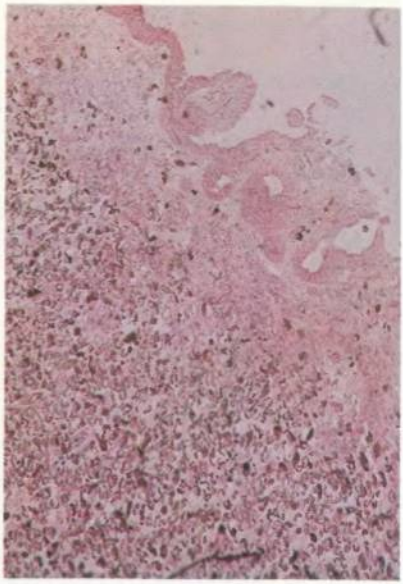

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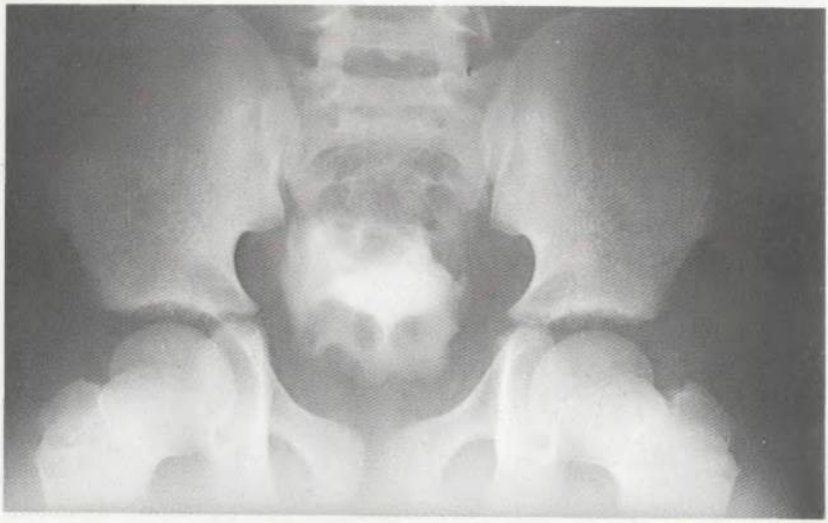

384 Nodules due to *S. haematobium* The appearance of a nodular lesion in the vesical wall as seen at open operation is well shown here.

385 X-ray of bladder with calcification Widespread fibrosis and eventually calcification of the bladder wall result in this 'fetal-head' appearance.

386 X-ray of dilated ureters Gross tortuosity and dilatation of the ureters result from stenosis of the ureteric orifices due to calcification.

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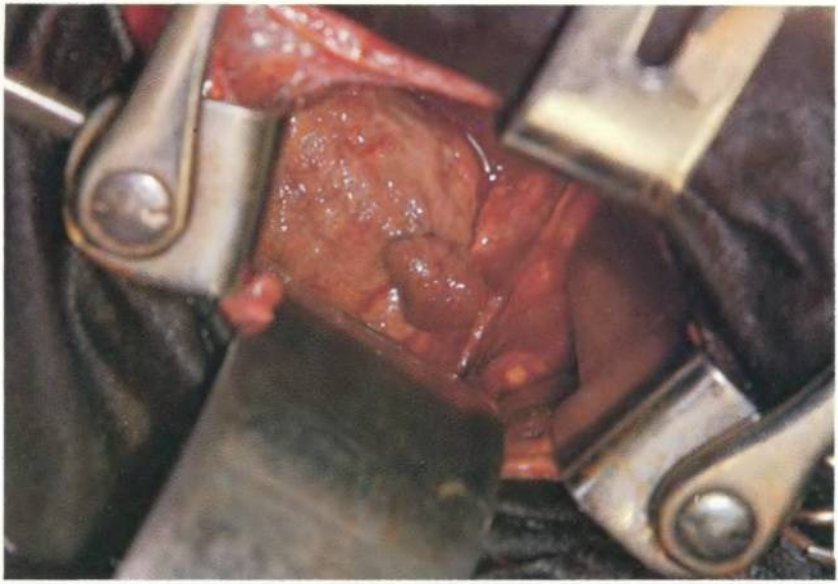

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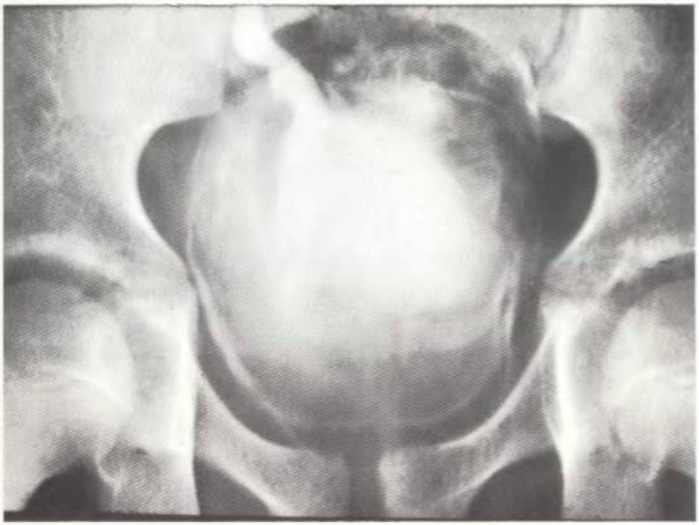

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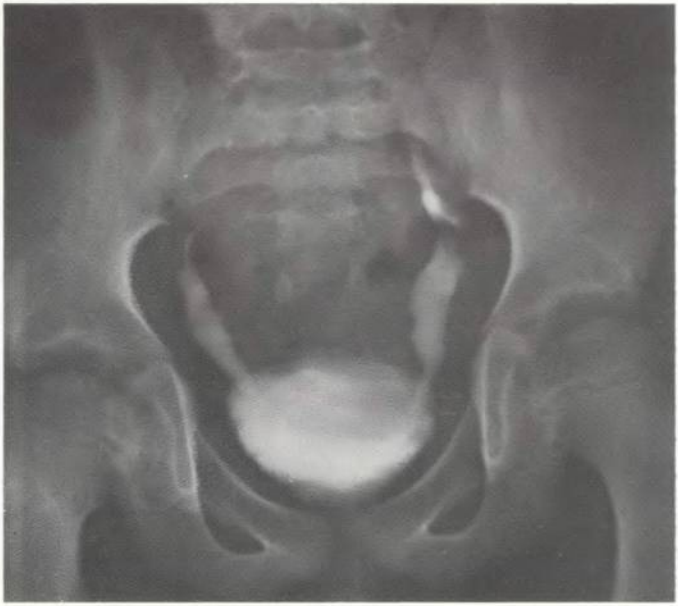


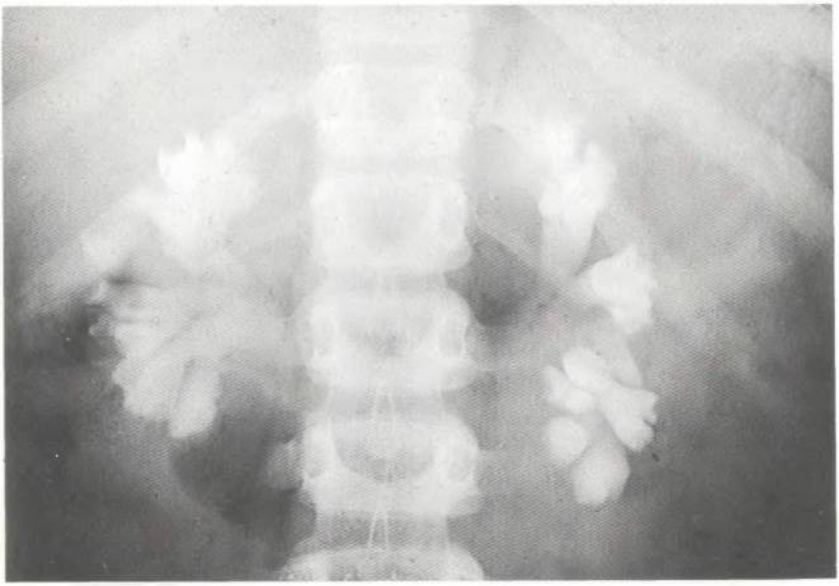
387 X-ray of kidney showing bilateral hydronephrosis Unilateral and bilateral hydronephrosis due to vesical and ureteric destructive lesions are not uncommon in haematobium infection.

388 Persistent hydronephrosis in child Hydronephrosis due to *S. haematobium* infection in children is often reversible with adequate antischistosomal drug treatment. In this six-year-old the lesion persisted despite therapy.

389 Squamous cell carcinoma In areas where *S. haematobium* infection is intense, the incidence of vesical cancer is high. Squamous-cell carcinoma is the type most commonly found, and ova of *S. haematobium* are often present in such tumours. Adenocarcinoma also occurs. *(× 90)*

390 Adenocarcinoma of bladder showing large numbers of eggs. *(× 20)*

387



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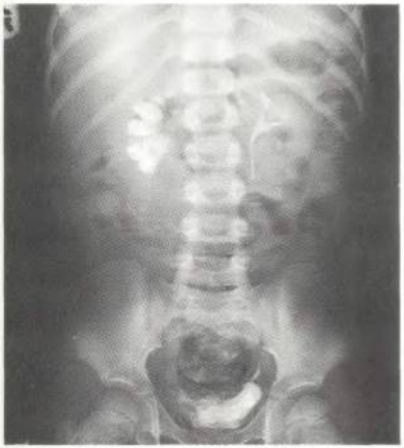

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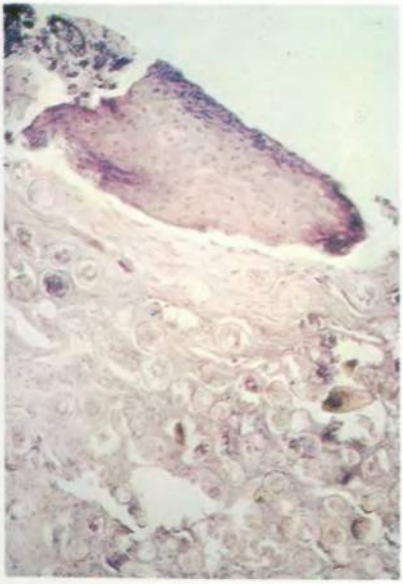

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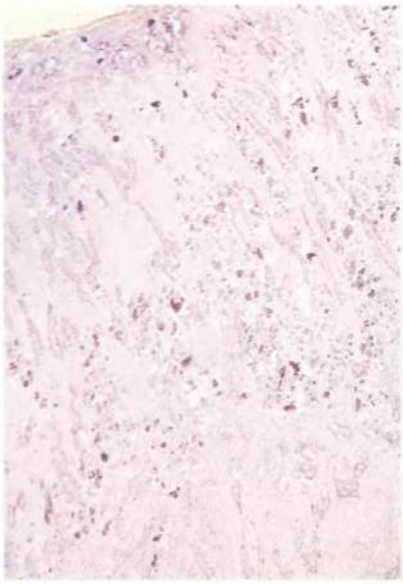

Schistosoma japonicum

S. japonicum infection is a zoonosis. It is found in a wide variety of vertebrate hosts, including domestic animals and bovines. Human infection frequently occurs in farm workers.

391 Radio-isotope scan of patient with *S. japonicum* infection Severe hepatic fibrosis and massive splenomegaly occur in *S. japonicum* infection, in the same manner as in *S. mansoni* (*see* **369**), often with ascites. This Chinese patient was infected both with *S. japonicum* and *Clonorchis sinensis*.

392 Philippino boy with gross splenomegaly Advanced lesions due to *S. japonicum* may be seen in children and adolescents.

393 Ova of *S. japonicum* in temporal lobe of human brain Schistosomal lesions in the brain may contain large numbers of ova in necrotic material, often surrounded by eosinophils. (Little cellular reaction is seen in this figure.) *(× 150)*

394 Eggs of *S. japonicum* in wall of colon The adult worms do not invade the vesical plexus, but usually inhabit the mesenteric plexus. The diagnosis of *S. japonicum* can usually be made by finding typical eggs in the faeces. However, it is commoner for eggs of *S. japonicum* to be deposited in ectopic sites than those of other schistosome species. *(× 150)*

395 Intradermal test In any type of schistosomiasis the diagnosis may be suggested in the absence of demonstrable ova by the response to an intradermal injection of a suitable antigen. This figure shows a typical response to antigen prepared from *S. mansoni*.

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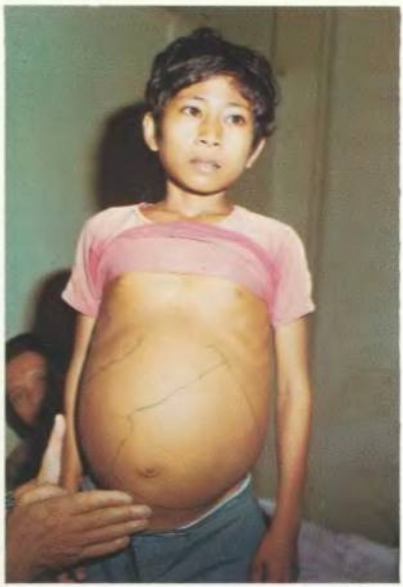

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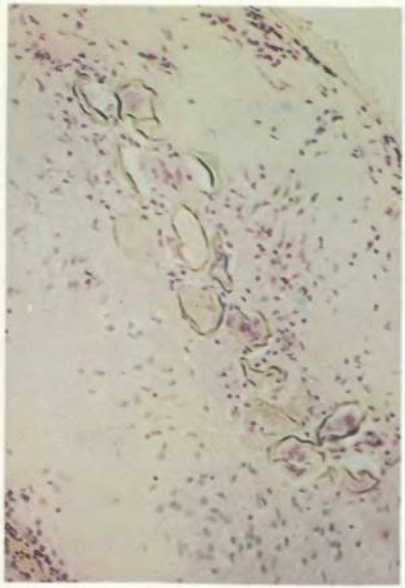

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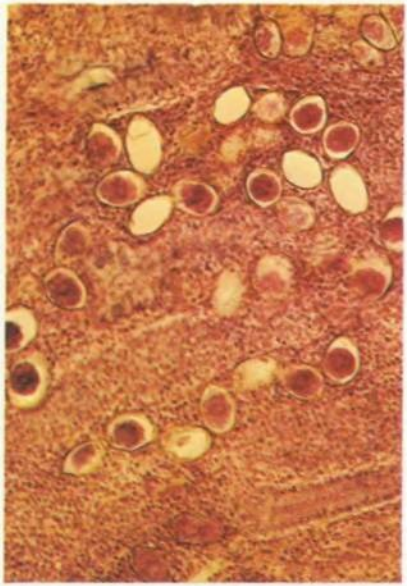

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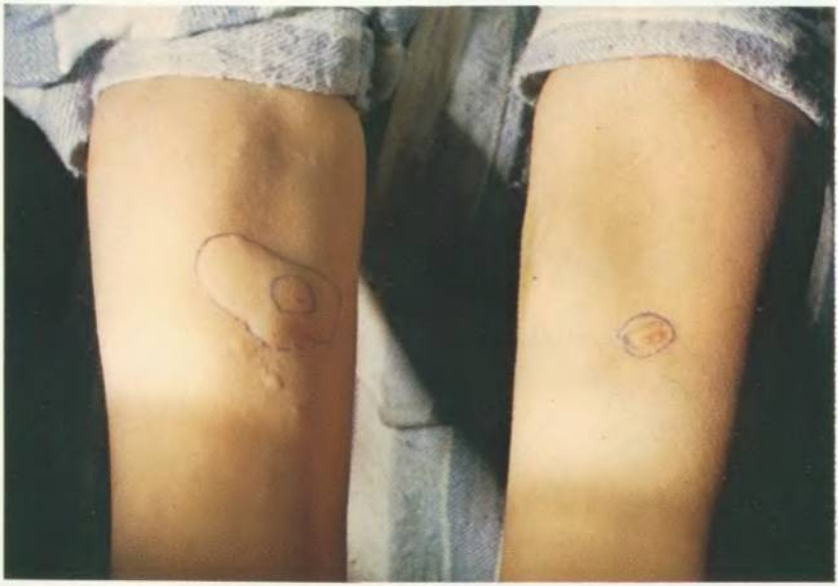

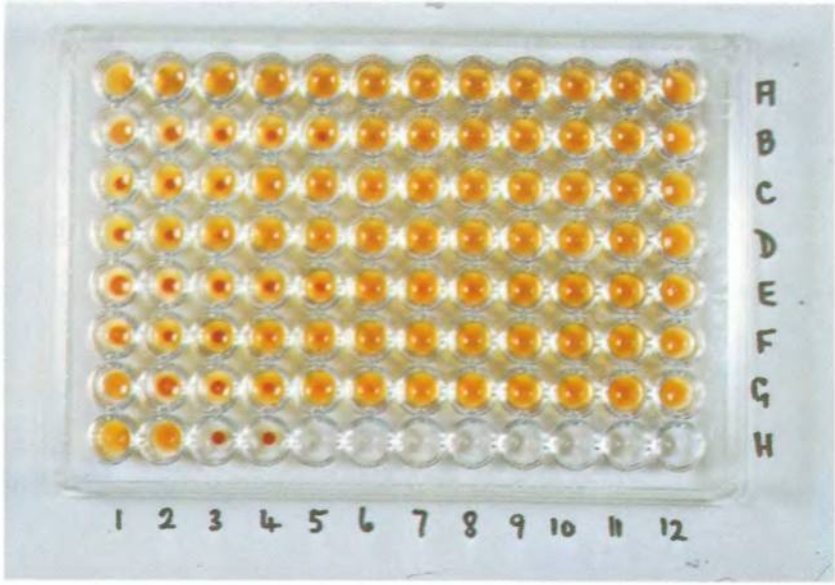

396 Complement fixation test Serological diagnosis is of particular value in epidemiological surveys. (Row A is a negative control serum. Positive reactions to *S. mansoni* from patients' sera are row B (1/25), C, E (1/32), F, G (1/16). H is complement control. D is considered negative at 1/8.)

Schistosomes Found Uncommonly in Man

397–399 Eggs of unusual schistosomes seen in man The eggs of *S. intercalatum* (**397**) are similar to those of *S. haematobium* but are found only in the faeces; *S. mattheei* (**398**) occurs in sheep and cattle. *S. rodhaini* (**399**) of dogs, felines and rodents may be mistaken for *S. mansoni*. *(× 250)*

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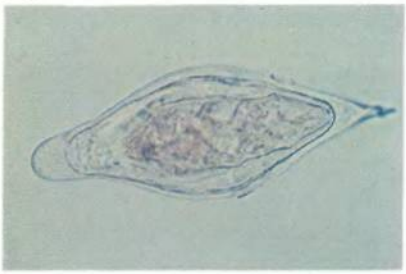

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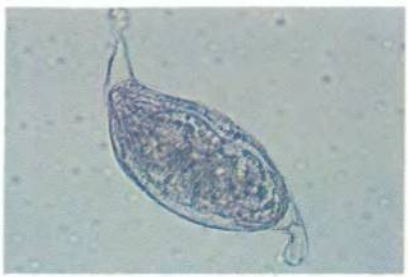

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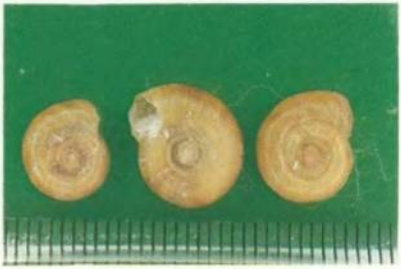

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THE INTESTINAL FLUKES*

400–407 The snail intermediate hosts of trematode infections of man A number of the molluscs listed in **Table XI** are figured here: *Oncomelania nosophora (× 3)* (**400**); *Thiara granifera (× 1½)* (**401**); *Biomphalaria glabrata (× 3)* (**402**); *Biomphalaria sudanica (× 1½)* (**403**); *Bulinus (Bulinus) senegalensis (× 3)* (**404**); *Bulinus (Physopsis) globosus (× 1½)* (**405**); *Segmentina* sp. *(× 3)* (**406**); *Lymnaea truncatula (× 3)* (**407**).

408 Comparative sizes of flukes From left to right: first row – *S. mansoni* ♂ and ♀ ; second row – *H. heterophyes*; third row – *O. felineus*, *C. sinensis*, *P. westermani*; fourth row – *F. hepatica* and *F. buski*.

*(*See* **Table XI**)

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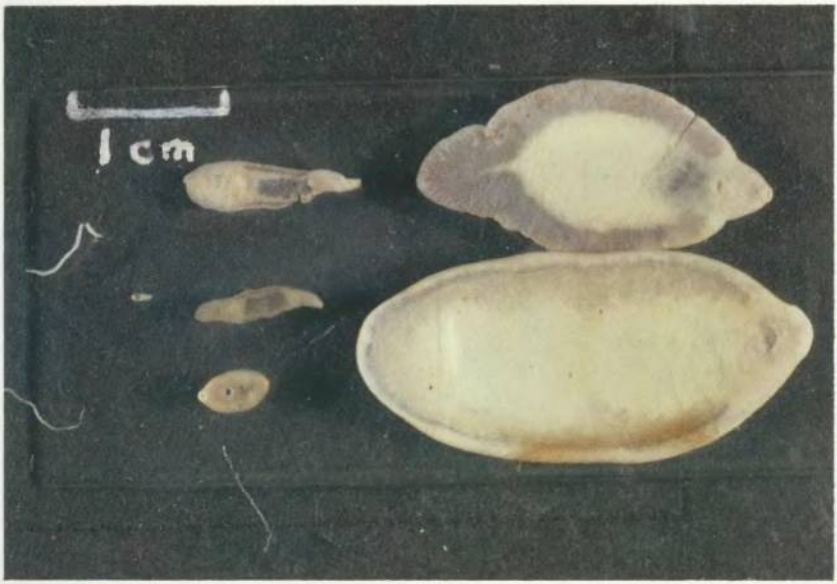


Fasciolopsiasis

409 Distribution of *Fasciolopsis buski* *F. buski* is limited to areas of the Far East. In certain localities, eg Northeast Thailand, almost the entire population of some villages may be infected.

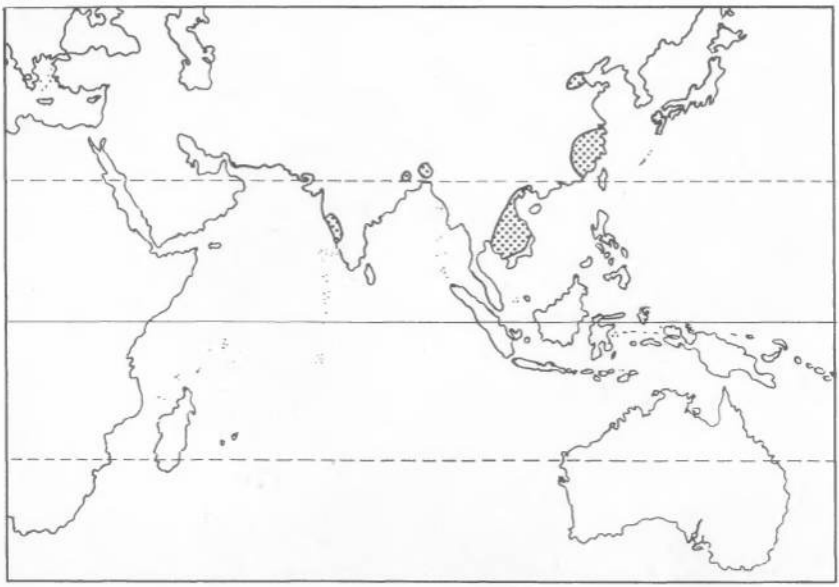
410 Egg of *F. buski* Miracidia hatching from eggs passed in the faeces after shedding their ciliated coats, invade snails of various genera including *Segmentina* and *Hippeutis*. The reproductive cycle in the snail differs from that of the schistosomes. After development through the sporocysts and two generations of rediae, cercaria emerge into the water. They then encyst on aquatic plants such as the water caltrop and water chestnut. *(× 200)*

411 Metacercaria of *F. buski* The metacercaria of *F. buski* with its cyst wall. *(× 200)*

412 Living *Segmentina hemisphaerula*.

413 Adult *F. buski* The adult *F. buski* is the largest parasitic trematode of man and may reach 7.5 cm in length. The pig is the main animal reservoir of infection for man. *(Natural size)*

409



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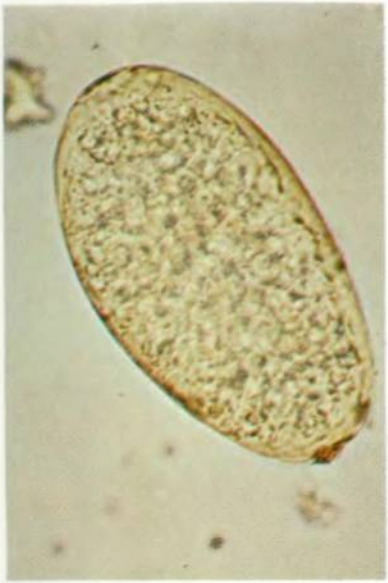

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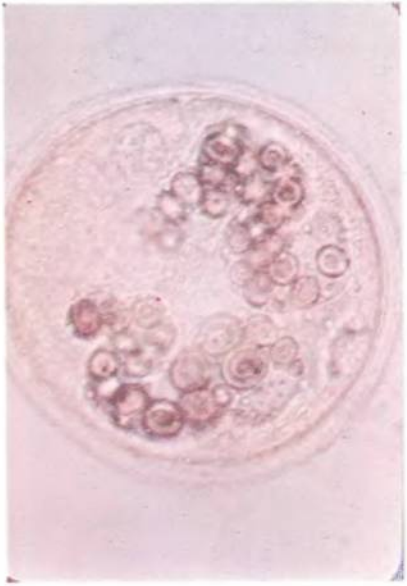

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414 The water caltrop 'Plantations' of water caltrop are harvested in endemic areas. The characteristic fruits of this plant are commonly eaten raw, and the metacercariae are thus swallowed. In the digestive tract they attach to the mucosa of the upper part of the small intestine where they mature. Water chestnuts commonly spread infection to children who peel the raw plants with their teeth, thus ingesting the attached metacercariae.

Uncommon Species

415 Adult *Heterophyes heterophyes* This is an uncommon but widely distributed trematode with a typical life cycle in brackish water snails such as *Pirenella* (*see* **Table XI**). The cercariae encyst on fish such as the mullet and infect man when improperly cooked fish is eaten. The adult trematode shown here is very small and lives in the middle part of the small intestine. The eggs (*see* **307**) are very similar to those of *Clonorchis* and *Metagonimus*. *(× 35)*

416 Adult *Metagonimus yokagawai* This is the most common heterophyid fluke of the Far East but is also found in the Mediterranean basin. The life cycle is similar to that of *Heterophyes* and the eggs of the two species can only be separated with difficulty. The adult worm shown here is also very small (1.4 × 0.6 mm) and lives in the upper and middle jejunum. Several genera of snails including *Semisulcospira* (*see* **Table XI**) are the first intermediate hosts for the species; the cercariae encyst on fish. *(× 35)*

417 Cyprinoid fish in an Eastern market The mullet and other fish living in fresh or brackish waters are common intermediate hosts for *H. heterophyes* and *M. yokagawai*. The metacercariae are attached under the scales or in the skin.

414

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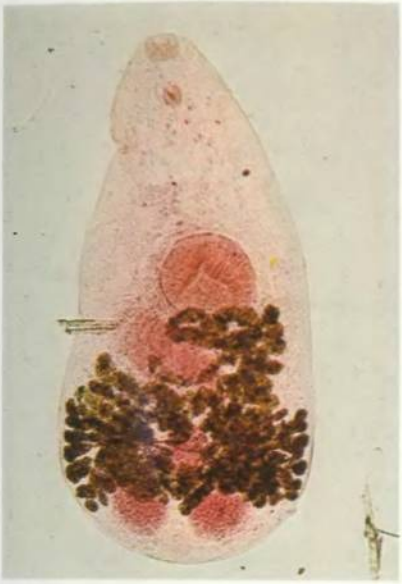

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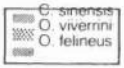
417

LIVER FLUKE INFECTIONS

*Clonorchis sinensis**

418 Distribution map *C. sinensis* is also known as the Chinese or oriental liver fluke. It is found in man and also in other fish-eating mammals in the areas shown (after Muller, 1975).

(*Now referred to as *Opisthorcis sinensis*)



419 *Ctenopharyngodon idellus*, a common host for cercariae of *C. sinensis* Fishponds are frequently contaminated by eggs in human faeces. Unlike *F. buski* the miracidia of *C. sinensis* hatch in the snail host after it has eaten the eggs (*see* **307**). Several genera of snails serve as intermediate hosts (eg *Bithynia* spp. **426**) for the development of sporocysts, rediae, and cercariae.

420 Cercaria of *C. sinensis* The cercariae leave the snail and encyst under the scales of various species of freshwater cyprinoid fish. *(× 40)*

421 Metacercaria in fish Man is infested by eating raw or incompletely cooked fish, a common delicacy among many Chinese. Metacercariae are thus ingested and excyst in the duodenum. *(× 100)*

422 *C. sinensis* in cat liver The young worms migrate up the common bile duct to the liver. They are seen here as whitish spots.

423 Adult *C. sinensis* At maturity they may reach two cm in length.

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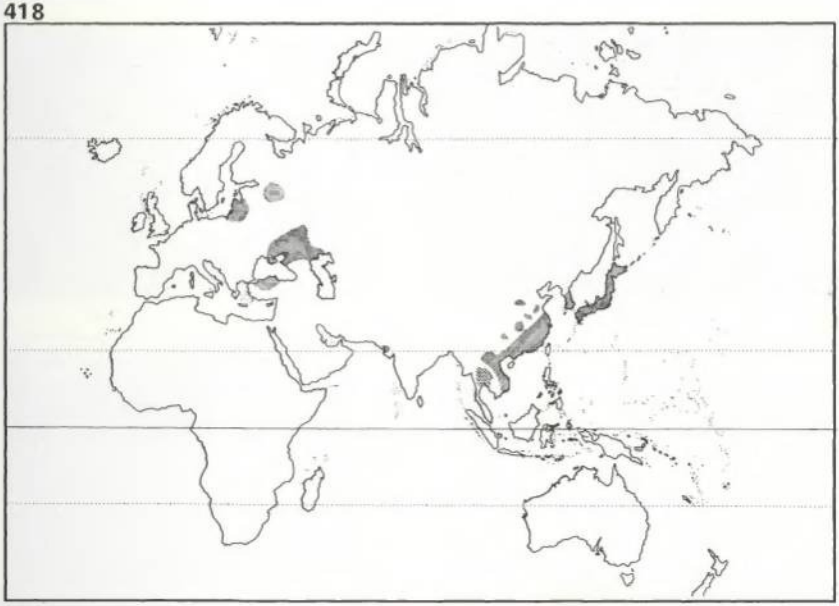

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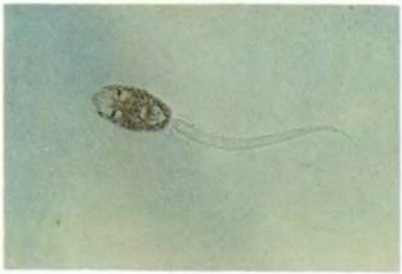

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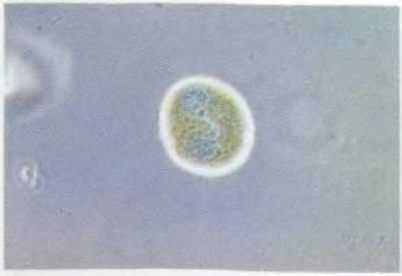

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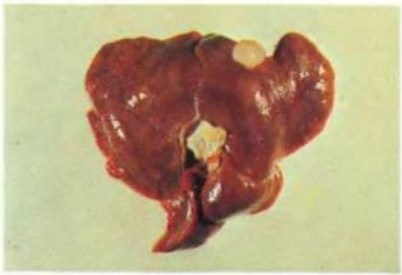

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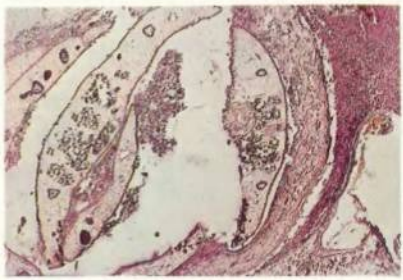

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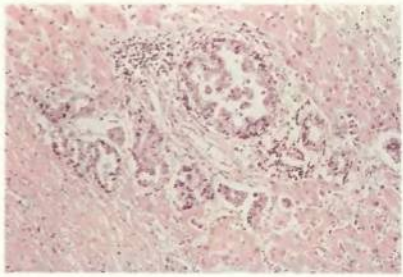

424 Section of *C. sinensis* in bile duct Direct mechanical damage and possibly toxic effects of the adult worms lead to fibrotic changes in the bile ducts. *(× 15)*

425 Cholangiocarcinoma of the liver Severe chronic infection with *C. sinensis* may lead to marked pericholangitic fibrosis, and finally multifocal cholangiocellular carcinoma of the liver. In this case metastases were widely distributed throughout the body. *(× 60)*

426 *Bithynia funiculata* The life cycle is similar to that of *C. sinensis*. Snails of the genus *Bithynia* serve as intermediate hosts of *O. felineus* and *O. viverrini* (*see also* **419**). *(× 3)*

Opisthorchis felineus and *Opisthorchis viverrini*

The distribution is shown in **418**. *O. felineus* is common in domestic cats, dogs, and some other animals in Eastern and Southeastern Europe, and parts of the USSR. It is largely replaced by *O. viverrini* in the Far East. This occurs mainly in Northeast Thailand where it infects up to 90% of the population in some villages. Cats as well as man serve as reservoirs of infection.

427 Lophocercous cercariae of *O. viverrini* (with keeled tails) The cercariae encyst in small fish and man is infected by eating the metacercariae in these. *(× 90)*

428 Metacercaria in fish Uncooked fish are the usual source of infection. *(× 200)*

429 *Cyprinus carpio*, host for metacercariae of *O. felineus* This edible freshwater fish is commonly infected in Taiwan.

426

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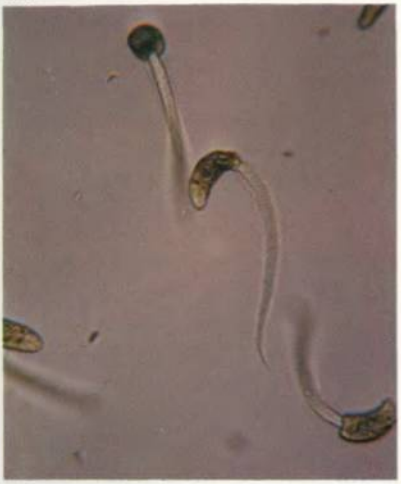

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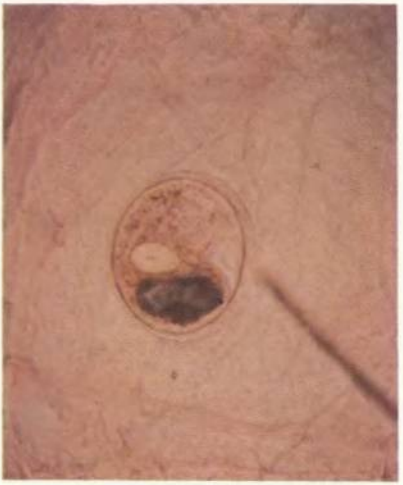

429

430–432 Adult flukes of *O. felineus*, *O. viverrini*, *C. sinensis* The adult flukes of *Opisthorchis* which are similar to those of *C. sinensis* live in the bile ducts and produce similar pathological changes. They can be distinguished by the structural details as seen in the figure. *(× 4½)*

Dicrocoelium dendriticum

434 Adult *D. dentriticum* from Egypt This is a rare infection of man but very common in sheep and other herbivora. The adult trematode grows up to 1.5 cm long and lives in the biliary tract. The eggs are similar to those of the *Clonorchis* group (*see* **307**). *(× 6)*

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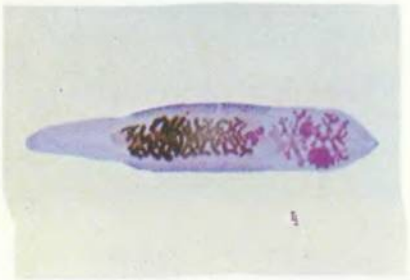

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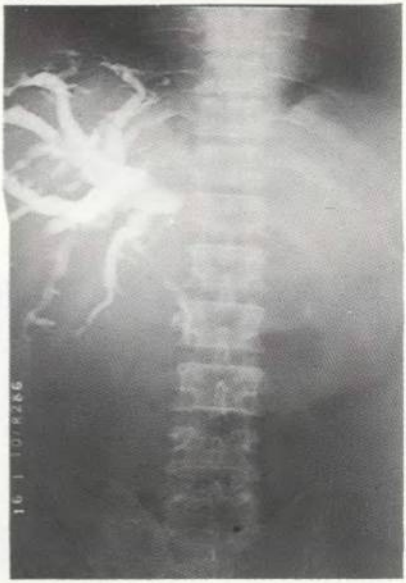

433 Cholangiogram of patient with opisthorciasis Dilatation of the main bile ducts and disorganisation of the biliary tract are seen.

434

Fasciola hepatica and *F. gigantica*

435 Ecology of fascioliasis, *Lymnaea* snail in wet pasteurage *F. hepatica* is a cosmopolitan disease of sheep, cattle and other herbivorous mammals. Man is commonly infected by eating wild watercress on which metacercariae have encysted.

436 *Lymnaea swinhoei* This snail is a host for *F. hepatica* in Taiwan.

435

436

437 Adult flukes in bile ducts of a sheep liver The adult flukes live in the bile ducts of sheep where they cause serious damage to the biliary tract.

438 Migrating flukes in liver After being swallowed the metacercariae pass through the intestinal wall and penetrate the liver via the liver capsule. The figure shows a sheep liver with migrating immature flukes. *(× 20)*

439 Adult *F. hepatica* in section of liver The surface spines of the adult fluke produce mechanical damage to the biliary epithelium. *(× 350)*

440–442 Miracidia, redia, and cercaria of *F. hepatica* Miracidia (**440**) hatched from eggs passed in faeces, infect snails of the genus *Lymnaea* where they develop through a typical trematode cycle, producing rediae (**441**), and cercariae (**442**). In England the common intermediate host is *L. truncatula* (*see* **407**). *(× 150)*; *(× 25)*;*(× 60)*

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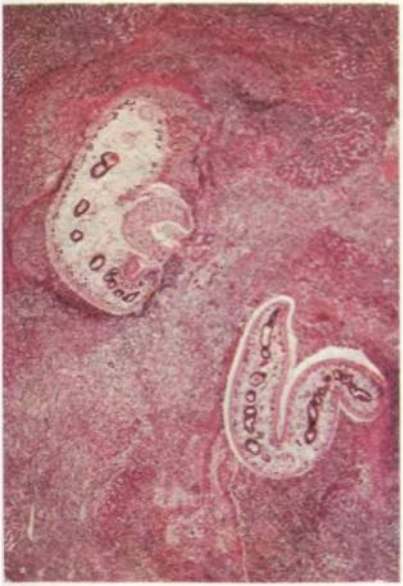

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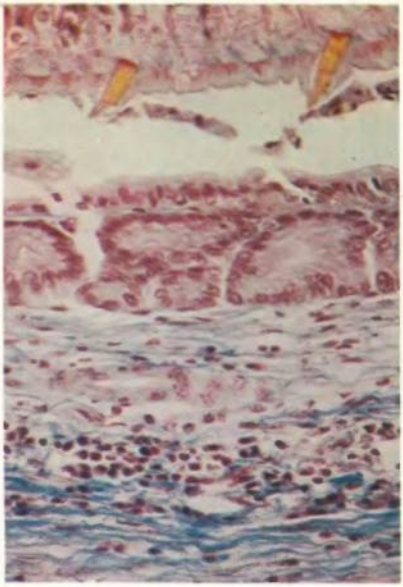

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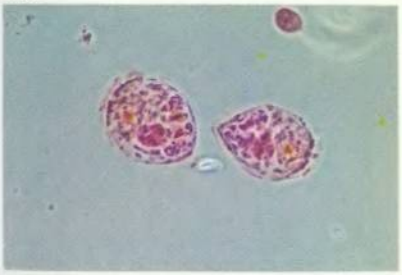

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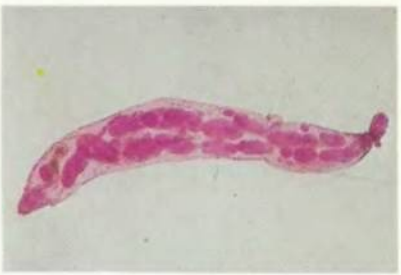

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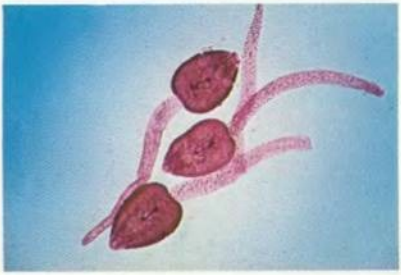
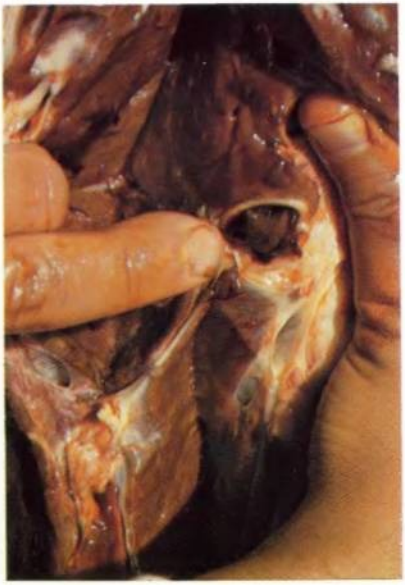


443 Metacercariae on grass The cercariae encyst on grass or other moist herbage such as watercress. When the metacercariae are ingested the cycle recommences. *(× 20)*

444 *F. gigantica* in bile duct of cow This species is the common liver fluke of cattle in Egypt where man is also infected occasionally.

443

444



PARAGONIMIASIS

445 Distribution of lung flukes Four species of *Paragonimus* have been identified in man. *P. westermani* commonly causes human infection in the Far East where it is widely distributed among other mammals. Several other species are probably involved. Human infections with *P. africanus* and *P. uterobilateralis* have been recognised in West Africa, and *P. heterotremus* in Thailand. Unidentified species occasionally produce infection in man in parts of Central and South America.

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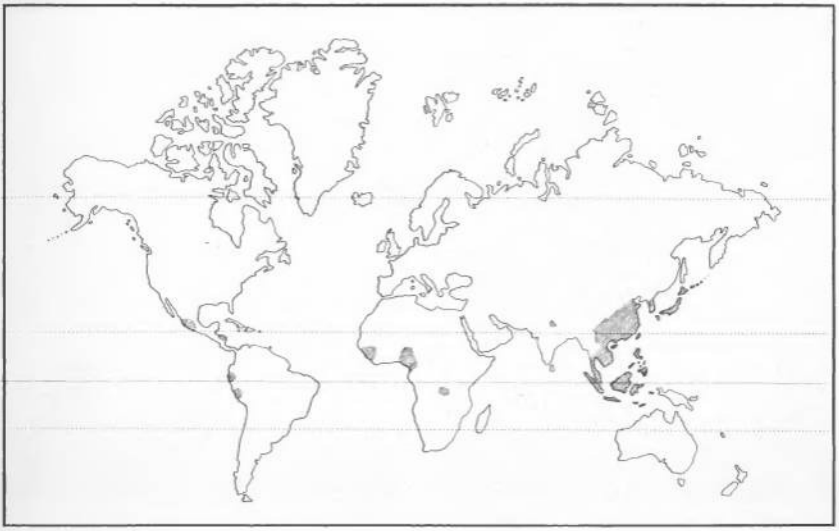

P. westermani

446 & 447 Adult *P. westermani* and *P. heterotremus* The adult fluke normally lives in the lungs. It is rather lemon-shaped and about one cm long when alive. The preserved specimens shown here have become flattened during preparation. *(× 6)*

448 Eggs in human sputum Eggs are passed in the sputum or swallowed to be passed later in the faeces. The miracidia hatching from the eggs penetrate snails of various genera including *Semisulcospira* and *Thiara* (*see* **Table XI**). After the usual cycle of development in the snail, microcercous cercariae emerge and encyst inside fresh water crayfish and crabs. Various crab-eating carnivores thus become the natural reservoirs of *Paragonimus* species.

449 *Potamon rathbuni*, a host of lung fluke Crabs are commonly eaten raw, or in the form of an uncooked paste with which the metacercariae are ingested.

446

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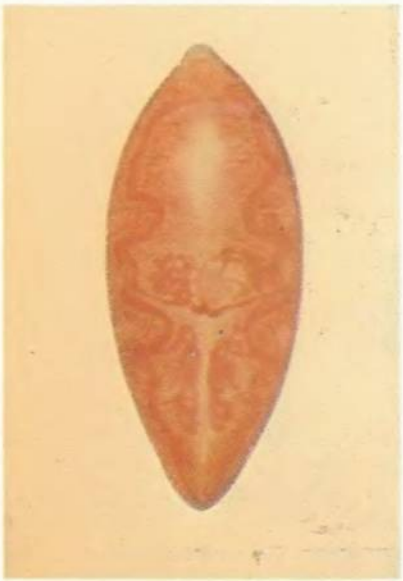

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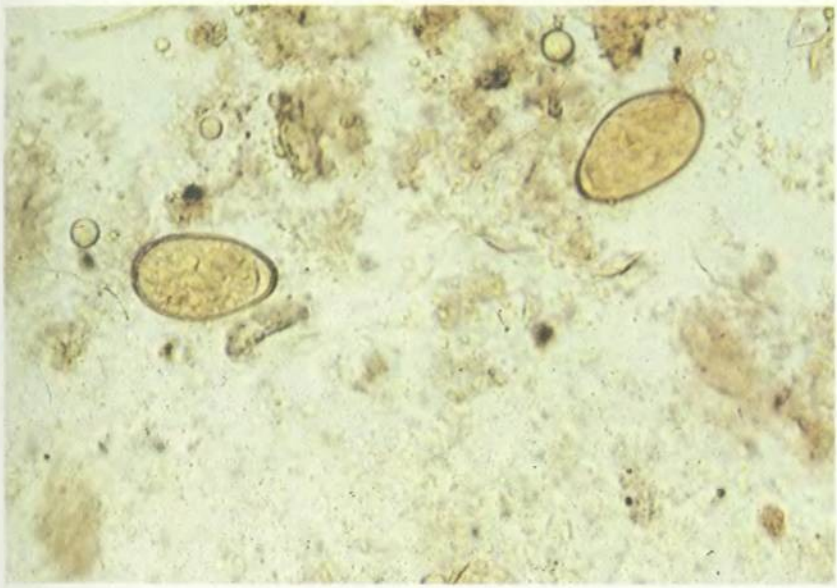

449

450 Metacercaria in crab gills Compare with the smaller metacercaria of *C. sinensis* (**421**). *(× 10)*

451 Lung of dog with adult *P. westermani* Metacercariae excyst in the duodenum from which they migrate through the peritoneal cavity and diaphragm to the lungs, where they mature inside capsules formed by the host tissue.

452 Section of lung The figure shows a section of lung containing encapsulated adult *P. westermani*. *(× 20)*

453 Chest X-ray Human infection is manifested by cough, haemoptysis, and other signs and symptoms which are commonly confused with those of tuberculosis. Typical shadows caused by the encysted adult trematodes may be seen on X-ray. This patient was infected with *P. uterobilateralis* in Eastern Nigeria. This species and *P. africanus* (which occurs in the nearby Cameroon Republic) have a similar life cycle and, like *P. westermani*, man is infected by eating crabs or crayfish. The animal reservoirs are not yet known.

454 Paragonimus cyst in brain *P. westermani* cyst found at post mortem in the brain of a 21-year-old Japanese girl.

450

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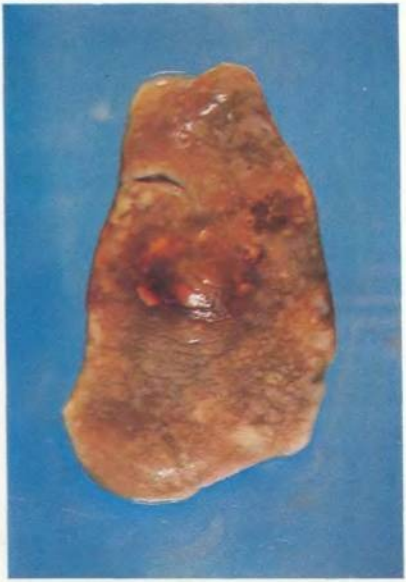

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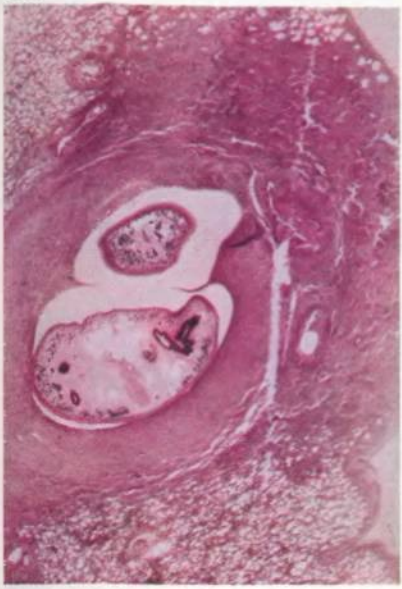

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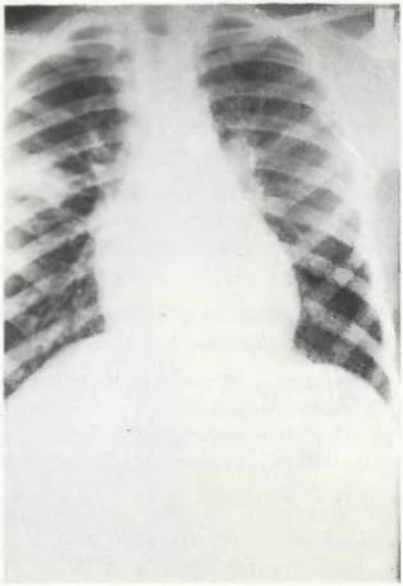

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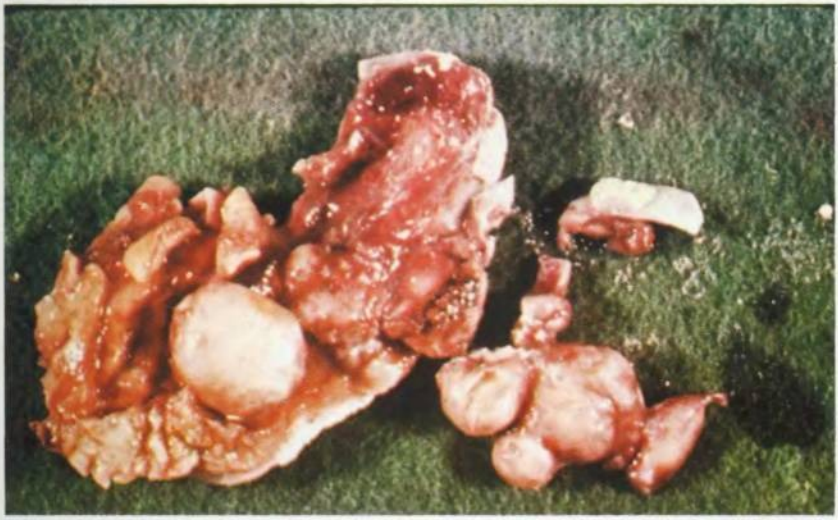


ANGIOSTRONGYLIASIS

455 Adult *Angiostrongylus cantonensis* The adult worm, about one to two cm long, is a common parasite of rodents in the Far East and Pacific. It lives in the pulmonary arterioles. *(× 3)*

456 First stage larvae in rat faeces The eggs passed in the bloodstream break through the pulmonary tract, are swallowed by the rodent and are passed in the faeces in which they may hatch to first stage larvae. *(× 100)*

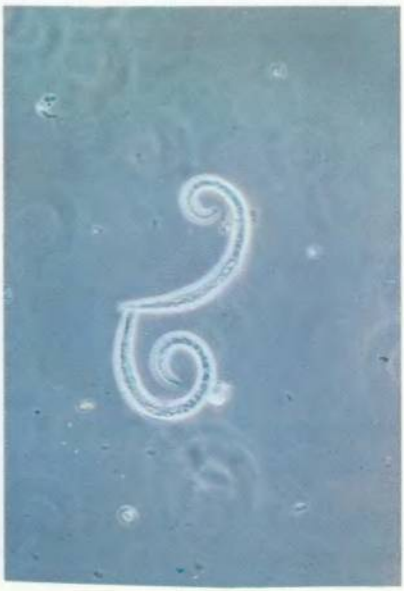
457–459 Intermediate snail hosts *Achatina fulica* **(457)**, *Cipangopaludina chinensis* **(458)**, *Bradybaena similaris* **(459)**. The larvae are eaten by snails of various genera including *Achatina*, as well as by some slugs and land planarians in which larvae develop to the third stage.

460 Third stage larvae in *Achatina fulica* New rats become infested when they eat the snails containing third stage larvae. *(× 100)*

461 Section of larvae in meninges of human brain Man may be infested by eating freshwater prawns but how these come to contain larvae is uncertain. He may also be infected by eating snails which contain the larvae. The larvae migrate to the brain where they cause an eosinophilic meningoencephalitis. *(× 60)*

455

456



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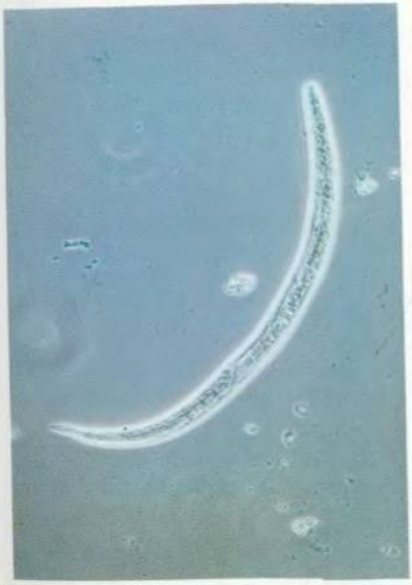

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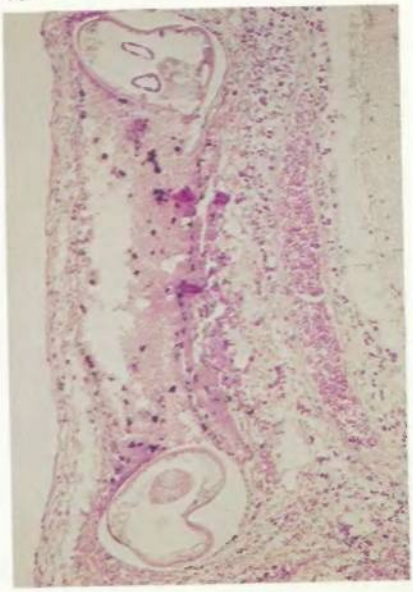


Part IV
Infections Acquired through the Gastro-intestinal Tract

Of the important pathogens that gain entry through the gastro-intestinal tract many are cosmopolitan in their distribution. Some cause diarrhoeal diseases (eg cholera, shigellosis, enteric fever) while others pass from the intestinal tract to cause disease in other organs (eg poliomyelitis, infective hepatitis, trichinosis). The pathogens include viruses, bacteria, protozoa, helminths and endoparasitic arthropods. The first three of these are directly infectious for man when they are passed in the faeces but, in the case of helminths, the eggs may become infectious only after maturation in the soil (eg *Ascaris*), or after passing through an intermediate host (eg *Taenia saginata*).*

The most important pattern of transmission is the passage of infective material from human faeces into the mouth of a new host, which is known as 'faeco-oral' transmission. This occurs mostly through inapparent faecal contamination of food, water, and hands – the three main points of contact with the mouth. Some of the pathogens that infect through the mouth are not excreted in the faeces, eg guinea-worm infection is acquired by drinking contaminated water, but the larvae escape through the skin. On the other hand, while the ova of hookworm are passed in the faeces the route of human infection is by direct penetration of the skin by the larvae after a period of incubation of the egg in the soil.

A number of the infections that are acquired through the gastro-intestinal tract characteristically occur in epidemic form, eg cholera and typhoid. Other may be more localised, affecting persons from the same household or institution, eg amoebiasis, enterobiasis. The most topical infection of this section is the ongoing pandemic of El Tor cholera which began in 1961, and which continues to spread by the symbiotic combination of a symptomless carrier state and wide-scale travel.

**Helminthiases acquired from the soil are included in Part II; those requiring a snail intermediate host in Part III.*

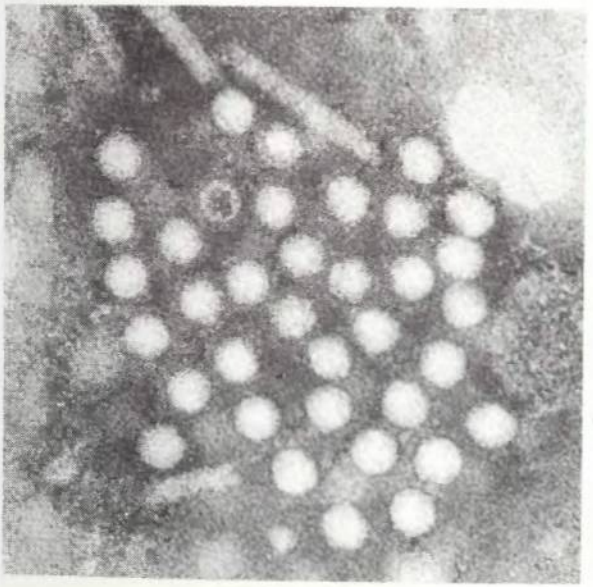
VIRAL INFECTIONS

Poliomyelitis

462 Ultrastructure of poliomyelitis virus Spherical particles measuring 25 nm in diameter. Both 'full' and 'empty' particles are shown. The virus is widely spread throughout the tropics. *(× 220 000)*

463 Ethiopian boy with paralysis of left leg Young indigenous children are predominantly affected, as well as non-immunised expatriates of all ages.

462



463

Infective Hepatitis

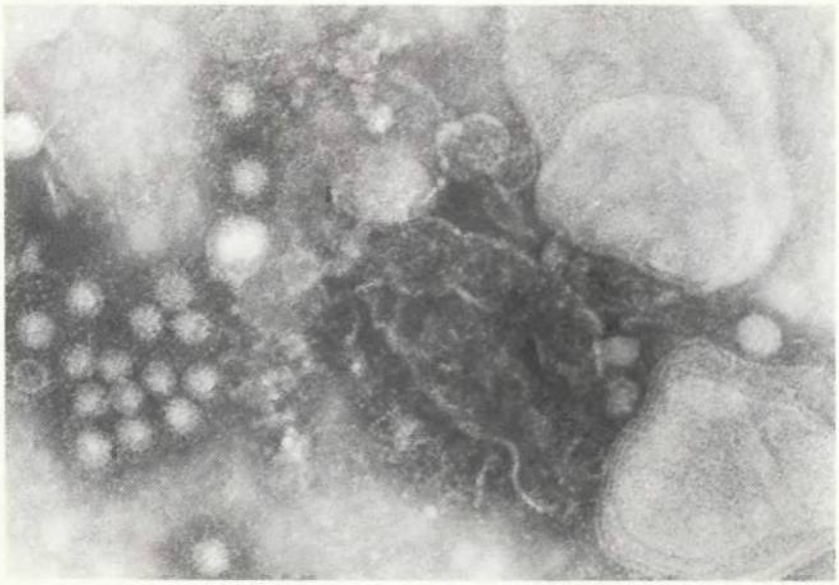
464 Ultrastructure of 'hepatitis A virus' Virus-like particles measuring 27 nm in diameter found in faecal extracts from an adult volunteer during the acute phase of hepatitis A after inoculation with MS–1 serum. The particles are heavily coated with antibody present in convalescent serum. *(× 252 000)*

465 Hepatitis B antigen-containing serum Serum containing hepatitis B antigen showing the presence of three distinct morphological entities: (1) Small pleomorphic spherical particles measuring 20–22 nm in diameter. (2) Tubular forms of varying length with a constant diameter of 20 nm and frequently with a terminal bulbous swelling. (3) Double-shelled spheroidal Dane particles, approximately 42 nm in diameter, with a core measuring 27 nm in diameter. There is substantial evidence that this particle is the hepatitis B virus. *(× 227 000)*

466 Jaundiced child with infective hepatitis Fever, anorexia, and later jaundice are characteristic clinical features. The disease is particularly severe in pregnancy. Note deep jaundice and spider naevus on the cheek.

467 Urine from child The urine is dark coloured and contains bilirubin and urobilinogen.

464



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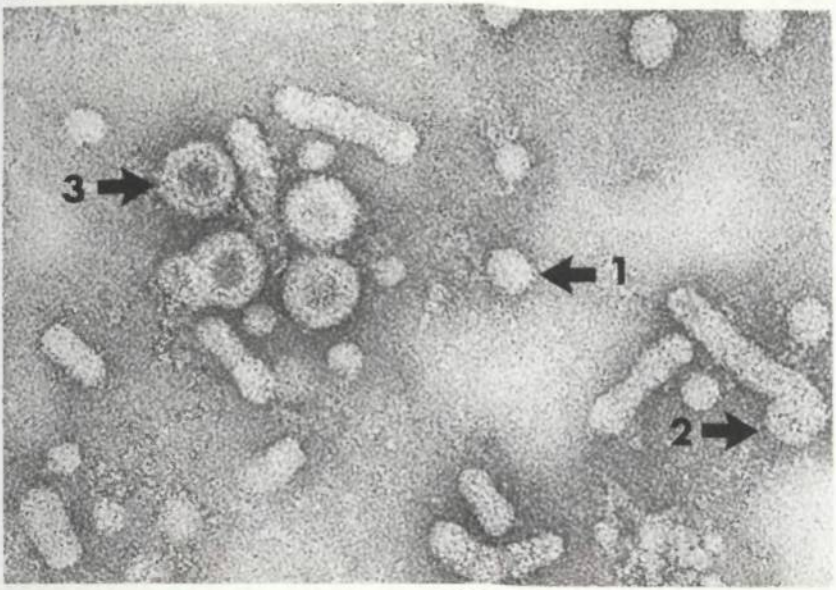

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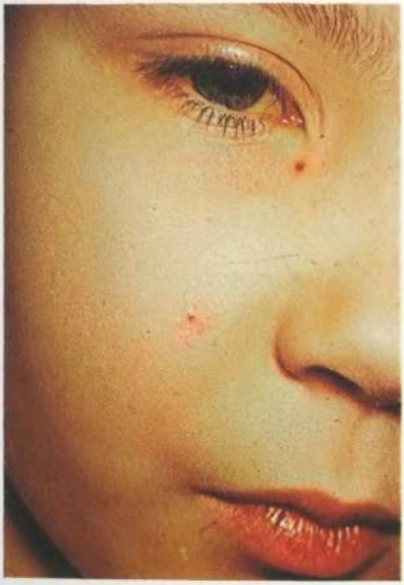

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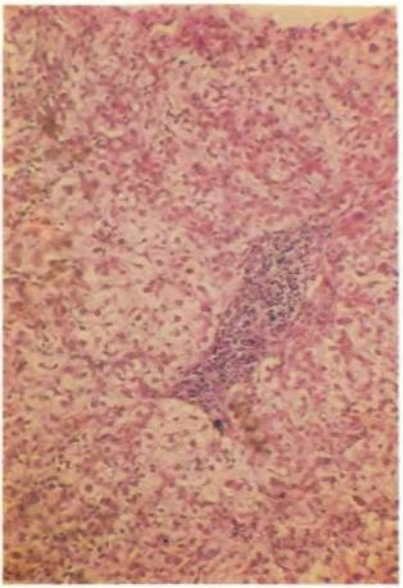

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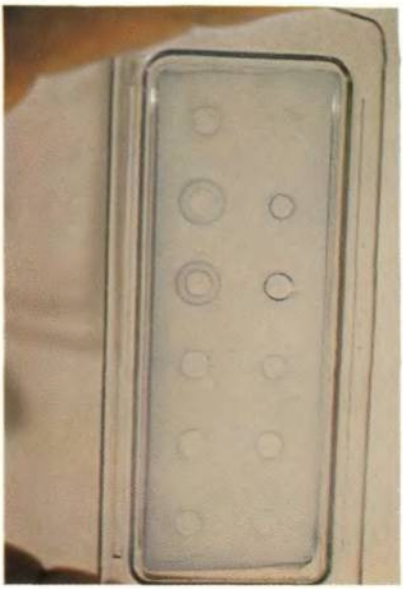


468 Biopsy of liver Histologically the characteristic features are ballooning and a feathery degeneration of the liver parenchymal cells. *(× 150)*

469 Single radial immunodiffusion test Hepatitis B antigen positive serum is readily detected by this simple serological procedure.

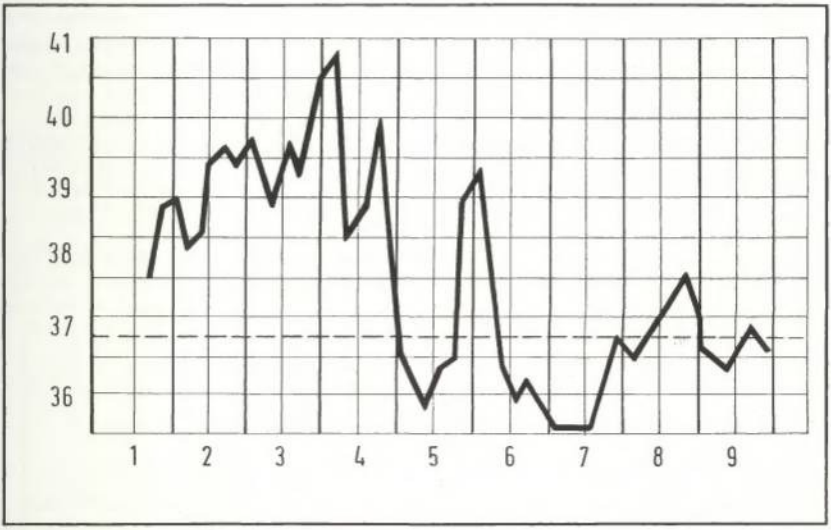
BACTERIAL INFECTIONS

Typhoid (Enteric) Fevers

470 Temperature chart The fever is high and accompanied by confusion and severe prostration ('typhoid state'). There is often a dissociation between pulse and temperature, and an accompanying leucopenia.

471 Rose spots The classical rose spots may appear irregularly, usually the abdominal wall, lower thorax, and on the back of the trunk.

470



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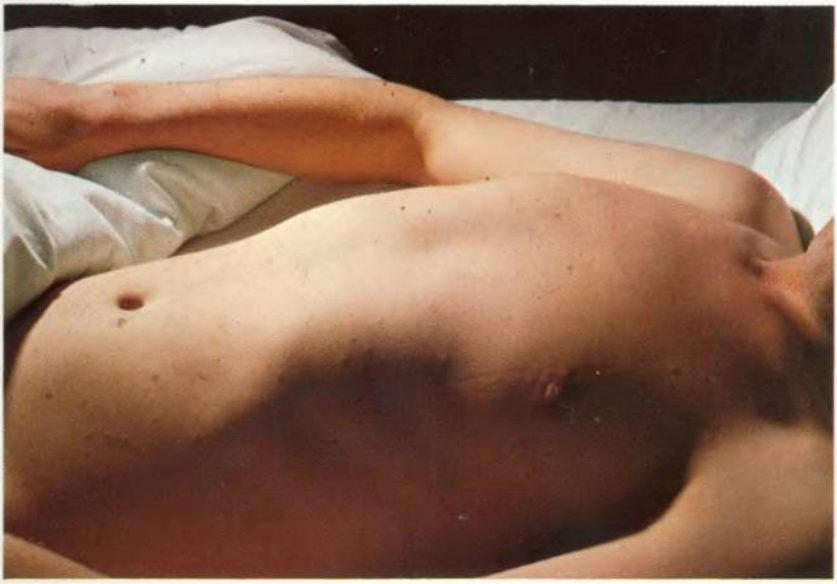
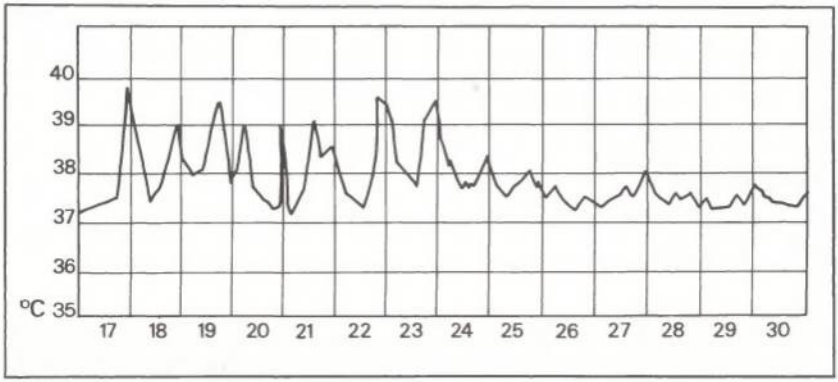


472 Temperature chart in case of Salmonellosis with *S. mansoni* infection An association has been established between chronic *Salmonella typhi* infection and *S. mansoni*. Eradication of the helminth infection is needed to eliminate the *Salmonella* organism.

473 Ulceration of Peyer's patches Intestinal haemorrhage and perforation are the two most serious complications of typhoid fever. They are due to ulceration of Peyer's patches.

472



473

Non-specific Gastroenteritis

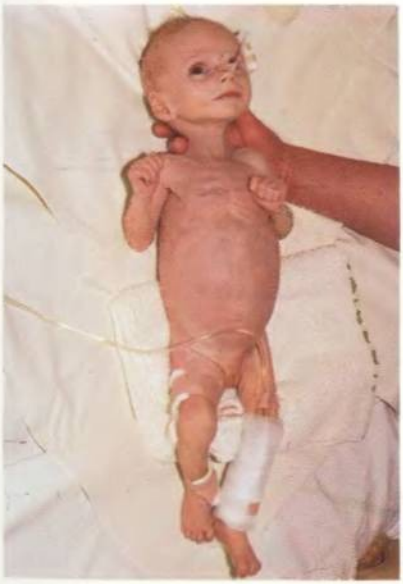
Gastroenteritis due to a variety of organisms is one of the major causes of childhood mortality in the tropics. The substitution of the milk bottle for breast feeding is increasing rather than decreasing the problem. Mismanagement of artificial feeding is a serious cause, due to pathogenic coliform organisms.

Unwashed fruits contaminated with pathogenic organisms by flies are also a common cause of non-specific gastroenteritis in children and adults.

474 Dehydration due to gastroenteritis Severe dehydration due to diarrhoea and vomiting is the salient diagnostic feature.

475 Marasmus complicated by dehydration In an already malnourished infant gastroenteritis may rapidly lead to death.

474



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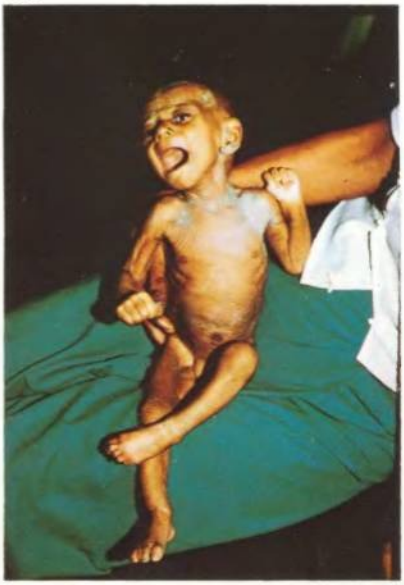
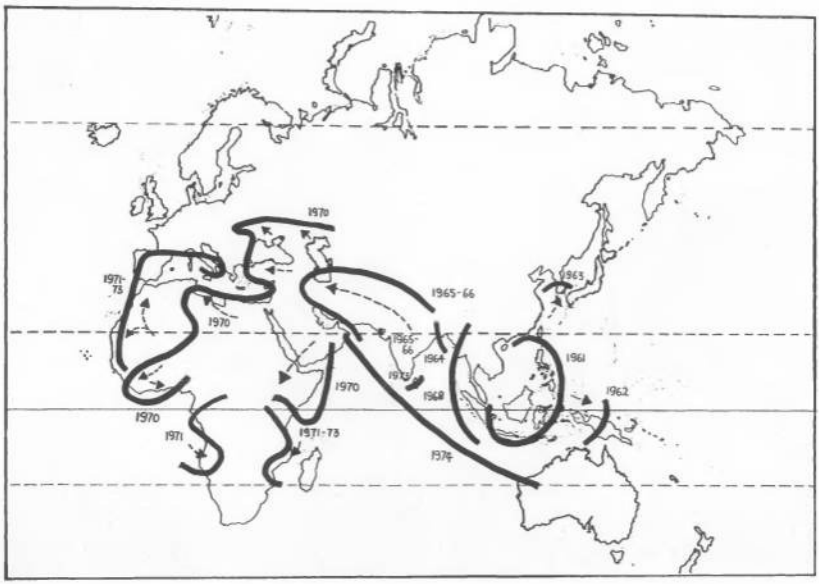


Cholera

476 Distribution map of cholera *Vibrio cholerae* El Tor biotype is responsible for the present pandemic of cholera in the world. Symptomless carriers are very important in the epidemiology of this disease.

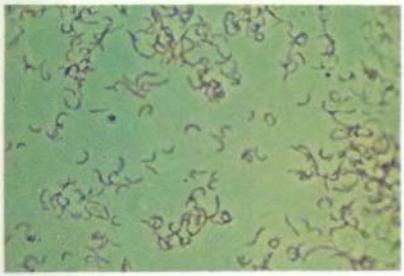
476



477 Cholera vibrios *Vibrio cholerae*, the classical organism, is limited to the Indo-Pakistan subcontinent. *V. cholerae*, El Tor is more widely distributed. *(×600)*

478 Cholera cot The disease is characterised by severe vomiting and watery diarrhoea. Nursing of patients in a cholera cot enables a rapid assessment of fluid loss to be made.

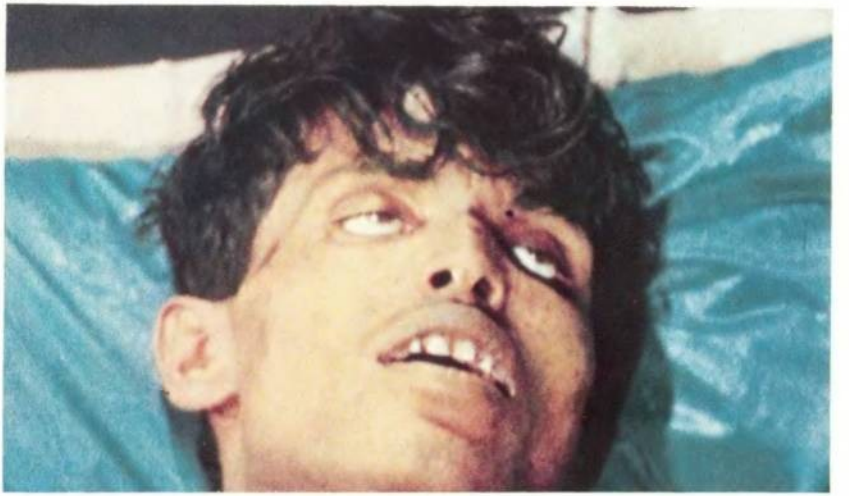
477



478

479 & 480 Dehydrated patient before and after treatment Dehydration (**479**) is the cause of death. Rapid rehydration with isotonic saline (**480**) is life saving.

479



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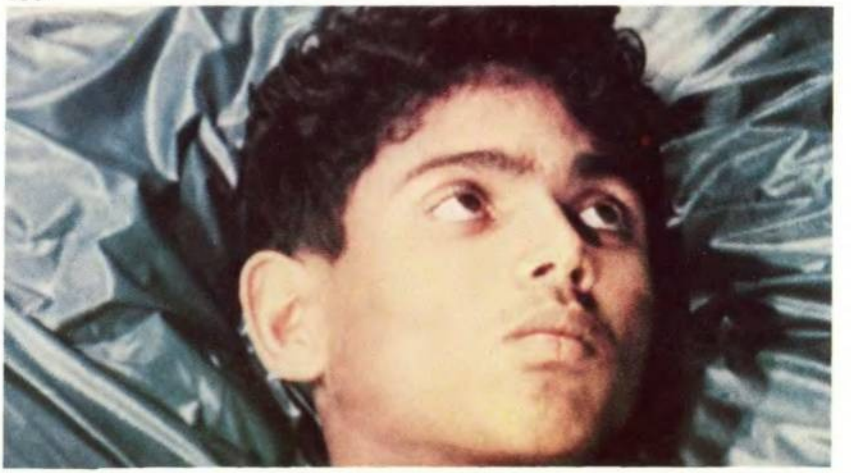
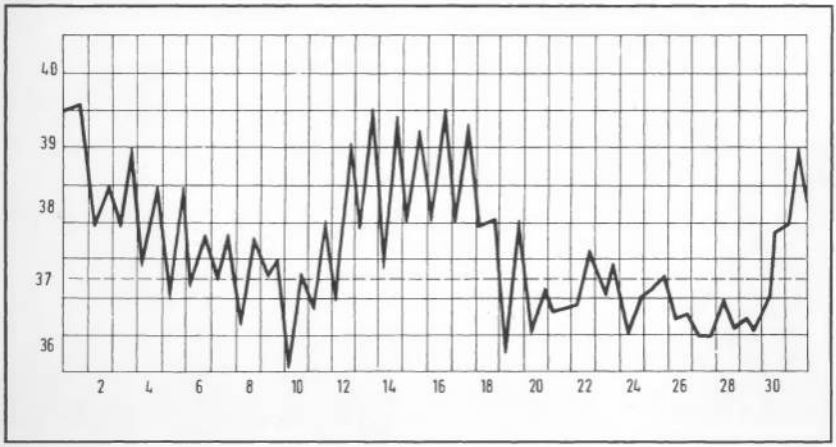


Brucellosis

Goats, cows and pigs are the most common animal reservoirs of infection with *Brucella abortus*, *B. melitensis* and *B. suis*. Infection is acquired by drinking raw milk or milk products.

481 Temperature chart The typical fever is remittent and undulating. Moderate leucopenia and splenomegaly are usual. A sharp drop of one or more degrees somewhere in the course is common and is a useful differential feature. In some areas the fever is atypical.

481



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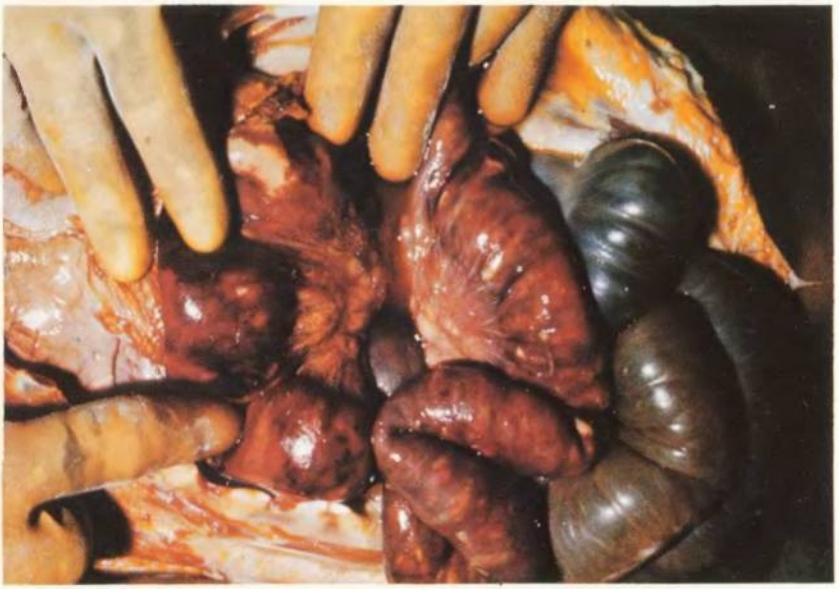


Enteritis Necroticans (Pigbel)

482 Appearance of intestine at operation Extensive sloughing and necrosis of the large bowel occurs in this condition which is related to infection with *Clostridium welchii* type C, due to the consumption of insufficiently cooked pork. A vaccine is now available for the prevention of this disease.

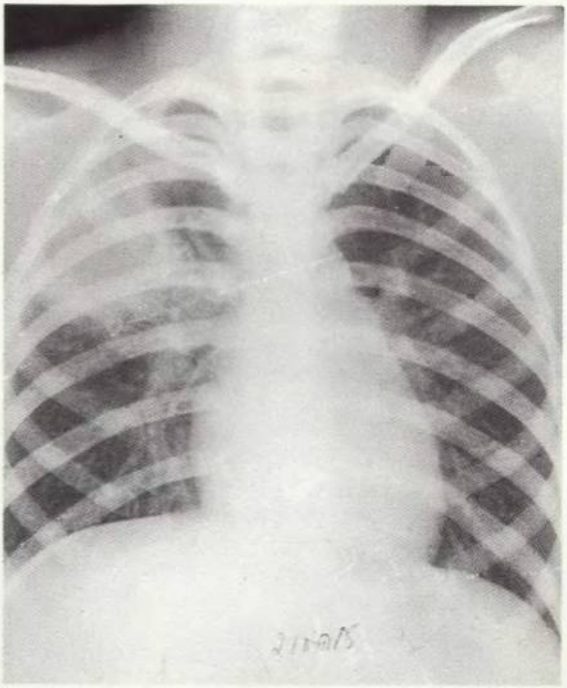
483 Old woman feeding her pig In the highlands of New Guinea, Enteritis necroticans is related to pig feasting which is an integral and complex part of the indigenous culture of highland tribes. The feeding of infant pigs may even take priority over the feeding of human infants.

483

Melioidosis

484 Right upper pneumonia due to melioidosis This disease occurs in the Far East and is due to infection with *Pseudomonas pseudomallei* (Whitmore's bacillus). The route of infection is uncertain and it may be water-borne or air-borne, or acquired by contamination of wounds with soil. Acute septicaemia may be followed by disseminated abscess formation, or the disease may be subacute from the start. This is an X-ray of a 14-year-old Thai boy who presented with pneumonia which failed to respond completely to antibiotic therapy. He died six months later with miliary peritoneal deposits containing *P. pseudomallei*.

484

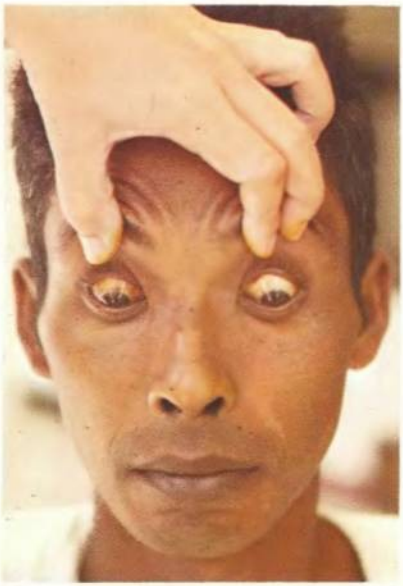


Leptospirosis

485 Injection of conjunctivae Infection with one of a variety of organisms of the genus *Leptospira* causes illness ranging from a mild, transient fever to severe hepatitis and liver failure. Injection and small haemorrhages of the conjunctiva are not uncommon. The disease is a zoonosis usually acquired from contact with water contaminated with animal urine, especially of rodents. It is global in distribution but commoner in tropical areas.

486 *Leptospira icterohaemorrhagiae* The organisms are readily seen in silver stained preparations, especially with a phase-contrast microscope. *(× 900)*

485



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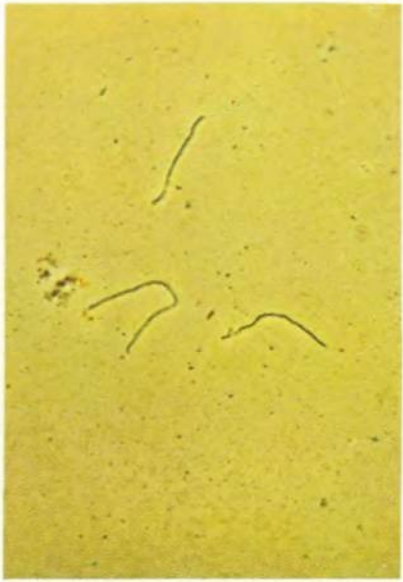
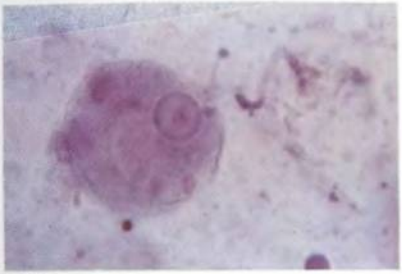


487–495 Comparison of trophozoites (487–491) and cysts (492–495) of intestinal protozoa *E. coli* (**487**), *E. histolytica* (**488**), *E. nana* (**489**), *I. butschlii* (**490**), *D. fragilis* (**491**), *E.* cyst (**492**), *E. histolytica* (**493**), *E. nana* (**494**), *I. butschlii* (**495**). (**487, 488** *trichrome* × *900*; **492–494** *iodine*; others *haematoxylin* × *600* except **495** × *900*)

*(*See* **Table XII**)

487



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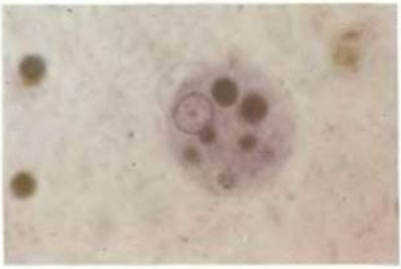

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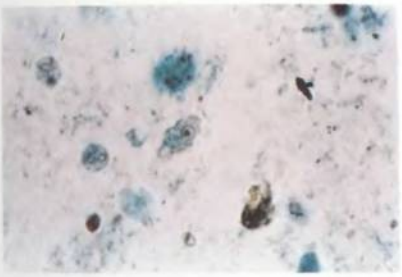

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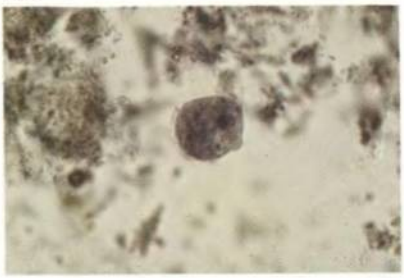

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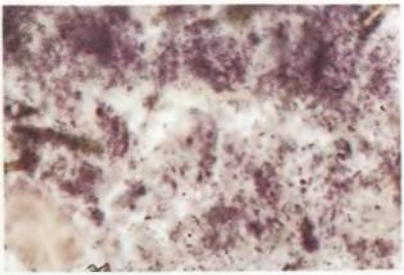

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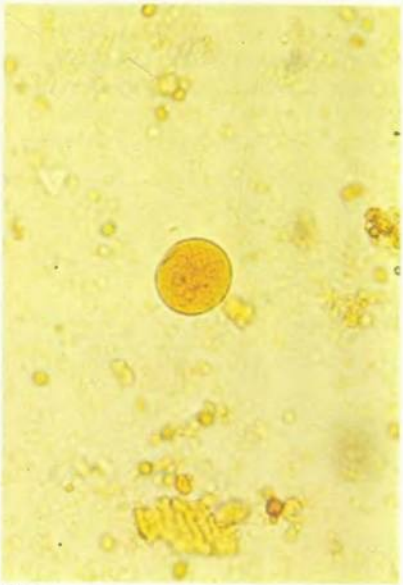

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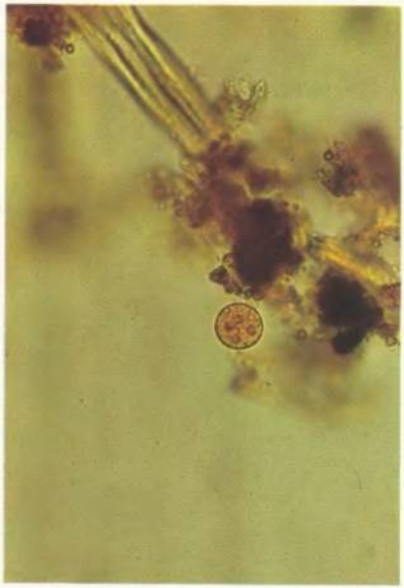

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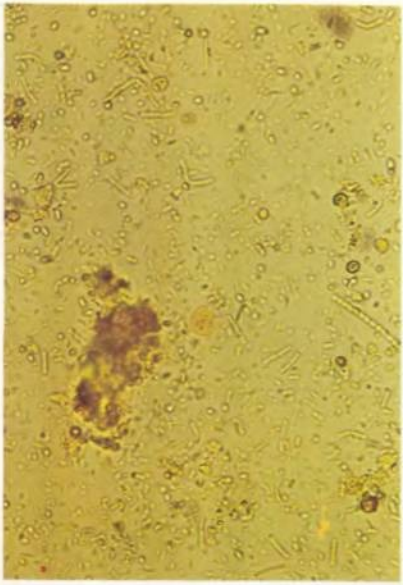

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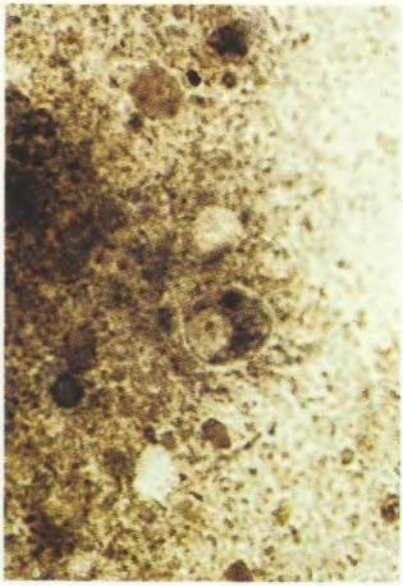
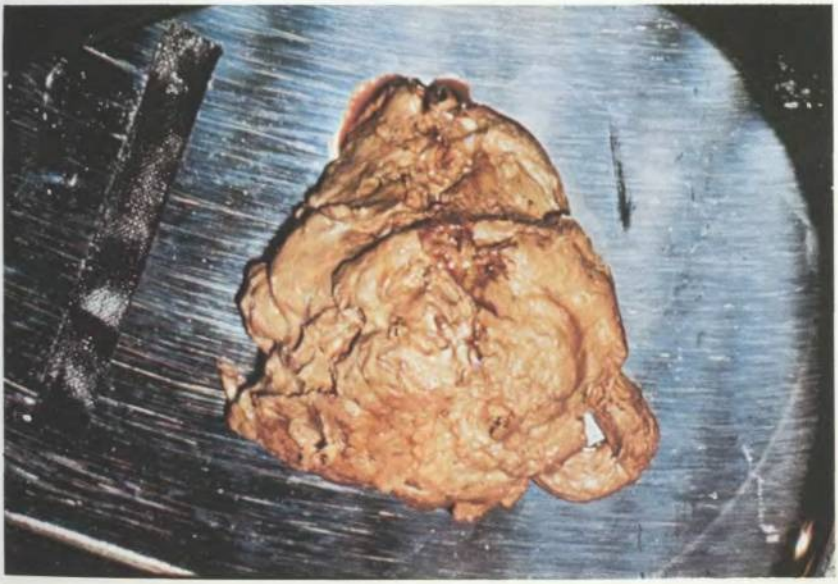


496 Macroscopic appearance of stool In amoebic dysentery the stool is loose, containing mucus and blood mixed with faecal material. As distinct from bacterial dysentery there is no cellular exudate.

496



497 Living *E. histolytica* Rather rapidly moving trophozoites of *Entamoeba histolytica* containing ingested erythrocytes may be found in a freshly passed specimen. *(× 900)*

498 Sites of predilection The commonest sites for localisation of *E. histolytica* in the intestine are the caecum and descending colon.

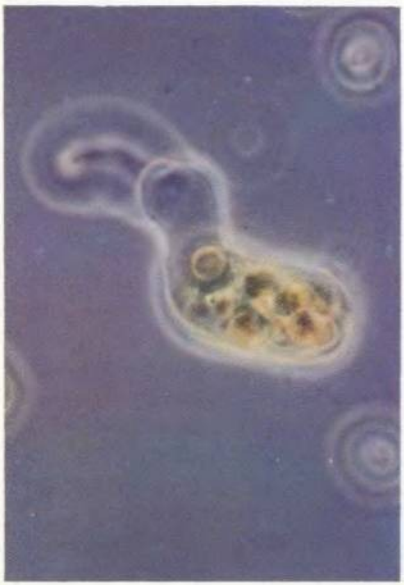
499 Amoebic ulceration of caecum In fulminating infections, the destruction of all layers of the intestinal wall is extensive, and ulceration may be confluent. These lesions are frequently seen in pregnancy and the puerperium.

500 Section of colon wall as seen in biopsy Typical 'flask-shaped' ulcers are seen in the intestinal wall which is invaded by *E. histolytica* trophozoites. The intervening mucosa is usually normal. *(× 150)*

501 Temperature chart of patient with amoebic liver abscess The triad of swinging temperature, profuse sweats and leucocytosis is indicative of liver abscess, especially when associated with pain in the right hypochondrium.

502 Scintoscan of patient with amoebic liver abscess The liver is often enlarged and tender, sometimes bulging in the abscess area.

497



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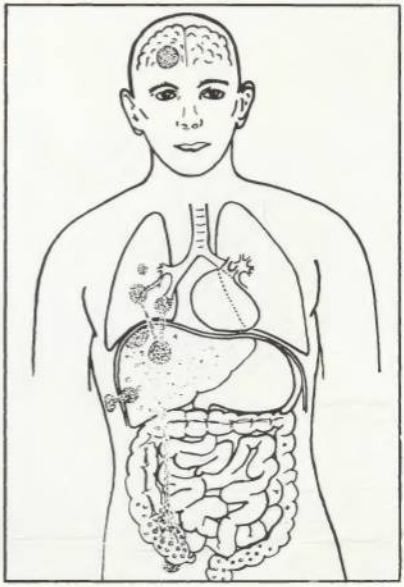

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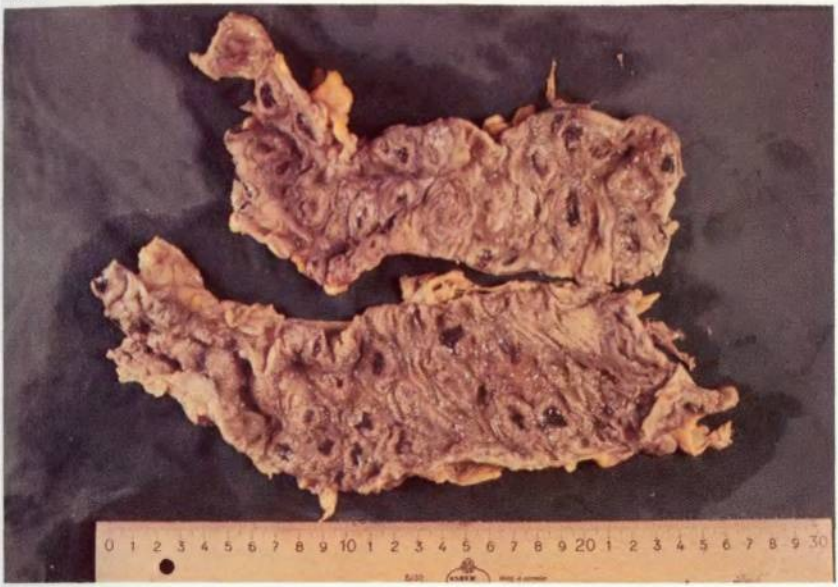

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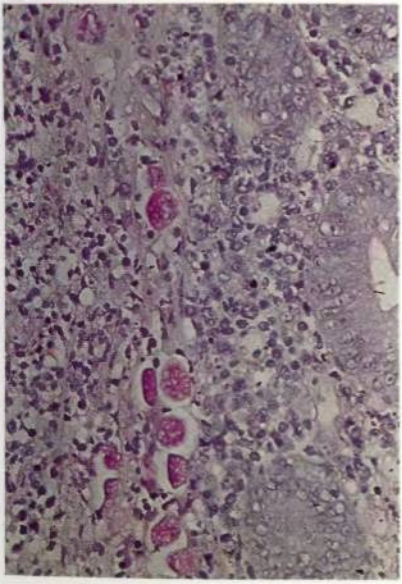

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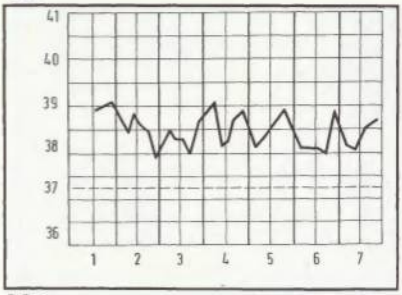

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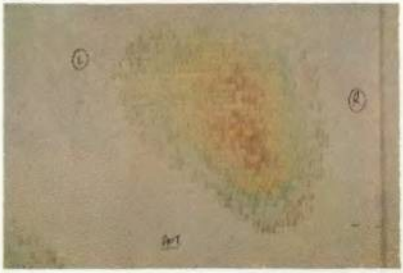


503 X-ray of liver abscess An amoebic abscess of the liver is usually single. The volume of the contents of the lesion may vary from 500 to 1 500 ml.

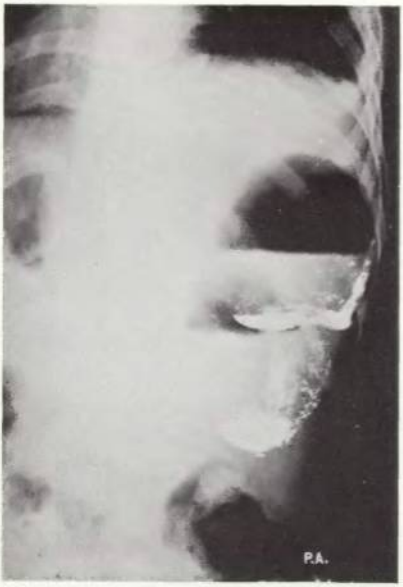
504 Aspiration of liver abscess Aspiration of large abscesses is usually needed as an adjunct to specific chemotherapy for successful treatment.

505 Amoebic pus The pus is chocolate-coloured. Trophozoites usually containing ingested red blood cells may be found in the pus if it is examined immediately after aspiration.

506 Macroscopic appearance of liver abscess The shaggy periphery is irregular and consists of stroma and layers of compressed liver parenchyma.

507 Section of liver abscess *E. histolytica* are often found in large numbers in the tissue at the periphery of the lesion. *(× 150)*

503



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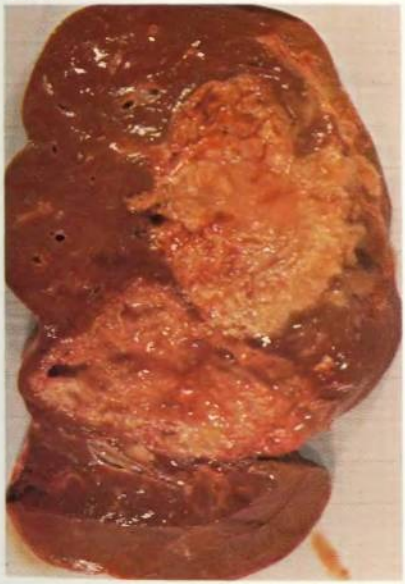

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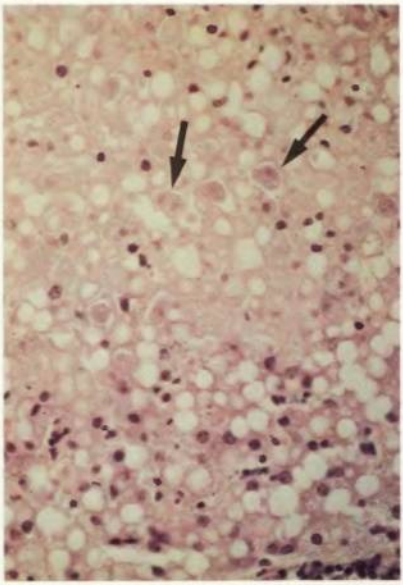


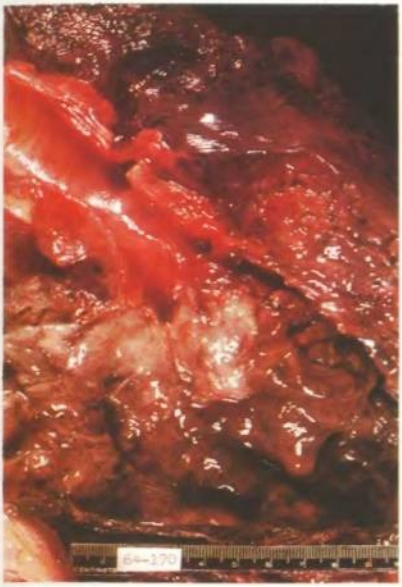
508 Amoebic abscess of lung Extraintestinal infections occur commonly in the liver, but any site of the body may be affected. The lung is sometimes affected by rupture of a hepatic abscess through the diaphragm. Typical amoebic pus may be coughed up with the sputum.

509 Amoebiasis of the skin Amoebic infection of the skin occurs. This patient was erroneously operated on for a perforated duodenal ulcer and no antiamoebic drugs were given. Sloughing of the skin occurred and amoebae were recovered from the skin lesion.

510 Amoebic balanitis Amoebic infection of the genital organs can result from normal or abnormal sexual intercourse.

511 Fluorescent antibody staining of *E. histolytica* FAT is the most sensitive immunodiagnostic test available for invasive forms of amoebiasis. It has proved a valuable adjunct to direct microscopical diagnosis of amoebiasis. Fluorescence of cultured *E. histolytica* exposed to serum from a patient with hepatic involvement is seen in this figure. *(× 900)*

508



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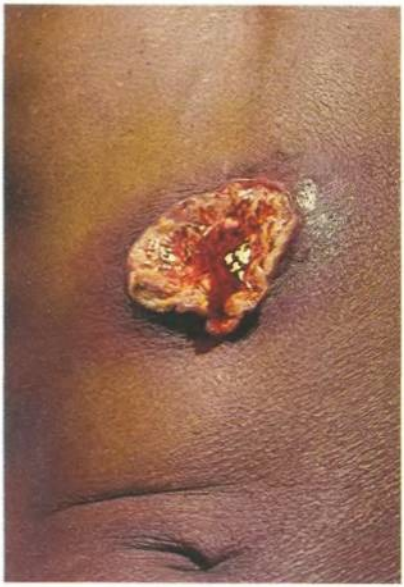

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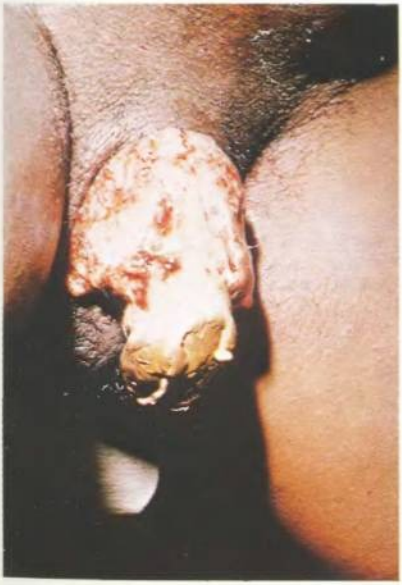

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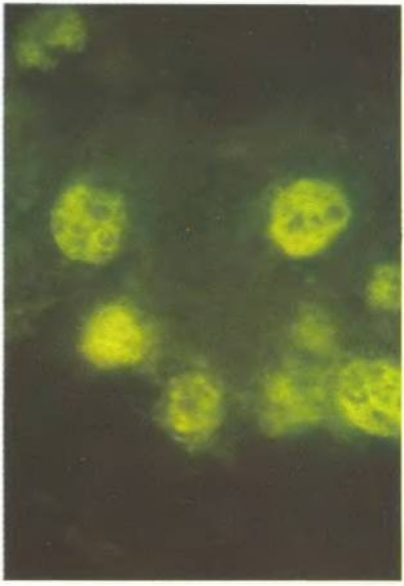
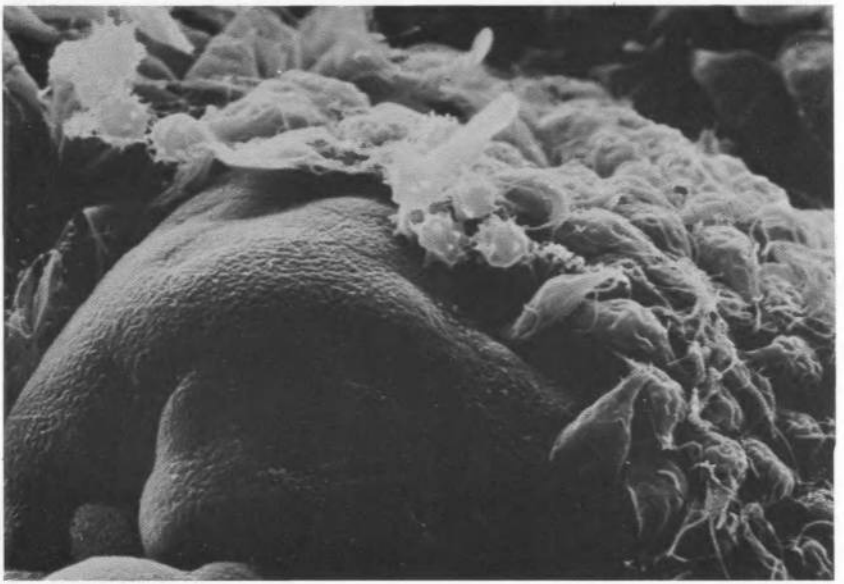


Giardiasis

512 Scanning electron micrograph of *Giardia lamblia* in jejunal biopsy Severe infection with *G. lamblia* can result in partial villous atrophy of the duodenum or jejunum with resulting steatorrhoea. Although the organism is commensal in many individuals, it is considered particularly pathogenic in children in the New World, and is a cause of diarrhoea and a malabsorption syndrome in travellers. *(× 1 000)*

513–517 Parasitic flagellates *Giardia* trophozoite (**513**), *Trichomonas vaginalis* (**514**), *Cheilomastix mesnili* (**515**), *Giardia lamblia* cyst (**516**), *Chilomastix mesnili* cyst (**517**). *C. mesnili* is non-pathogenic. *(× 900)*

512



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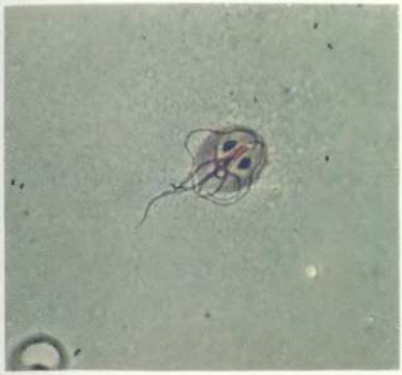

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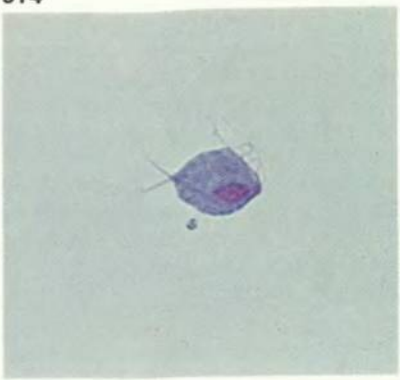

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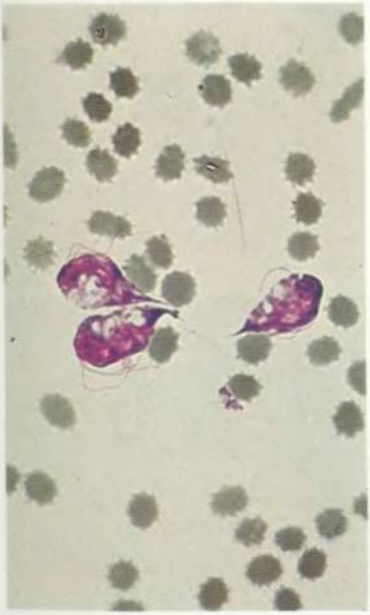

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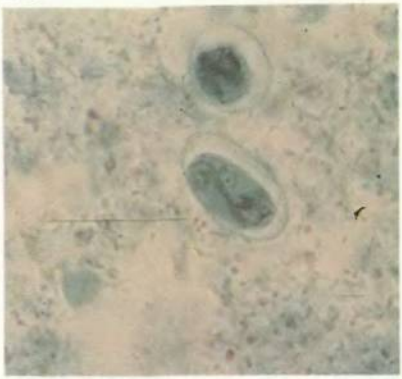

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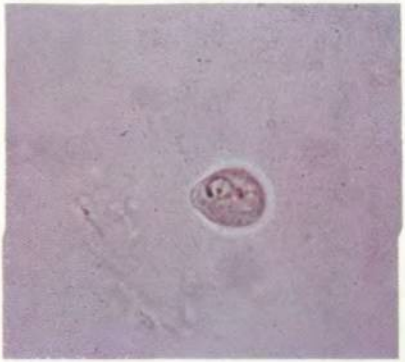


Coccidial Infections*

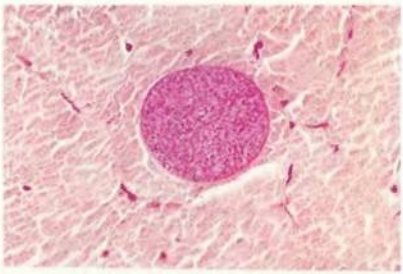
518 Section of sarcocyst in human muscle In the muscles of the intermediate host *Sarcocystis* forms fusiform cysts containing large numbers of bradyzoites. Although it is rarely diagnosed in life this condition may be common in man who harbours the sarcocysts of a number of different species, none of which have so far been identified. '*Sarcocystis lindemanni*' is no longer considered to be a valid species. *(× 250)*

519 *Sarcocystis hominis*-like oocysts in intestinal epithelium. This coccidian parasite is common in man and is acquired from the consumption of raw or inadequately cooked beef in which the sarcocyst stage occurs. In this case man is the definitive host. Sporulated oocysts or single sporocysts are passed in the faeces. *S. suihominis* is acquired from pork. *(× 325)*

520 *Isospora belli* oocysts *I. belli* unsporulated oocysts are passed in the faeces. The infection may be associated with diarrhoea, due to development of the parasite in the intestinal epithelium, or symptomless. *(× 450)*

*(*See* **Table IV**)

518



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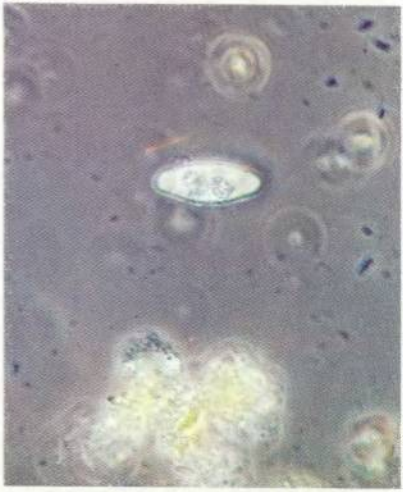


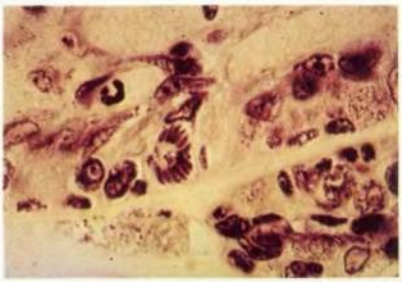
Toxoplasmosis

521 *Toxoplasma gondii* in epithelial gut cell of a cat, the natural host The definitive host of the coccidial protozoan, *Toxoplasma gondii* is the cat which passes infective oocysts in its faeces. These will develop to a cystic asexual stage in a very wide variety of mammalian hosts, including man. Infection is acquired either from ingesting these from raw meat, or through contamination by oocysts from cat faeces. *(× 600)*

522 Sporulated oocysts of *T. gondii* The tiny oocysts (10μ diameter) are passed unsporulated. *(× 325)*

523 Endozoites of *T. gondii* in a leucocyte Asexual and sexual reproduction occur in the epithelium of the cat's intestine. In man acute infection may result from ingestion of any stage, a proliferative phase of endodyogeny occurring in many tissues. *(Giemsa × 900)*

521



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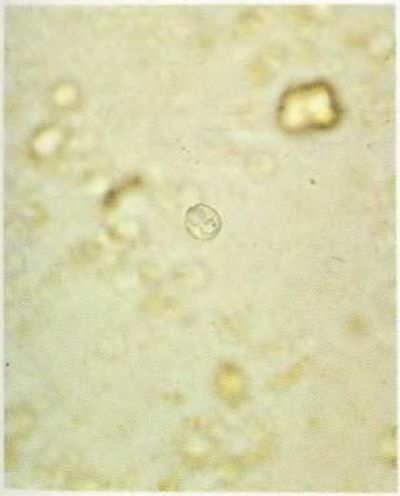

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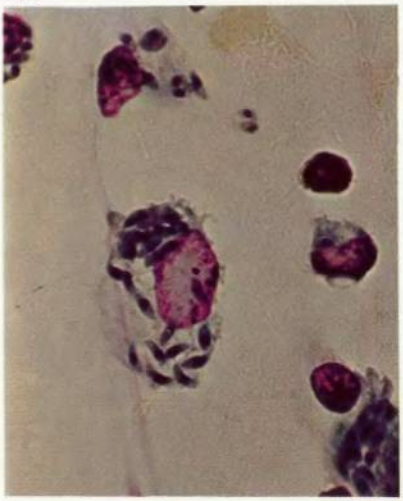
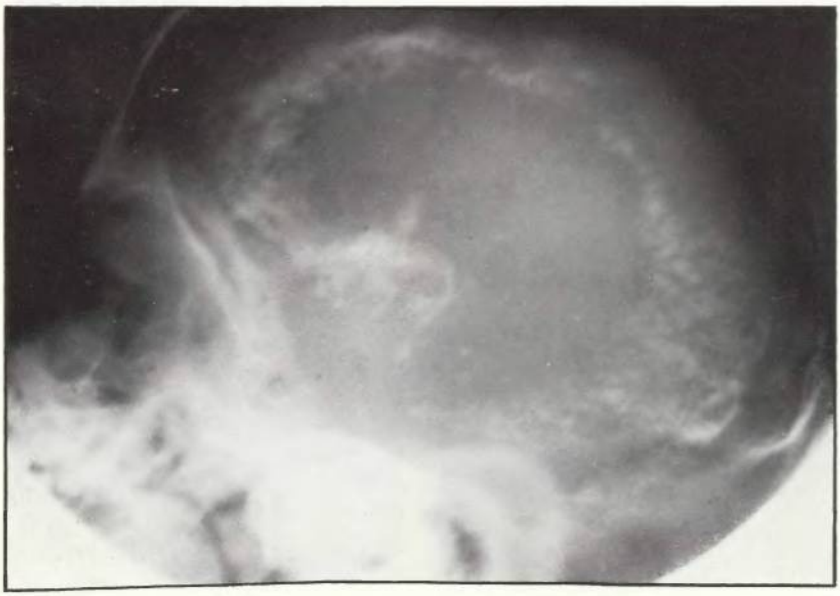


524 Brain X-ray showing calcification in the ventricles and sub-ependymal tissues Transplacental infection, usually from an acutely infected mother, in the fourth month of pregnancy produces congenital toxoplasmosis with typical calcification of the sub-ependymal tissues, and sometimes dilation of the ventricles due to rapid proliferation of the parasites.

525 Post-mortem appearance of brain Irregular areas of calcification in the lateral ventricles are seen in this brain of a seven-week-old child dying of toxoplasmosis. Mental disorders and blindness are common in children who survive.

526 Ventricular exudates in congenital toxoplasmosis Cross section through brain of six-week-old infant showing fibrinous ventricular exudate and septa, with periventricular necrosis (Case 4 of Frenkel and Friedlander, 1951 *U.S. PHS Publication No 141*)

524



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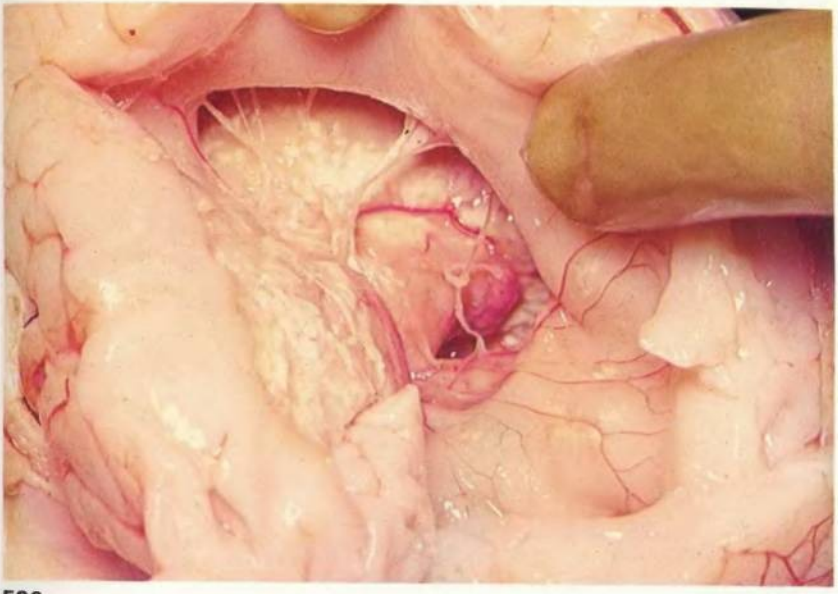

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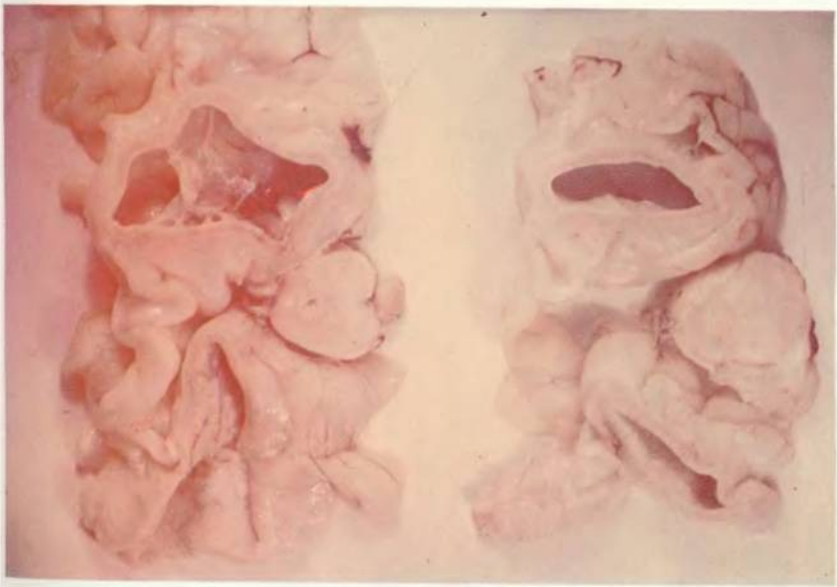


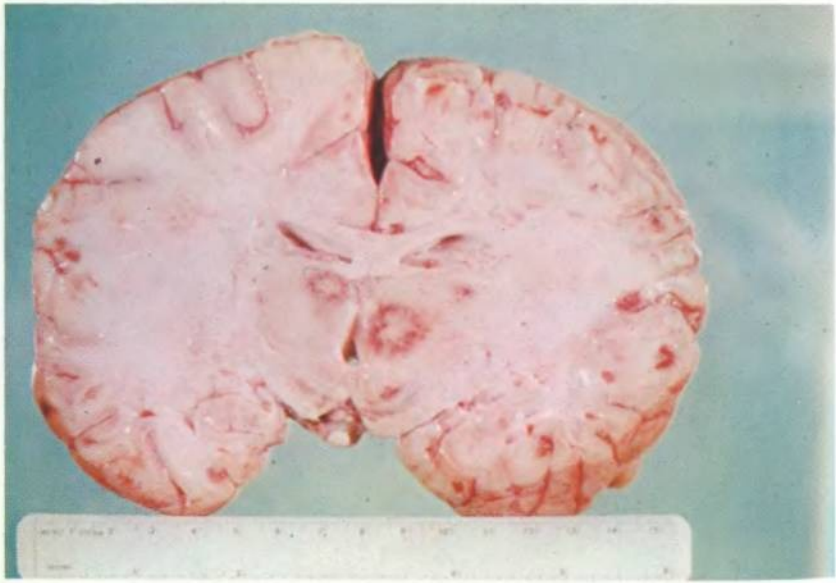
527 Fatal toxoplasmosis relapse secondary to immuno-supression Brain from a 64-year-old woman who received immuno-suppressive therapy for multiple myelomatosis (Case 3 of Frenkel *et el, Human Path* 1975, **6**, 97).

528 Fundal changes in congenital toxoplasmosis Congenital toxoplasmosis may produce choroidoretinitis in later years. This painting is of a macular lesion showing ectopic choroidal pigmentation. Defective vision and squint may result.

529 Histology of lymph glands Acute infection of older individuals may be cryptic, or it may produce a prolonged low fever with lymphadenopathy. Lymph node biopsy shows follicular hyperplasia with active germinal centres containing large, pale histiocytes. These contain ingested plasma cells, not parasites, but *T. gondii* may be isolated from such biopsies by inoculation into mice. *(× 20)*

530 Cystozoites in *Toxoplasma* cysts in brain section (phase contrast) As the individual develops a measure of immunity, *T. gondii* produces typical cysts which contain numerous trophozoites formed by endodyogeny. These resting stages produce no cellular reaction in the surrounding brain tissue. *(× 20)*

527



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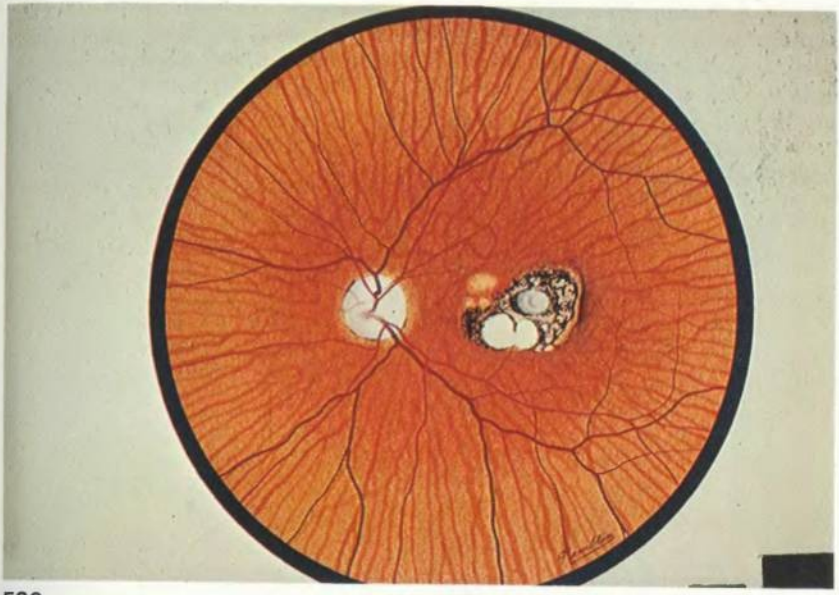

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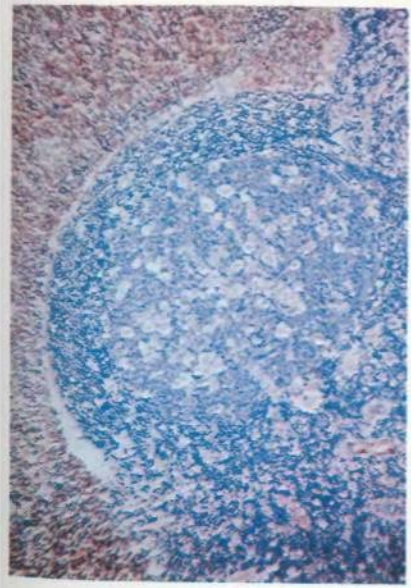

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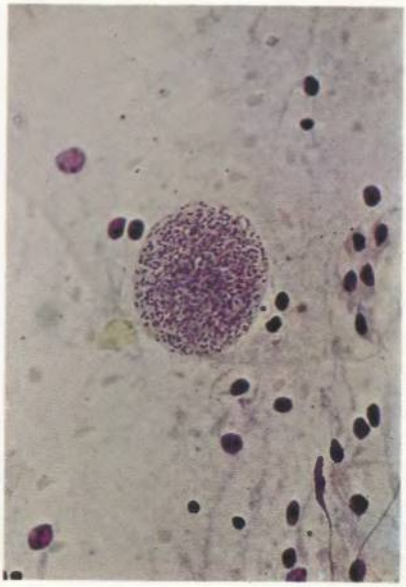
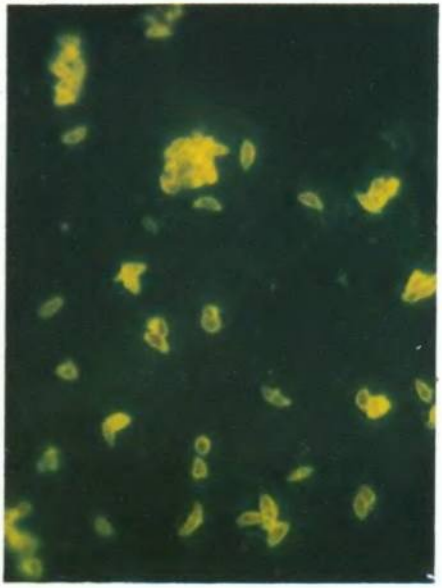


531a & b Immunofluorescent test for toxoplasmosis (Negative control **531a**; positive **531b**.) The Sabin Feldman dye test is also commonly employed. Methylene blue stains *T. gondii* trophozoites obtained from mouse peritoneal exudate, but in the presence of antibody-containing serum the uptake of dye is inhibited by 50% or more. High titres occur in acute infections but a low positive titre remains indefinitely. Other useful serological tests are the CFT, direct agglutination, and toxoplasmin skin test.

531a

531b



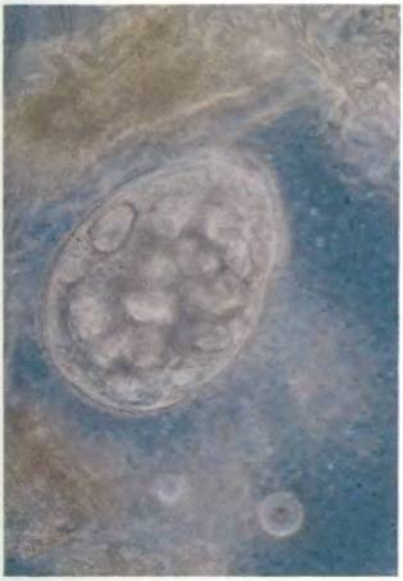
Balantidiasis

Balantidium coli is a common commensal of the large intestine of wild and domestic pigs, but is pathogenic to man and other primates in which it causes severe diarrhoea. Active trophozoites and cysts are readily seen in fresh faecal specimens.

532 ***Balantidium coli*** Living trophozoite in gut of pig. *(× 350)*

533 Section of intestine Extensive ulceration of the colon and rectum may occur in severe cases. *B. coli* trophozoites are seen infiltrating the submucosa in this section of human colon. *(× 90)*

532



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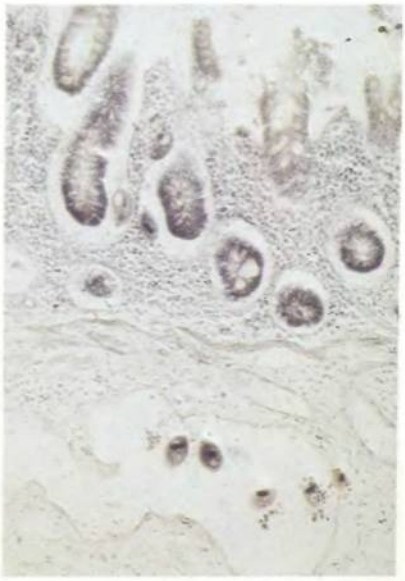

Trichinosis

534 & 535 ***Trichinella spiralis*** Larvae in meat crush preparation (**534**) and free in stomach (**535**). Trichinosis in man commonly results from eating raw or inadequately cooked pork or pork products, such as sausages. Pigs usually acquire the infection by eating infected rodents. The disease often occurs in small outbreaks traceable to a single source. *(× 550)*

536 Parasitic female *T. spiralis* Larvae excyst in the small intestine and develop into minute adults in the mucosa. Mature females deposit larvae which, some five days after infection migrate through the tissues to reach muscles in which they again encyst. Larviposition may continue for a week or more. Finally the larvae become calcified. *(× 60)*

537 Bush pig A common animal reservoir of infection in the tropics is the bush pig. The flesh of other carnivores such as the bear may also infect man.

534

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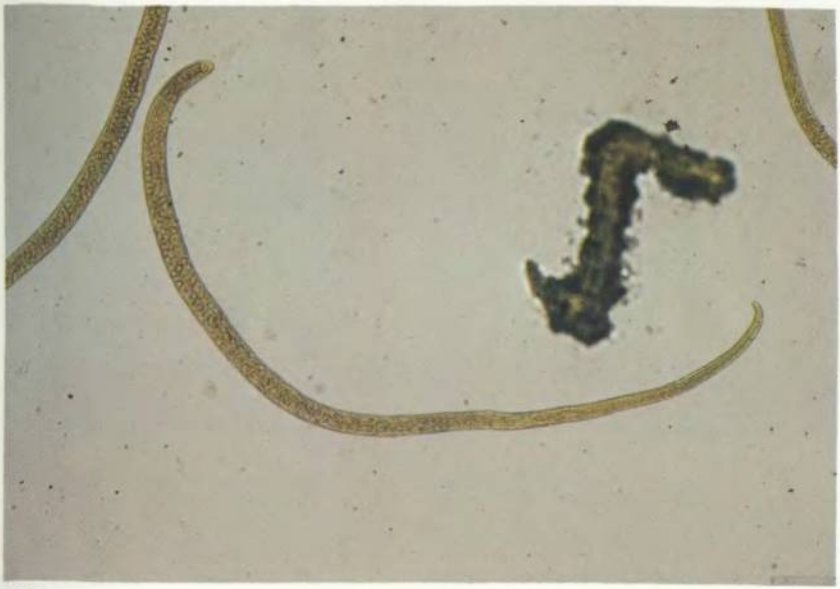


537

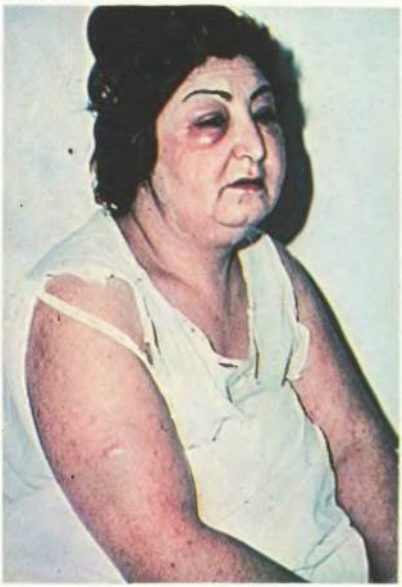
538 Patient with acute trichinosis The four cardinal features of the disease are fever, orbital oedema, myalgia and eosinophilia.

539 Fluorescent antibody test The gel-diffusion test has also proved a useful diagnostic procedure. Other serological tests are available for the detection of humoral antibodies which reach high titres in the acute stage, but are not protective. (Immunity is largely cell-mediated.) (*Bottom:* negative control)

540 Larvae in muscle of fatal human case Encysted larvae are found in the muscles at biopsy (or post mortem). Calcification of the encysted larvae occurs in about 18 months and may be detected on X-ray, but the encysted larvae remain alive for years. *(× 20)*

* (*See* **Table VIII** for classification and figs **293–307** for eggs.)

538



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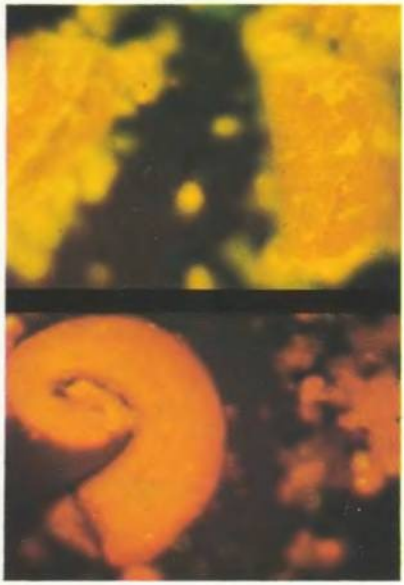

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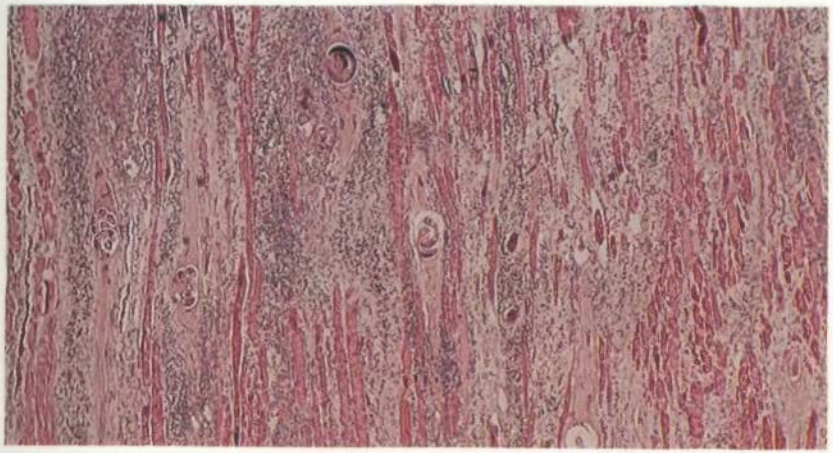
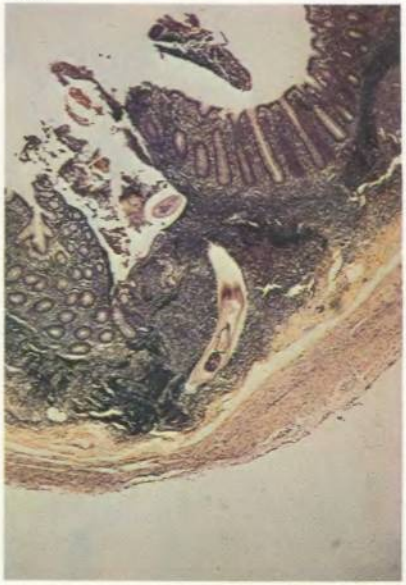


Enterobiasis

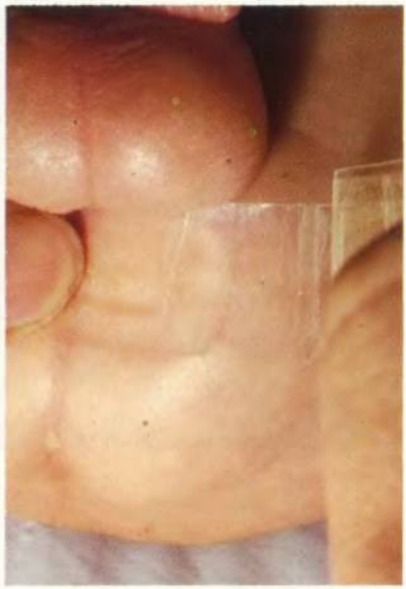
541 Adult pinworms in appendix *Enterobius vermicularis* are small, white and threadlike. The males have coiled tails and measure 2.5 mm. The females which measure about 10 mm long, emerge to the perianal region where they lay some 10 to 15000 eggs then die. The embryonated eggs are directly infectious on ingestion, hatch in the duodenum, and the larvae pass to the caecum where they mature. Occasionally the adult worms cause appendicitis. *(× 20)*

541



542 & 543 Scotch tape swab to demonstrate perianal eggs The eggs are found on the perianal skin. They adhere to the Scotch tape which can then be placed on a slide and examined directly under a microscope. Because of severe pruritus ani, children frequently reinfect themselves from eggs under their fingernails. Bedding is also a source of infection which tends to persist in households and institutions such as orphanages.

542



543

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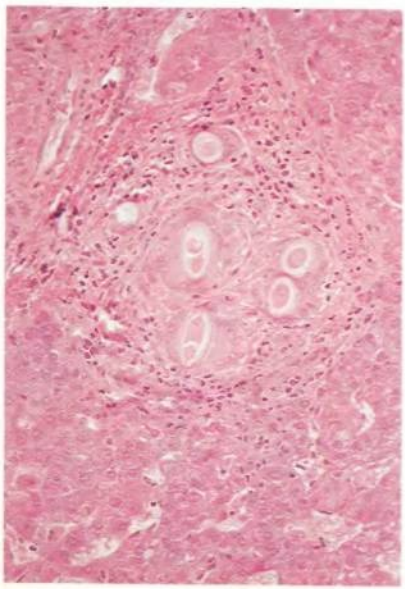
545

Capillariasis

546 *Capillaria hepatica* in liver section Human infection with this rodent nematode is very rare. The adult worms live in the portal tracts. Eggs, resembling those of *Trichuris* (*see* **302**), are infective only after undergoing maturation in the soil. Adult worms in the liver cause hepatic enlargement and severe parenchymal damage. Note the giant cell reaction around the eggs. *(× 150)*

547 Egg of *C. philippensis* This species in which the adults live in the upper small intestine has occurred in epidemic form. A malabsorption syndrome, sometimes fatal, develops in heavy infections. The life cycle remains unknown. *(× 350)*

546

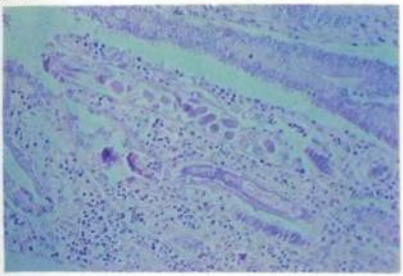


547

548 Adult *C. philippensis* invading small intestine *(× 100)*

549 & 550 Adult *C. philippensis* from human faeces The figure shows the posterior end of the male and vulval region of the female. *(× 150)*

548



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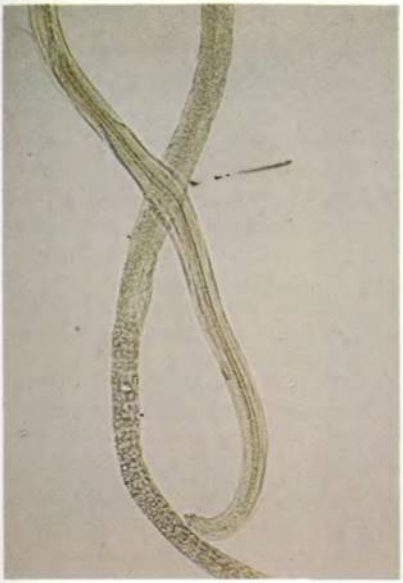
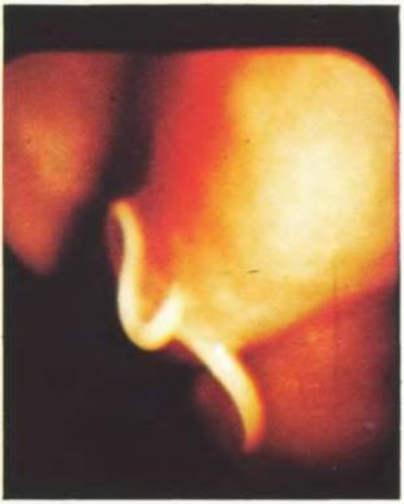


550

Anisakiasis

551 Larval *Terranova* *Anisakis* and *Terranova* species are ascarid parasites of marine mammals. The larvae, about two cm long, found in fish may infect man from the ingestion of raw fish meat ('Herring worm disease'). Various intestinal symptoms may develop depending upon the location of the invasive larvae. The figure shows a *Terranova* larva as seen by gastroscope in the stomach wall of a 53-year-old Japanese woman.

551



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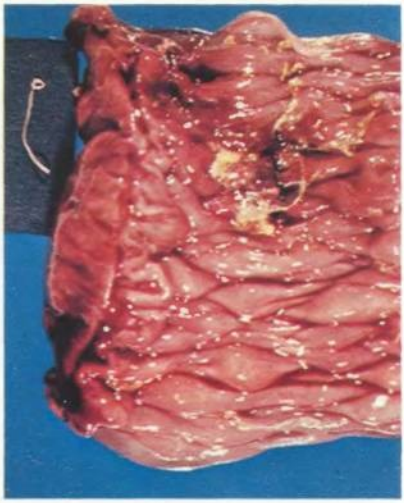


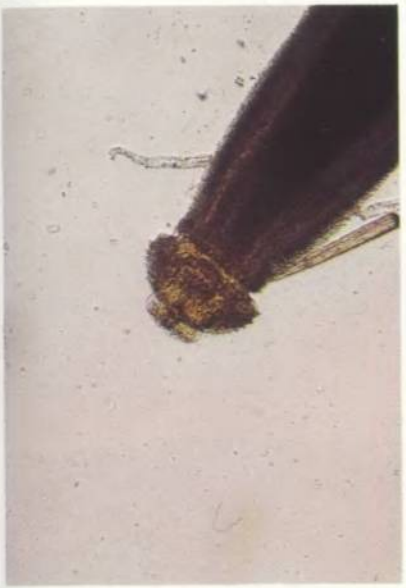
552 Larva removed from small intestine Larval fish ascarid that produced ulceration of the intestine of another Japanese patient.

Gnathostomiasis

553 Head of adult *Gnathostoma spinigerum* The adult is about two to three cm long and usually lives in the stomach of dogs, cats and wild felines. It is found throughout Southeast Asia. There are two intermediate hosts. Man acquires infection by eating fermented fish, a delicacy in Thailand, or any other form of raw fish. The parasite cannot mature in man, but migrates causing cutaneous and visceral larva migrans. *(× 30)*

554 Second stage larva in *Cyclops* Larvae hatching from eggs passed with faeces into fresh water, infect *Cyclops* water fleas, and later fish that eat the *Cyclops*. Here the third stage larva develops. *(× 90)*

553



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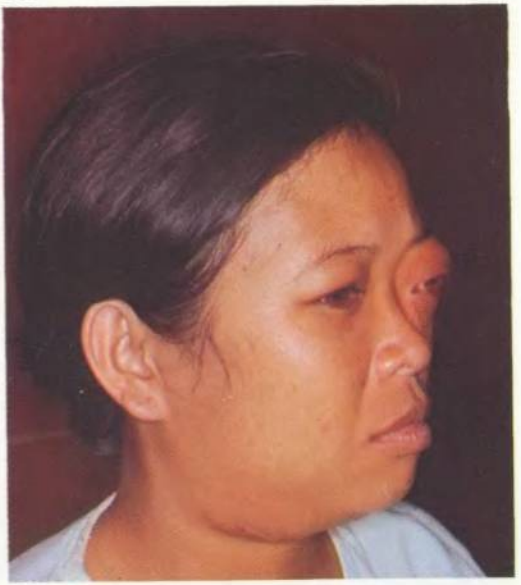


555 Periorbital larva migrans One of the characteristic clinical features is a migrating subcutaneous swelling associated with boring pain and eosinophilia. Third stage larvae may be recovered surgically from swellings in suitable locations. Cerebral lesions with focal signs are not uncommon in Thailand.

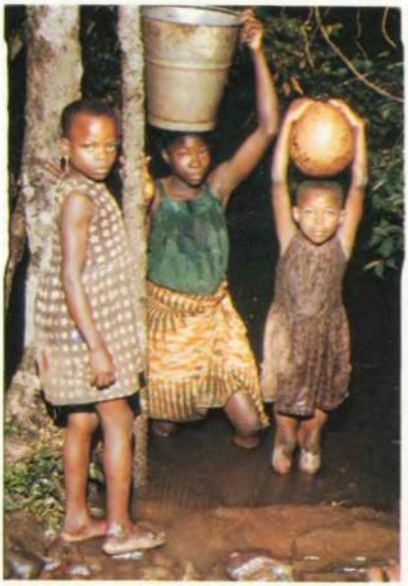
Dracontiasis (Guinea Worm Infection)

556 Insanitary water supply being infected Note the open infective lesions on this girl's foot. Guinea worm occurs in parts of Africa, India, the Middle East and Brazil where water is drawn from shallow pools or primitive wells. Larvae are swallowed inside water fleas when unboiled water is drunk. The larvae emerge in the intestine from where they migrate to, and mature in the subcutaneous tissues.

557 Larva of *D. medinensis* The free-swimming first stage larvae enter water fleas of *Cyclops* and allied genera. Here they develop and await ingestion by a new definitive host. *(× 60)*

558 Contamination of surface water A papule forms where the female reaches the skin surface, and ulcerates when the skin is immersed in water. A loop of the worm's uterus prolapses and ruptures, releasing large numbers of rhabditoid larvae into the water.

556



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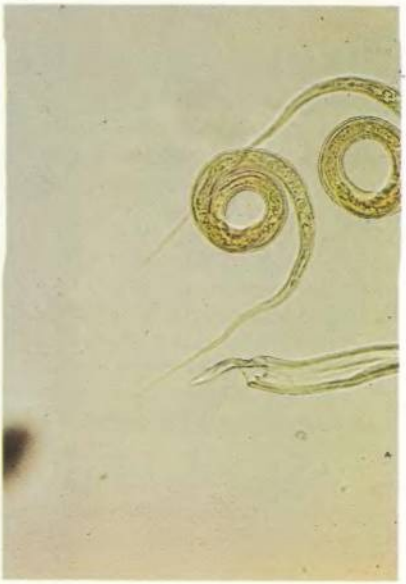

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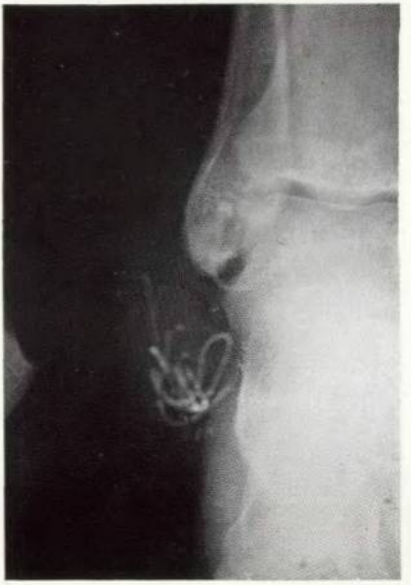
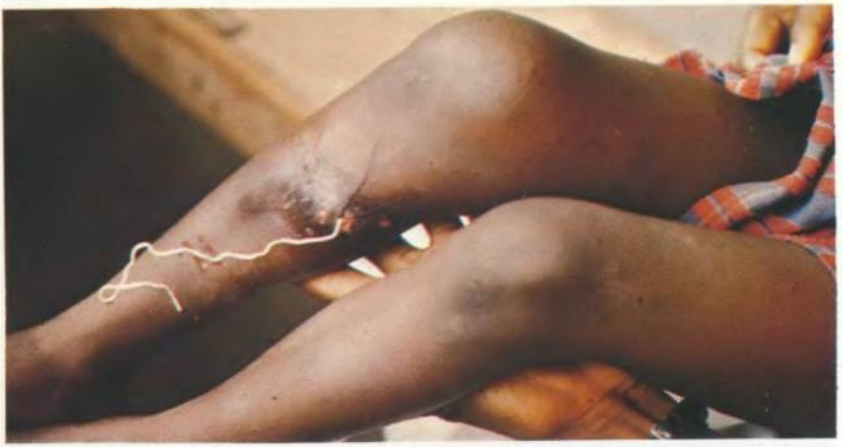


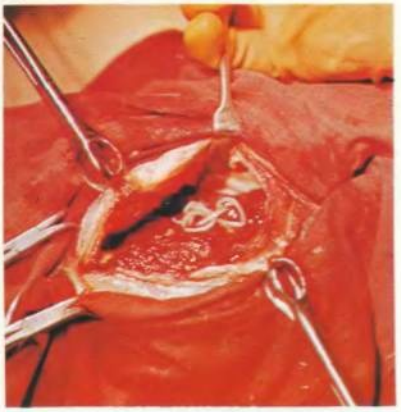
559 X-ray of calcified worms The size of an adult female *Dracunculus medinensis* can be judged from this X-ray of a calcified worm in the ankle. Females attain up to 100 cm but males only 20 to 40 cm in length.

560



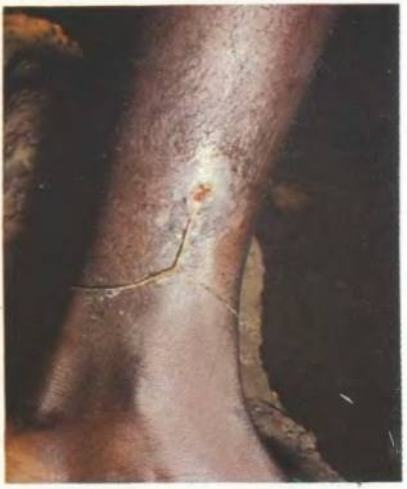
560 Adult guinea worm in knee joint Heavy infestations may cause considerable disability and arthritis may be caused by female worms in the vicinity of joints.

561



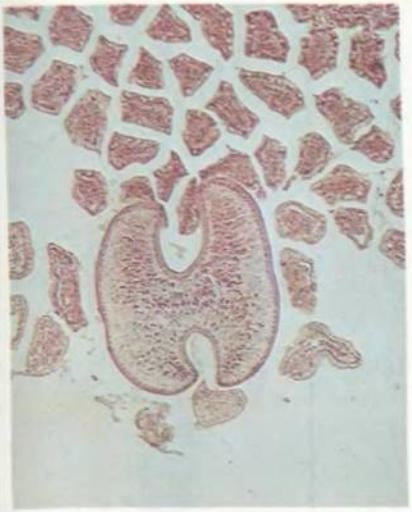
561 Operative removal of worm from knee.

562



562 Extraction of female worm Adult females are commonly extracted by progressively winding them round a matchstick as they emerge from the subcutaneous tissues. Chemotherapy has made the procedure easier and less hazardous.

563



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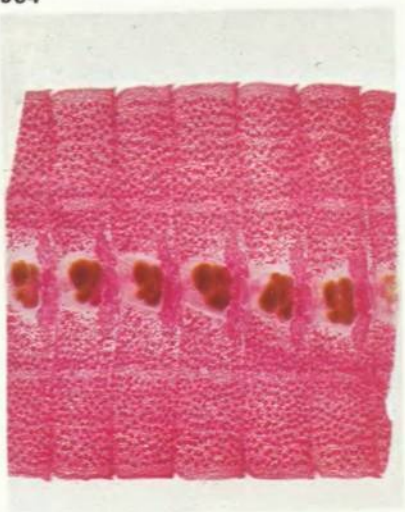


563 & 564 Cross section of head, and segments of adult tapeworm The fish tapeworm, *Diphyllobothrium latum*, is common around large lakes in Europe, North America and elsewhere. A mature adult may reach 10 m in length. The head and mature proglottids have a typical morphology. Mature worms may produce one million eggs daily.

565 Procercoids in tail end of *Cyclops* water flea From the operculated eggs actively swimming round hexacanth coracidia emerge. After ingestion by water fleas (*Cyclops* and others) they form procercoid larvae in the haemocoel of the copepods. *(× 90)*

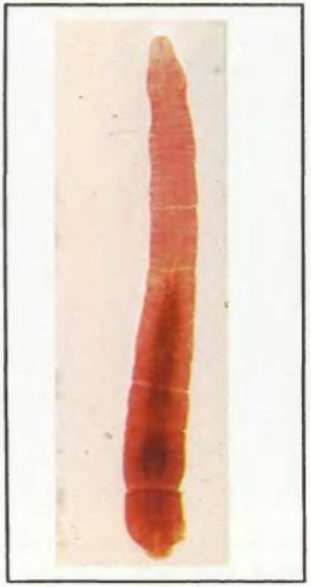
565

566 Plerocercoid in fish When the host is ingested by a fish, the larva emerges to form a migrating plerocercoid (sparganum) which comes to lie in the muscle. These develop into adults in the gut of man if the fish is consumed incompletely cooked. *(× 6)*

567 Macrocytic anaemia in blood film The classical clinical feature of the infection is the development of Vitamin B_{12} deficiency with a resultant macrocytic anaemia. *(× 1250)*

*(*See* **Table XIII**)

566



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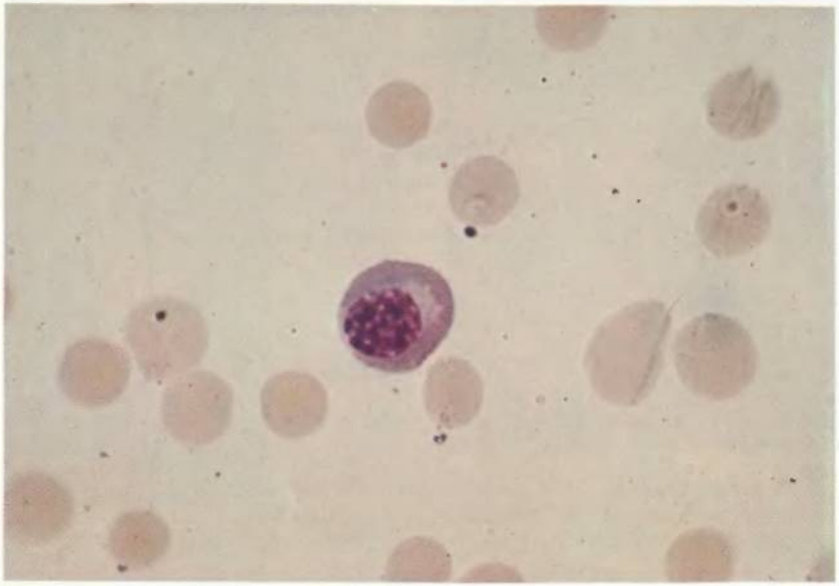


Sparganosis

568 Edible frog and sea food on a Thai market stall Sparganum occur in a variety of amphibious animals including frogs, and these also may be infective to man if ingested. They are the larvae of tapeworms of the genus *Spirometra* that are common in various canines and felines. The first stage larvae are formed as procercoids in *Cyclops*. Ingestion of these larvae produces sparganosis in man, since the larvae cannot mature in this abnormal host.

568

569

569 'Sparganum mansoni' The sparganum larvae proliferate, often in the subcutaneous tissues, where they may become encysted in large nodules from which they can be removed surgically. When localised in the periorbital tissues or under the conjunctiva, severe oedema may result. Surgical removal of the larvae is necessary. This condition in Vietnam and Thailand may follow the application of frogs as a poultice for inflamed eyes! *(× 3)*

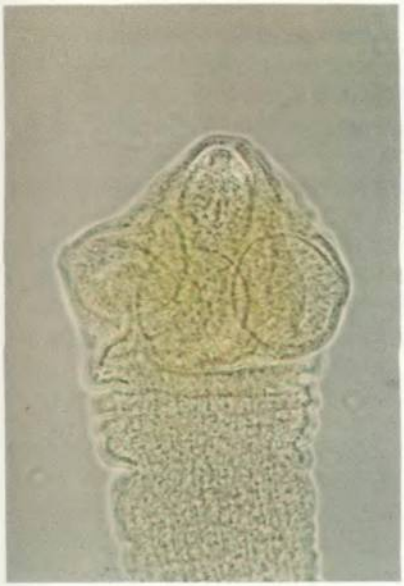
Dipylidium caninum (Dog Tapeworm)

570 & 571 Scolex, and egg capsules in mature segment The adult *D. caninum* which reaches a length of 10 to 70 cm, is the common tapeworm of domestic and wild canines and felines. Eggs in which larval hooks may be seen are passed in clusters in capsules as the mature segments disintegrate. *(× 9) (× 15)*

572 Dog flea larva and adult The eggs are ingested by larval dog, cat or human fleas. They develop first procercoid, then cysticercoid larvae in the haemocoel of the insects in which they remain when the fleas grow to the adult stage. Man becomes infected by accidentally swallowing infected fleas. *(× 15)*

573 Immature cysticercoid from dog flea *(× 90)*

570



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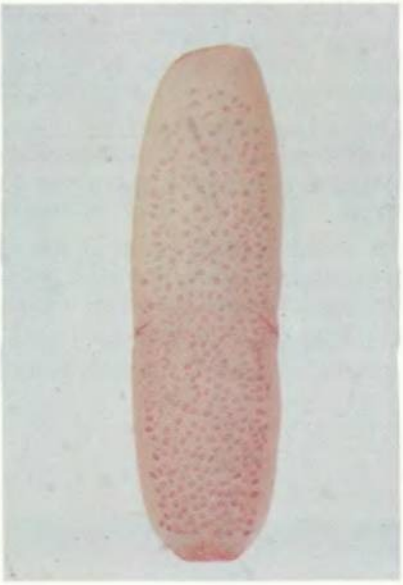

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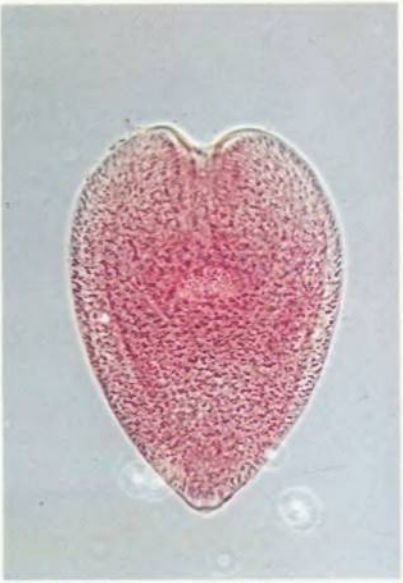

Hymenolepis nana (Dwarf Tapeworm)

574 Hexacanth oncosphere This cosmopolitan tapeworm reaches only 2.5 to 4 cm in length and lives in the small intestine of man and rodents. Infection is acquired directly by ingesting eggs. After ingestion by a mammal or by certain insects the eggs (*see* **304**) hatch into the hexacanth oncospheres. *(× 250)*

575 Cercocyst in intestinal villi In the mammal these penetrate into the villi of the small intestine. There they mature into tailless cysticercoids (cercocysts) which leave the villi, move further down the gut and become attached to other villi, where they mature to adult tapeworms. *(× 100)*

576 Cysticercoid in insect If the egg is eaten by an insect the oncosphere metamorphoses to form a tailed cysticercoid in the insect's body cavity. Further development takes place if the insect is ingested by man. *(× 60)*

577 Mature proglottids Man is probably the only source of human infection, rodents being infected possibly by a different (non-human) strain. *(× 15)*

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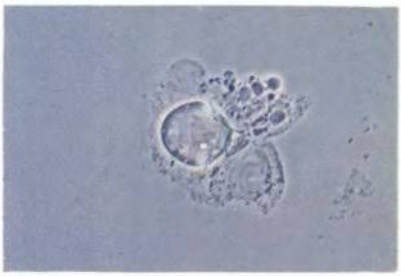

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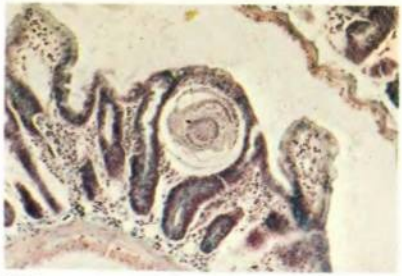

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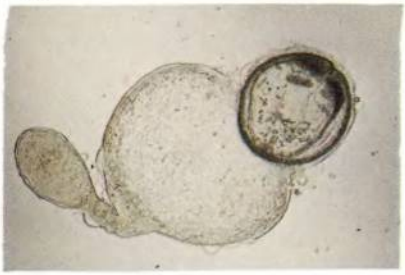

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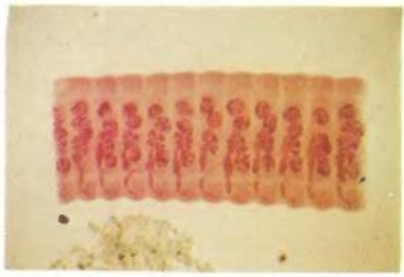
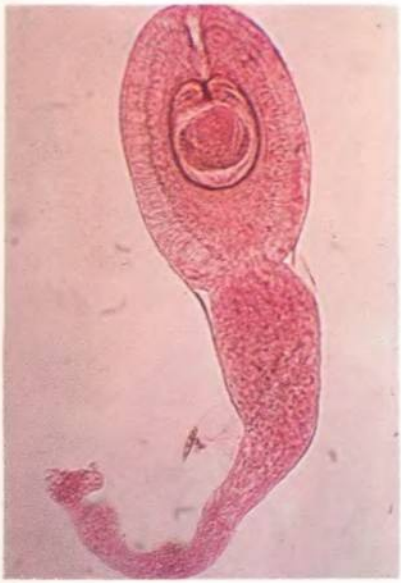


Hymenolepis diminuta (Rat Tapeworm)

578 Cysticercoid of *H. diminuta* in arthropod This is the common cestode of rodents. Adults measure 20 to 60cm in length; their eggs resemble those of *H. nana* (*see* **305**) but lack the polar filaments. An intermediate insect host is essential in this life cycle and a wide variety of coprophagic arthropods serve as intermediate hosts. Occasionally man is infected by accidentally swallowing infected arthropods, eg rat fleas, or larvae of grain moths and beetles. *(× 90)*

579 *Tribolium confusum* These grain pests are typical intermediate hosts of *H. diminuta*. *(× 9)*

578



579

Taenia solium (Pork Tapeworm)

580 Head and part of segments of adult tapeworm The adult worm may attain two to seven m in length. *(× ⅓)*

581 Cysticercus in pork The larval cysticercoid stage occurs in the pig. Man is infected by ingesting uncooked pork. *(× 3)*

582 & 583 Scolex of *T. solium* and *T. saginata* The head of *T. solium* is armoured with hooks in addition to four suckers. Its gravid segments (*see* **588**) contain a central uterus with less than a dozen lateral branches. The eggs are similar to those of other *Taenia* species. *(× 40)*

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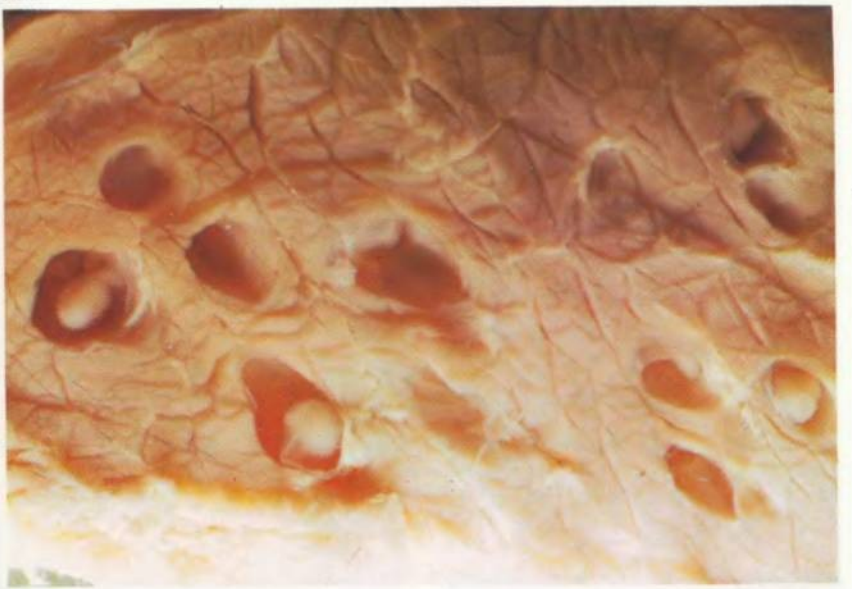

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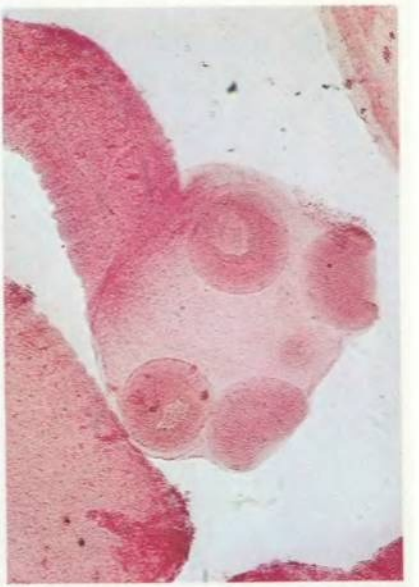


584 X-ray of cysticercosis of soft tissues The cysticercoid stage of *T. solium* (Cysticercus cellulosae) can also occur in man if eggs are released in the intestine, sometimes following incorrect use of taenicidal drugs. When calcification occurs the cysticerci are seen by X-raying the soft tissues adjoining the thighs and shoulders.

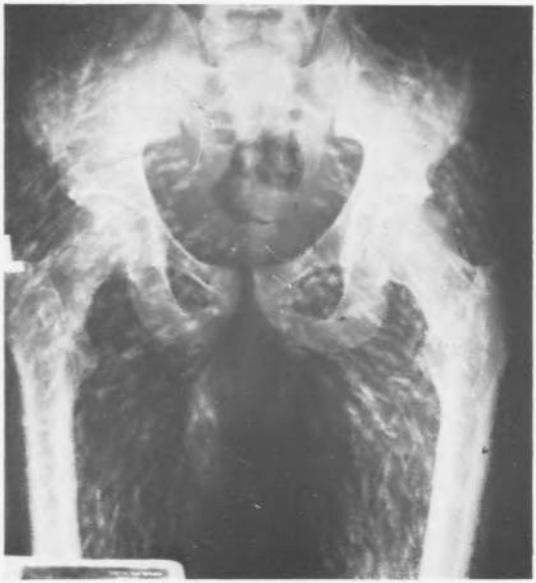
585 Section of cyst in human muscle Cysticerci lodging in muscle or subcutaneous tissues can occasionally be palpated. The figure shows a section of a typical Cysticercus cellulosae removed from a chest wall. *(× 20)*

586 Cysticercosis of brain When cysticerci lodge in the brain calcification can occur, but usually much later than in the soft tissues, and often after epilepsy has manifested itself.

Taenia saginata (Beef Tapeworm)

587 & 588 Mature proglottids of *T. saginata* and *T. solium* The head of *T. saginata* (*see* **583**) is provided with four sucking devices but no hooks as in *T. solium* (*see* **582**) and the segments contain a central uterus with 15 to 20 lateral branches. The adult *T. saginata* attains up to 10 m in length. *(× 6)*

584



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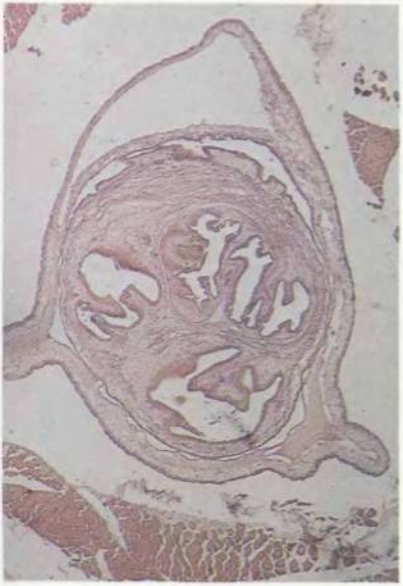

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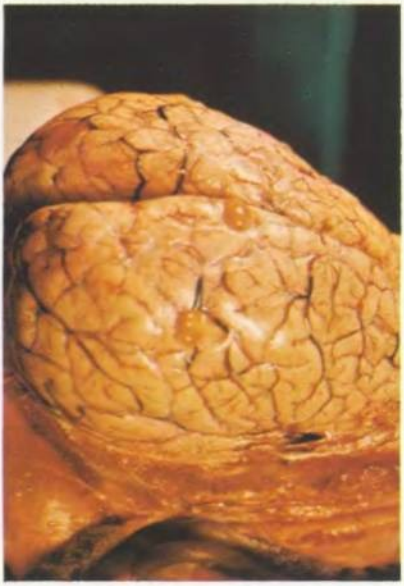

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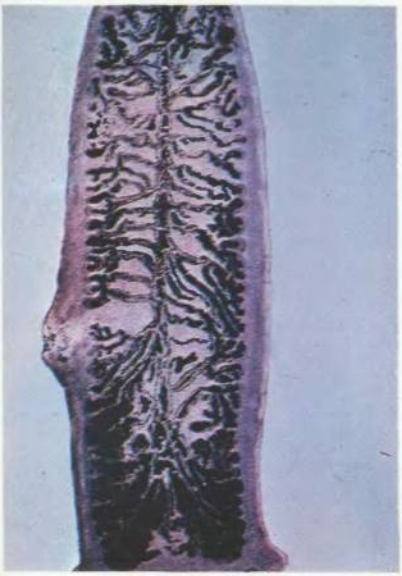

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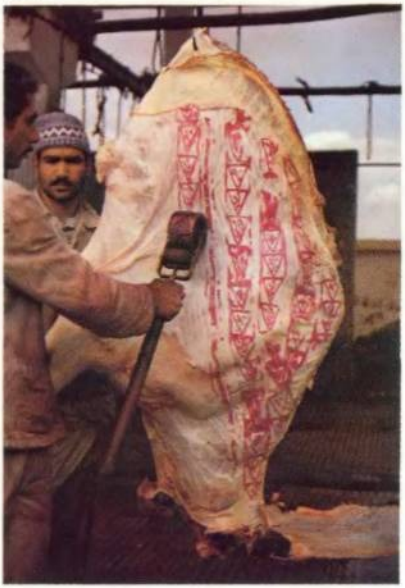


589 Cysticercus in beef The cysticercal stage occurs only in cattle, and man is infected when he eats raw or partially cooked beef.

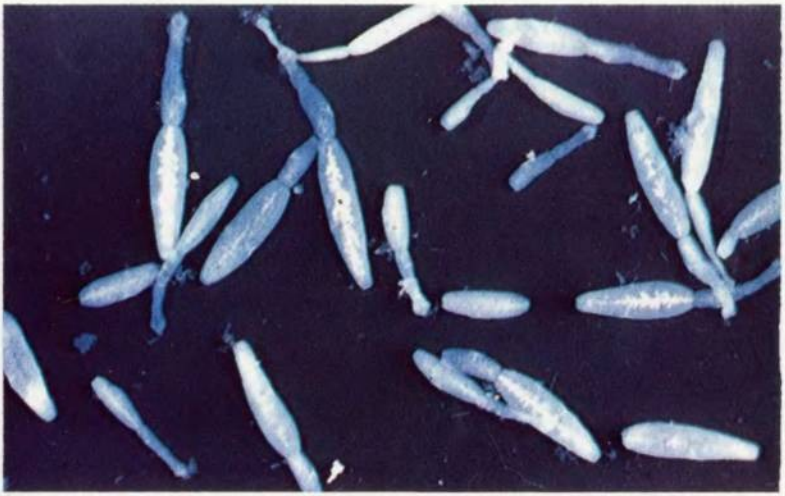
590 Meat inspection – certificate of health Taeniasis is prevented by strict abattoir supervision including adequate inspection of carcasses, and condemnation of 'measly' meat.

Hydatidosis

591 Adult *Echinococcus granulosus* This dog tapeworm is only about five mm long and inhabits the small intestine. Dogs are infected by eating offal of sheep, cattle or pigs containing hydatid cysts. The scolices in the cysts evaginate in the animal's intestine and mature into the adult worms. Typical taeniid eggs (*see* **306**) are passed in the dog's faeces. *(× 9)*

592 & 593 Hydatid cyst of liver From ingested eggs that hatch in the duodenum, the hooked embryos enter the circulation where they are carried to various sites. The liver is commonly affected. The hydatid cyst is usually unilocular, with a double wall comprised of an outer laminated layer, and an inner nucleated germinal layer. The figures show a 1150 g cyst *in situ* (**592**) and after extraction (**593**).

591



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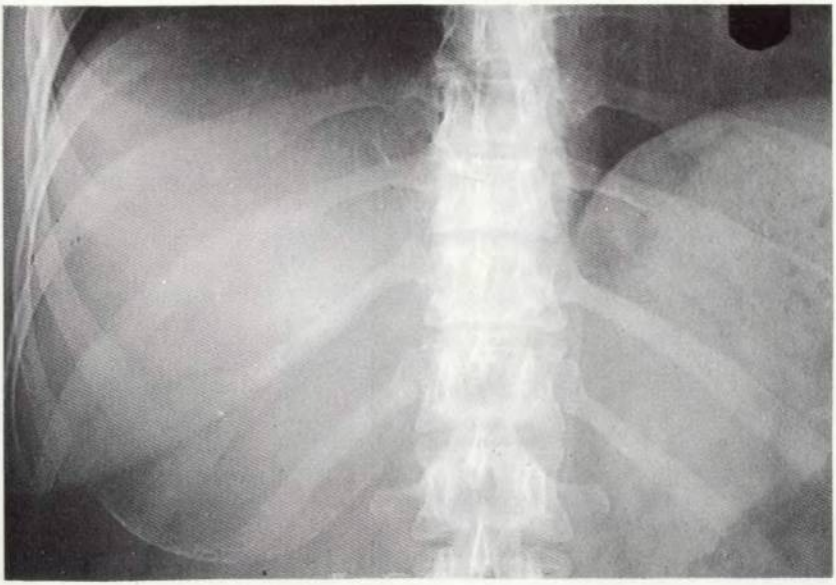

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594 Daughter cysts and hydatid sand The germinal layer produces brood capsules inside which grow scolices. These daughter cysts are attached to the parent cyst wall or may float free in its milky fluid contents as the so-called 'hydatid sand'. Rupture of a cyst into the tissues results in dissemination and further growth of these daughter cysts.

595 Hydatid cyst in brain This cyst was found in the brain of a four-year-old girl.

596 X-ray of lungs Hydatid cysts of the lung are not uncommon.

594

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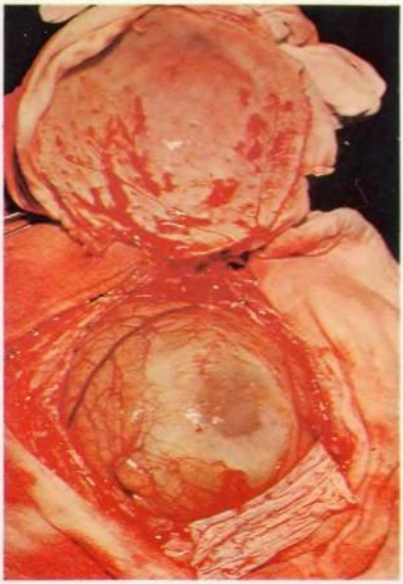

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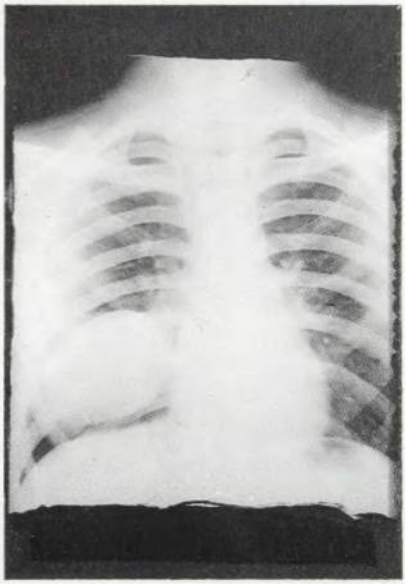


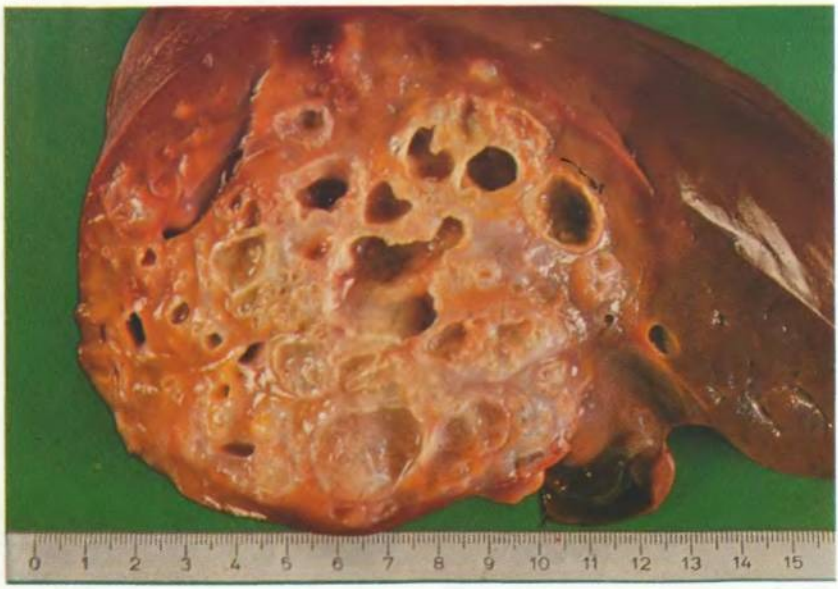
597 Hydatidosis in human liver Multilocular, or alveolar cysts, are caused by infection with *Echinococcus multilocularis*. The adult of this species is found in wild canines, and the usual larval hosts are rodents. The alveolar cyst in the human liver may mimic hepatic carcinoma, but it is usually only discovered at post mortem, as in this case.

598 Section of alveolar hydatid cyst *(× 90)*

599 *Coenurus cerebralis* of sheep This is the larval stage of *Multiceps multiceps*. The adult lives in the intestine of the dog and the 'Bladder worm' larva is usually found in the brain of sheep. Fortunately infection of man is rare.

600 *Coenurus* in human eye *(× 31)*

597



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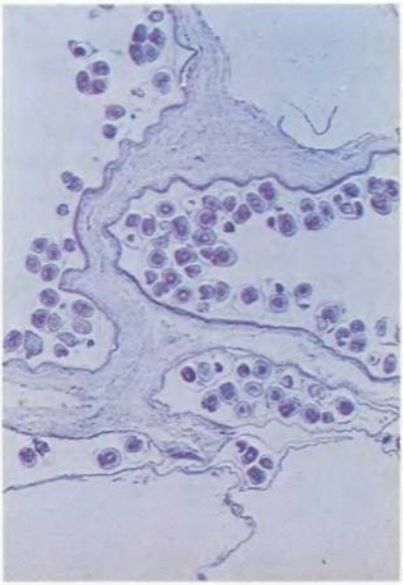

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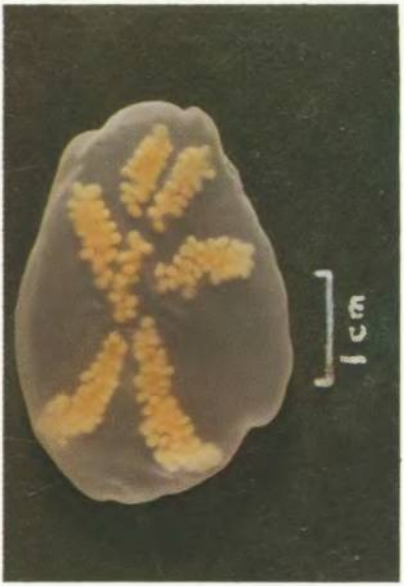

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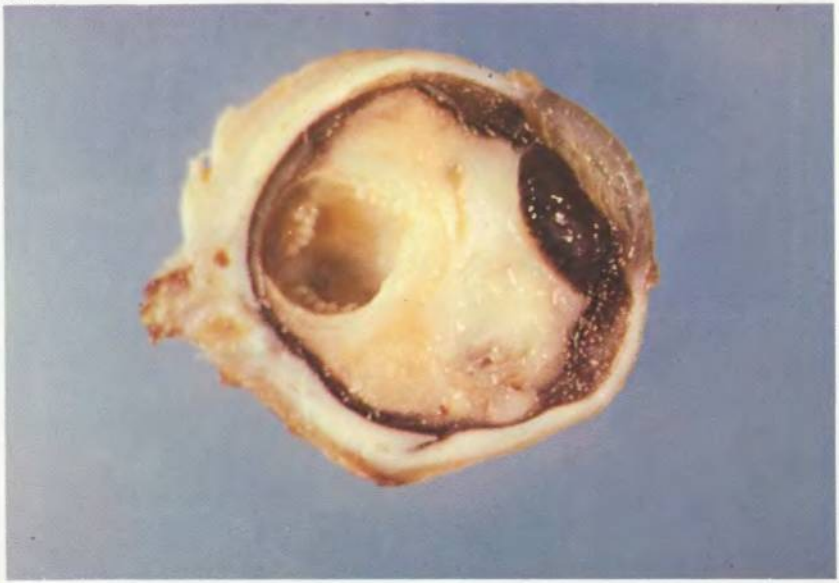

PENTASTOMIASIS

Linguatula serrata (Tongue Worm)

601 Third stage larva in rabbit lung These endoparasitic and highly specialised parasites have embryonic and ultrastructural affinities to the Arthropoda. Man may be infected by eating inadequately cooked food containing third stage larvae. Other carnivores are also infected. *(× 20)*

602 Halzoun syndrome The parasites migrate to the nasopharynx where they produce large adults that block the airways, and cause deafness. Eggs are passed in the nasal secretions. Facial oedema is a common sign.

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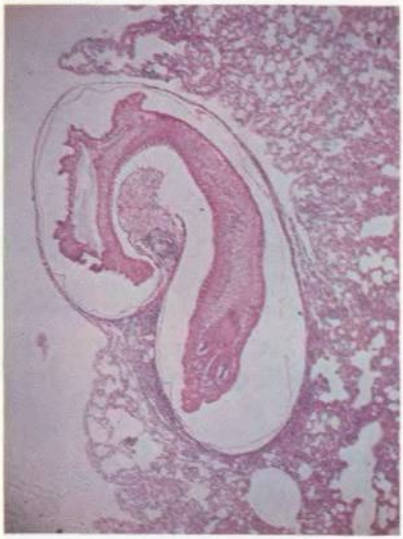

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603

603 Adult *Linguatula serrata* Cephalic third of a typical adult tongue worm. Note the oral opening and four hooks. *(× 1½)*

Other Pentastomids

604 & 605 Eggs and first stage larva of *Porocephalus* The eggs and primary larvae with their four bifurcate legs point to the arthropod origin of this parasite of North American snakes (*Porocephalus crotali*). (**604**: × *100*, **605**: × *160*)

606 *P. crotali* third stage larva After ingestion of eggs in food or water by a secondary host (usually a rodent), primary larvae emerge in the gut. They penetrate the gut wall and encyst in various tissues. This infective third stage larva lies subperitoneally in a rodent, the normal intermediate host. *(× 3)*

607 Adult *Porocephalus crotali* in rattlesnake lung Eggs of this 'lungworm' are passed in saliva or in faeces. This species is common in snakes, which acquire infection by eating rodents containing third stage larvae. *(× $\frac{1}{15}$)*

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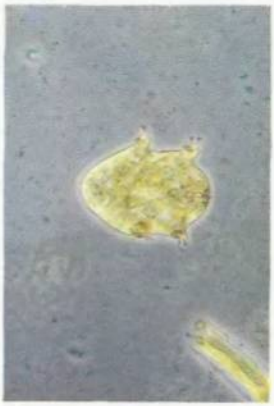

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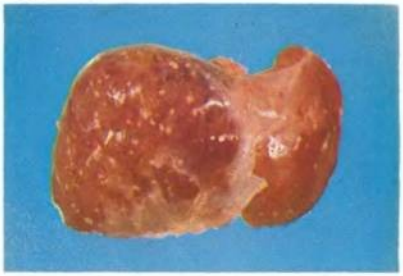
608 Larvae of *Armillifer armillatus* Third stage larvae are seen under the capsule of this liver from a Nigerian at post mortem.

609 Adult males and a female *Armillifer armillatus* This lungworm is a common parasite of several species of African snakes. The usual intermediate hosts are rodents but man is quite commonly infected with the larvae.

610 X-ray of pentastomid larvae in man Calcified third stage larvae are seen in this X-ray of an African patient.

611 Pentastomid larva in eye The eye is a rare site for pentastomid larvae, the abdomen being mainly affected in man.

608



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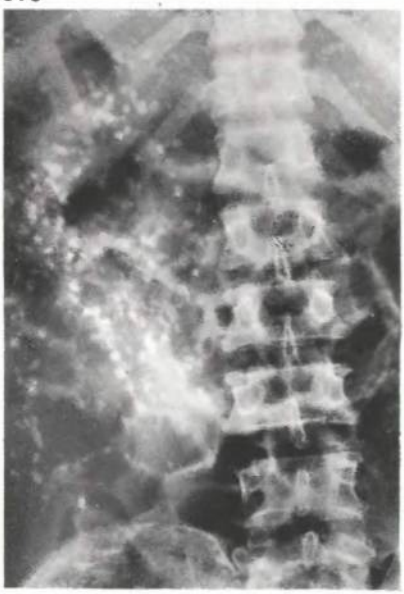

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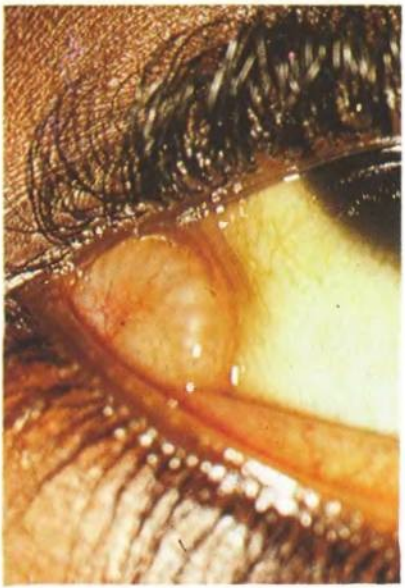

Part V
Infections Acquired through the Skin and Mucous Membranes

The infective agents include viruses, bacteria, protozoa, helminths and arthropods. In one group, transmission of infection is by contact with contaminated persons or objects. In the other group, infection may be acquired by exposure to infected soil (hookworm)*, water (schistosomiasis*, leptospirosis), by the bites of animals (rabies) or through wounds (tetanus). The mode of transmission may be by direct, or by indirect contact.

The smallpox eradication campaign organised by the World Health Organisation has now resulted in the disappearance of the disease. Yaws is now almost a curiosity although sporadic cases still occur. Trachoma however remains an important blinding disease although considerable progress in its control has been made in the Middle East. The venereal diseases are more important in the tropics than has hitherto been appreciated, while the non-venereal treponematoses are widely distributed in the world. The greatest concentration of leprosy is in the Indian sub-continent (three million) while the highest prevalence rates are in Africa (about 100 per 1 000). One fifth of the estimated 15 million cases are under treatment. Europe has 52 000 (mainly in Southern Europe), and over 900 patients have been notified in England since 1951.

A wide variety of fungi infect skin, hair and nails without deeper penetration of the host tissues. Other fungi cause deep mycoses that can result in some of the most disfiguring lesions seen in clinical medicine.

**These conditions are dealt with in Parts II and III respectively.*

Smallpox

612 Ten years ago smallpox was endemic in many countries of the tropics. A successful smallpox eradication campaign was organised under the auspices of the WHO. Global eradication of this disease, has now been confirmed by WHO.

1975 (December) 1967 Eradication confirmed

613 Distribution of rash The distribution of the rash is centrifugal. It is accompanied by marked toxicity.

614 Facial lesions The lesions are concentrated on the face as contrasted with the body.

615 Haemorrhagic smallpox In some cases the lesions become confluent and haemorrhagic.

616 Ultrastructure of smallpox virus The causative agent is a large ovoid DNA virus. This can readily be identified in fluid from the cutaneous lesions. *(× 63 000)*

617 Primary smallpox vaccination response Vaccination immunises the individual for a period of three years.

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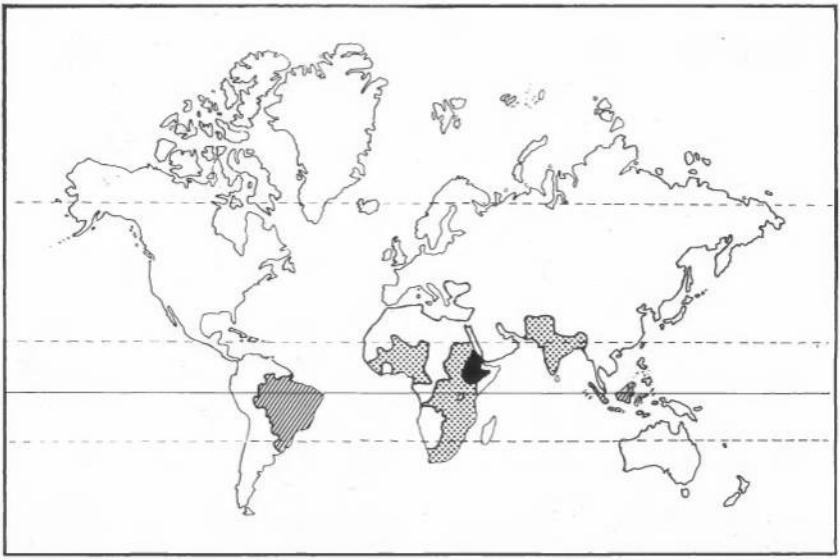

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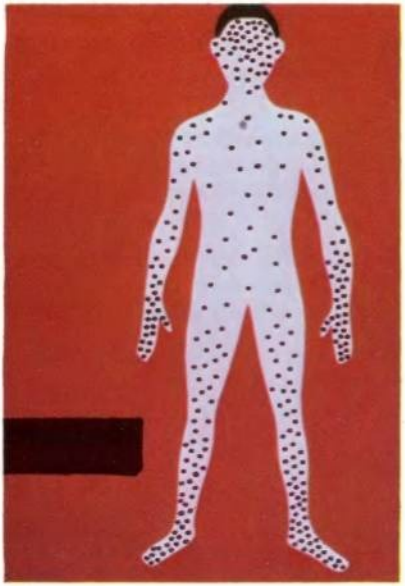

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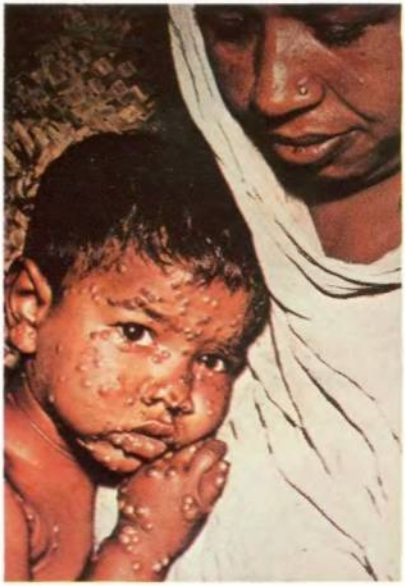

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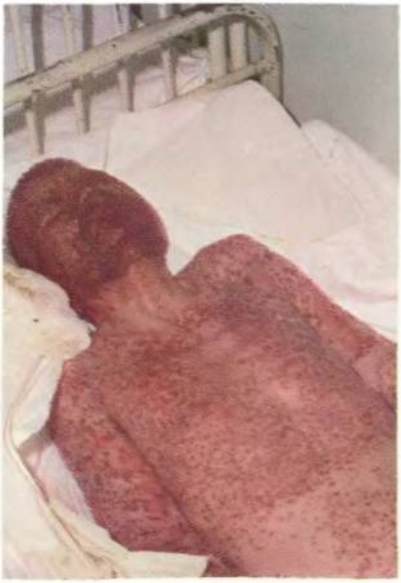

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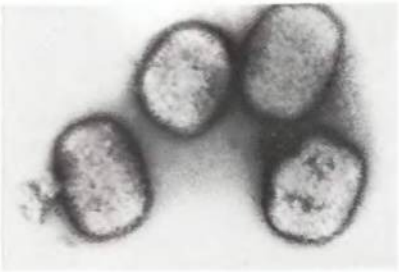

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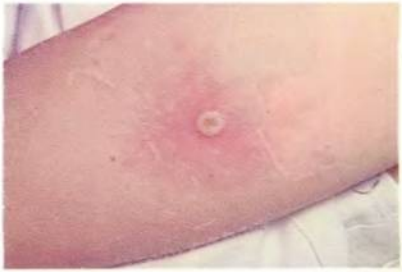

Trachoma

618 Trachoma distribution Trachoma is particularly common in the Middle East and Africa, as well as other parts of the tropics. It is caused by a virus of the *Chlamydia* group (also known as *Bedsonia*). Solid colour indicates high incidence; lines indicate lower incidence.

619 Early lesions Small pinhead-sized, pale follicles beneath the epithelium over the tarsal plates, especially in the upper lid are a characteristic feature of the disease. The so-called 'TRIC' virus may be identified at this stage, and up to the time scarring commences, in epithelial scrapings.

620 Entropion and trichiasis Scarring of the tarsal plates may be extensive and result in entroprion of the edge of the lid. The eyelashes point inwards and rub against the cornea (trichiasis), adding to the damage already done by the virus.

621 Late corneal scarring and trichiasis The end point of trachoma is frequently blindness due to corneal scarring and other complications.

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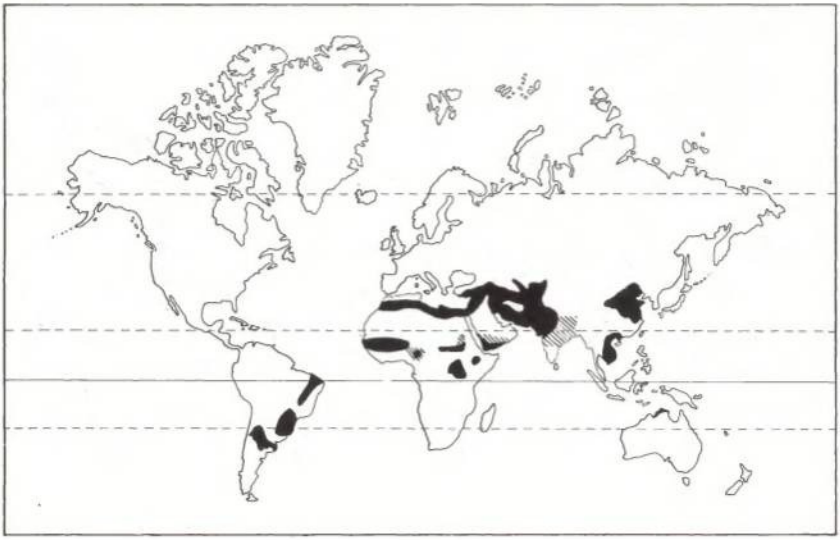

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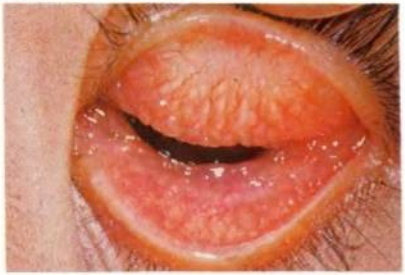

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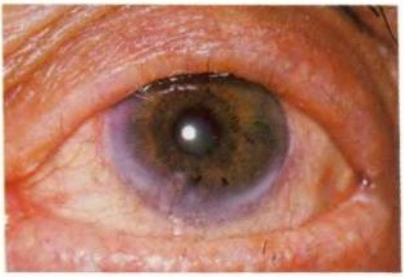

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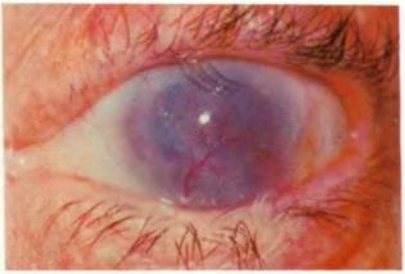

Lymphogranuloma Venereum

622 Inguinal adenitis Inguinal lymphadenitis is a common feature, resulting in a large, sausage-shaped mass, over which the skin is shiny and purplish in colour. The disease is caused by another *Chlamydia* species which can be seen as elementary bodies in Giemsa stained leucocytes. Serious genito-anorectal lesions can result.

623 Frei test The frei skin test is a delayed hypersensitivity reaction of value in the diagnosis. The test is read at 48 hours and 96 hours after the injection of 0.1 ml of the antigen which is prepared from virus cultured in chick embryos. (This disease is also called lymphogranuloma inguinale, climatic bubo, or esthiomène.)

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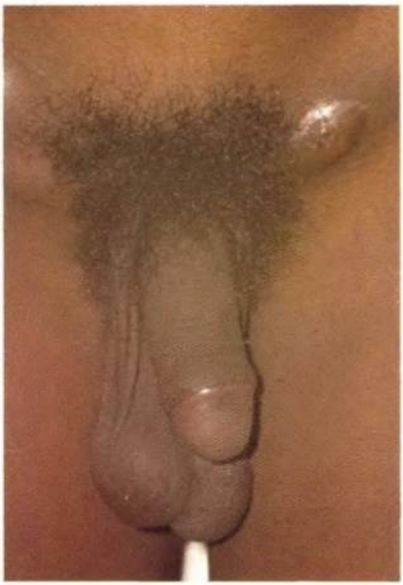

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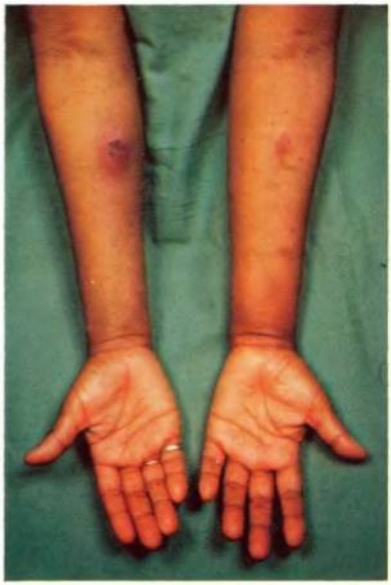


Rabies

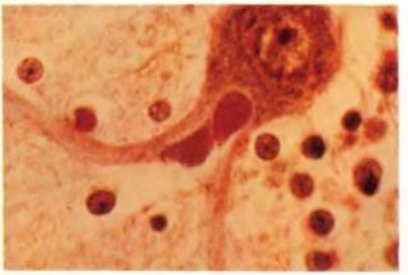
624 Negri bodies in nerve cells of rabid dog Although the dog, and to a lesser extent the cat, is the main urban transmitter of infection, foxes and other feline species as well as vampire bats are natural hosts and may also transmit the disease to man. Intracytoplasmic Negri bodies in brain cells are pathognomonic of rabies. *(× 600)*

Herpes simplex

625 Acute herpetic ulcerative gingivostomatitis This condition due to *Herpes simplex* virus in children with severe protein-calorie malnutrition causes a serious illness seen only uncommonly in the developed world.

626 Liver in disseminated *Herpes simplex* infection Disseminated infection may affect the internal organs, eg liver, brain, heart, etc. This complication is usually fatal.

624



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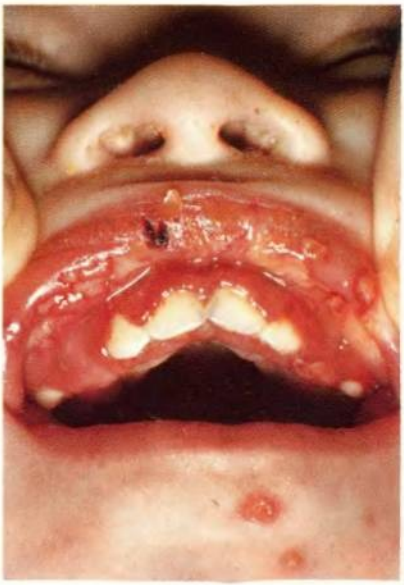

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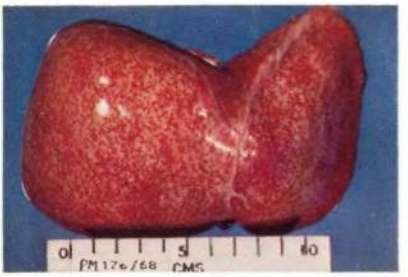


BACTERIAL INFECTIONS

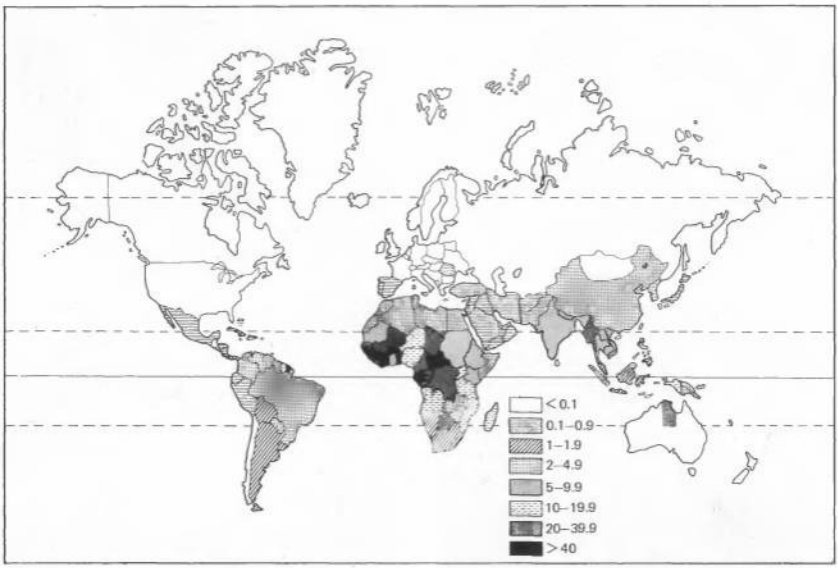
Leprosy

627 Distribution of leprosy Leprosy is particularly common today in Africa, the Indian subcontinent, Southeast Asia and South America. (Rates per 1000 shown on figure)

628–630 Preparation of skin smear A biopsy from a nodule will be smeared and stained with Ziehl-Neelsen stain.

631 & 632 Organisms in skin biopsy stained by TRIFF method *Mycobacterium leprae* is an acid and alcohol fast organism readily seen in sections (**631**) or smears (**632**) of the skin of lepromatous patients. *(× 100) (× 600)*

627



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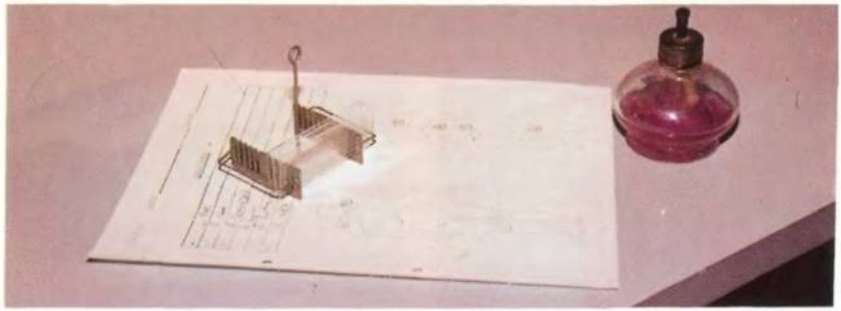

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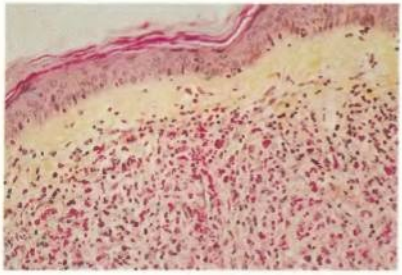

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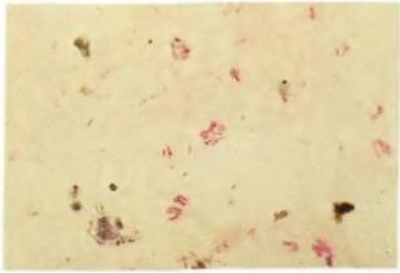


633 Lepromin test The 'Mitsuda' reaction, usually attains its maximum in four to five weeks and indicates the degree of sensitivity to the mixture of antigens (leprosy bacilli) injected. The reaction is expressed in mm with or without ulceration and is read about the 21st day after injection. In patients with lepromatous leprosy the reaction is completely negative. In patients with tuberculoid leprosy it is variably positive.

634 Early macules An early sign of leprosy, the *indeterminate* macule, is slightly hypo-pigmented and ill-defined. It retains tactile-sensitivity, sweating function, and hair growth.

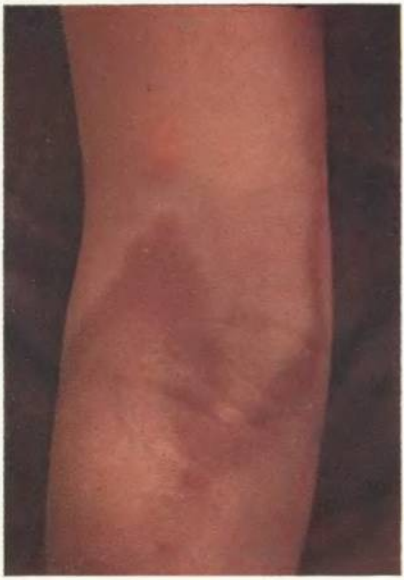
635 Tuberculoid leprosy The early tuberculoid lesion is characterised by macules showing loss of sensation and hypopigmentation.

636 Ulnar nerve lesion Damage to the ulnar nerve in tuberculoid leprosy leads to weakness and wasting followed by complete paralysis, and atrophy of the ulnar-innervated hand muscles, resulting in the characteristic picture of the 'main de predicateur'.

637 Loss of extremities in late tuberculoid leprosy Neurotrophic atrophy eventually leads to the loss of phalanges, especially following trauma resulting from the anaesthesia. This man shows almost complete loss of hands and feet.

638 Nerve thickening Thickening of the great auricular nerve is also common in tuberculoid leprosy.

633



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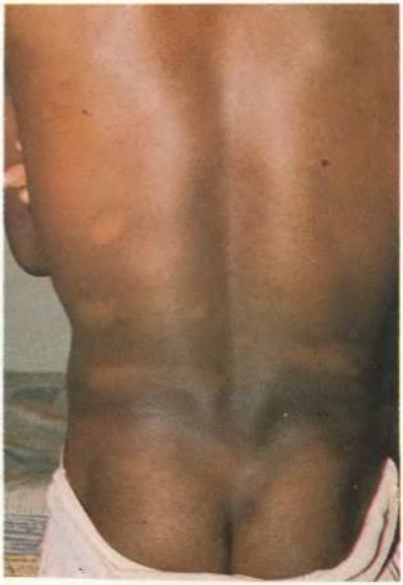

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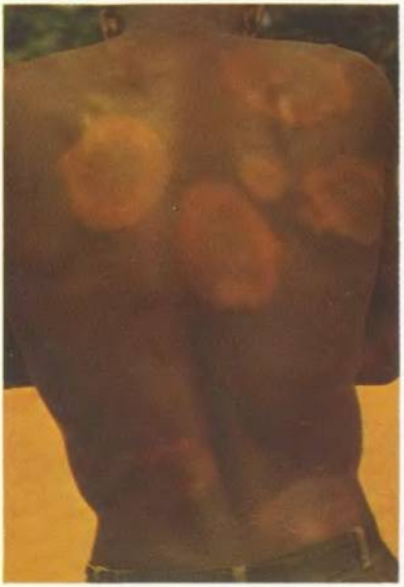

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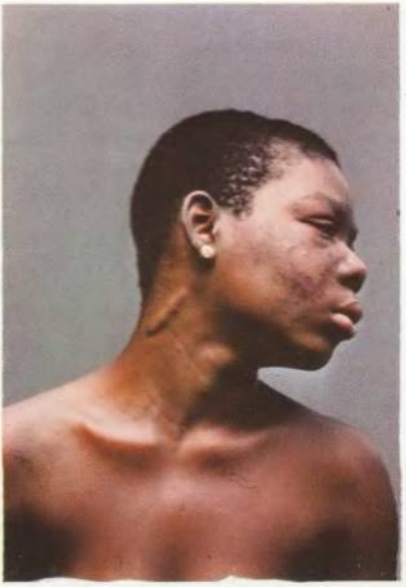

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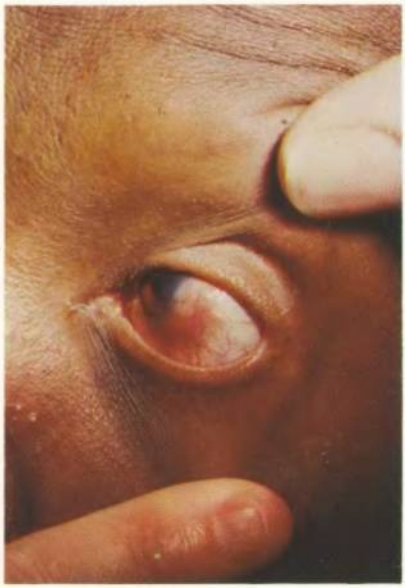


639 Lepromatous leprosy Lepromatous leprosy, showing extensive infiltration, oedema and corrugation causing 'leonine facies'. Note depilation of eyebrows and face, and thickening of ear.

640 Lepromatous nodule in eye Leprosy is a common cause of blindness in the tropics.

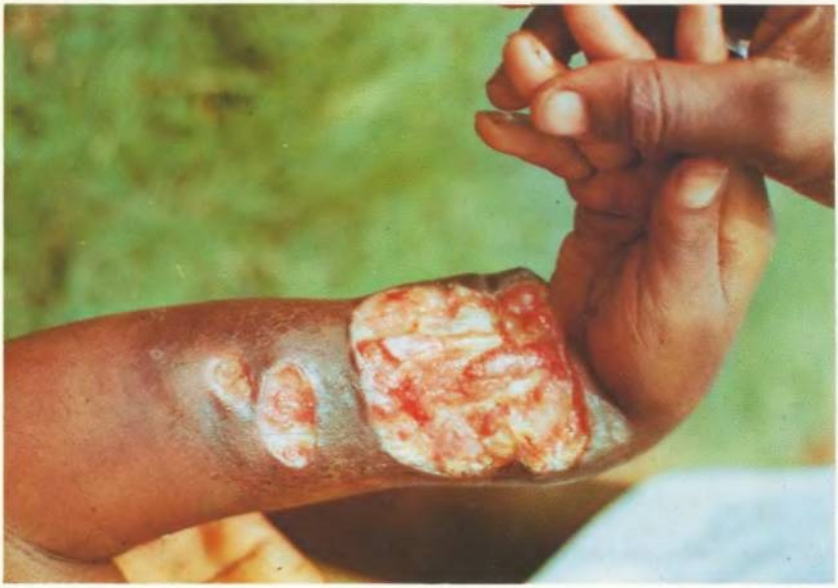
Mycobacterium ulcerans and Other Tropical Ulcers

641 Typical 'Buruli' ulcer in a Nigerian child The condition is characterised by gross, necrotising skin ulcers in which numerous acid fast bacilli are present (*M. ulcerans*). The disease occurs in localised tropical areas in all continents.

642 *M. ulcerans* in section of ulcer Acellular necrosis occurs involving the dermal layers and subcutaneous fat. Acid-fast bacilli are found in the necrotic material. *(× 900)*

643 Tropical (phagedaenic) ulcer Chronic necrotising ulcers involving the skin and subcutaneous tissues are common in country areas in the humid tropics. They contain a mixed bacterial flora including *Borrelia vincenti* and fusiform bacteria.

641



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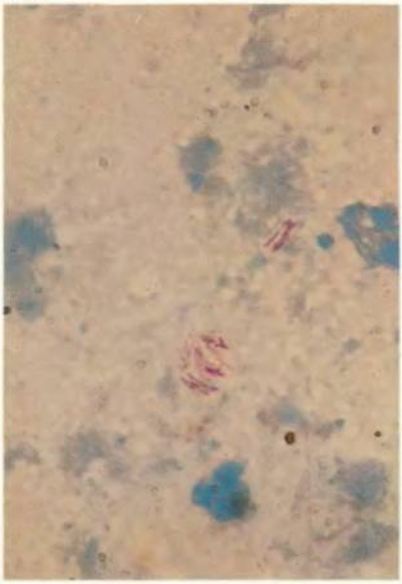

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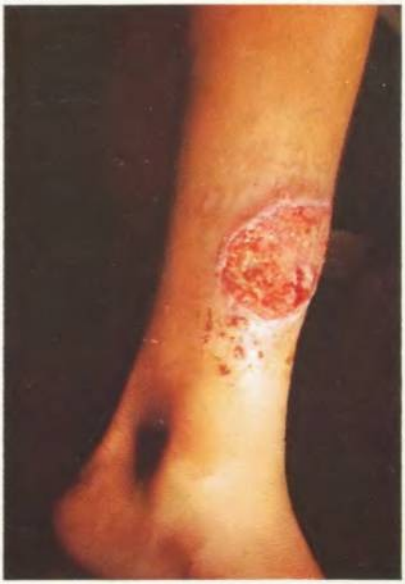

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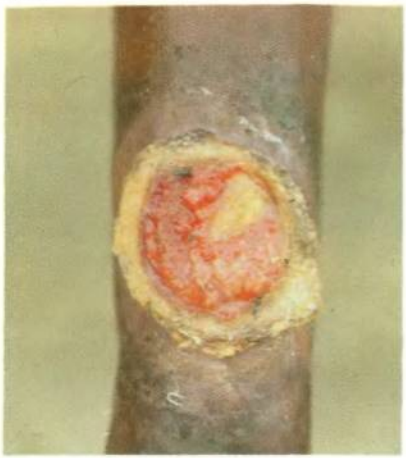

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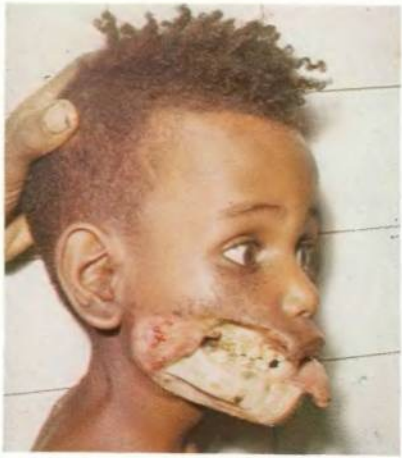
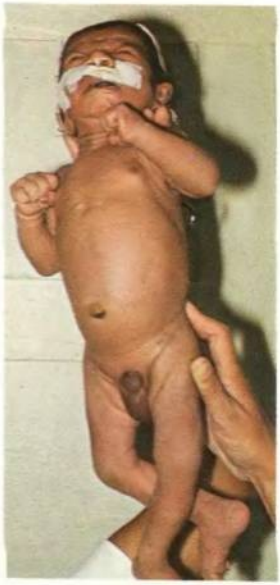


644 Bone involvement in tropical ulcer Sequestra result when bone involvement occurs.

645 Cancrum oris A gangrenous condition of the facial region associated with Vincent's organisms may follow any acute systemic disease in malnourished infants in the tropics. Gross disfigurement usually results.

646



Tetanus

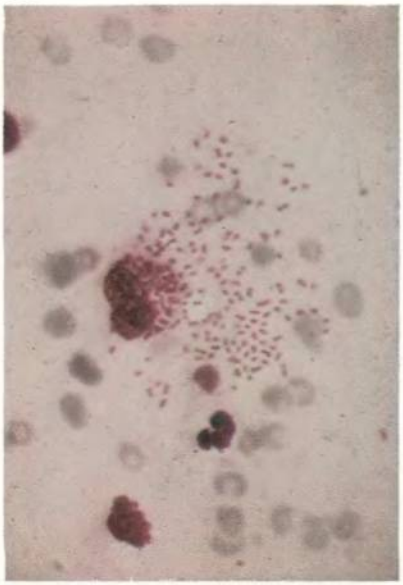
646 Tetanus neonatorum Tetanus is an important infection in the tropics. Infection through the umbilical cord is common in tropical conditions unless the mothers are previously immunised. One of the characteristic features is the 'risus sardonicus' resulting from spasms of the facial muscles.

Granuloma Inguinale (Donovanosis)

647 Donovan bodies in exudate *Donovania granulomatosis* is an encapsulated, gram-negative coccobacillus which in lesions in man is seen within phagocytes, the 'Donovan bodies'. It is venereally transmitted. *(× 900)*

648 Donovanosis of penis and adjacent skin of leg The ulcerated lesion is deep and the floor is covered by a thick, offensive, purulent exudate. Secondary contact lesions are common.

647

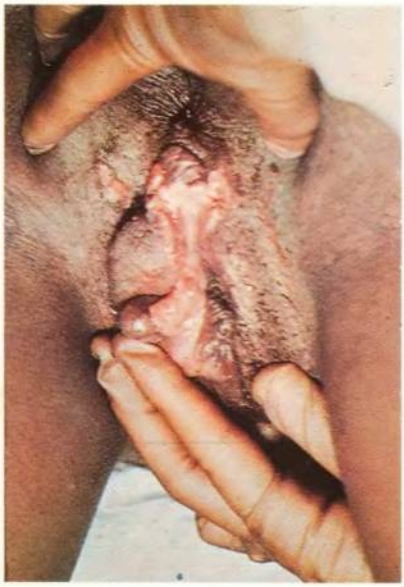


648

649 Late donovanosis The disease runs a very chronic course. Mutilating ulceration of the genitalia may occur, and ano-rectal involvement is common. In comparison with lymphogranuloma venereum, the lymphatics are not primarily involved.

650 Donovanosis of female genitalia As in lymphogranuloma venereum severe deformity of the genitalia can occur in the female.

649



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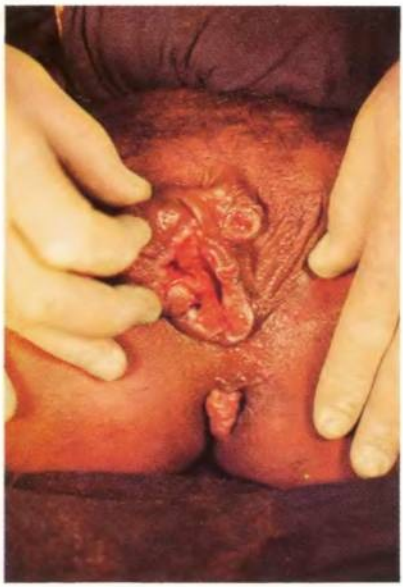


Gonorrhea

651 Urethral discharge in gonorrhea Gonorrhea is widespread in the tropics where chronic gonococcal salpingitis is a common cause of infertility in women. Gonorrheal urethral strictures are often seen in men.

651

Yaws

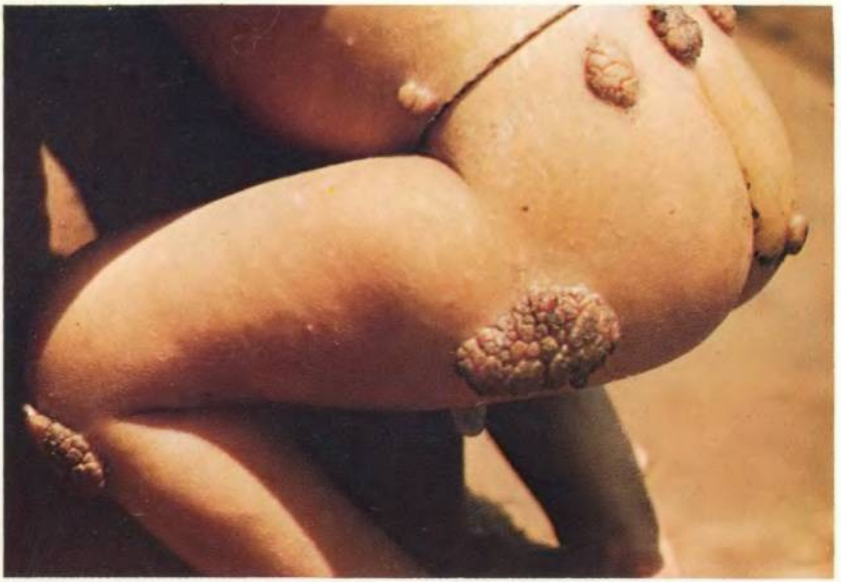
652 Secondary framboesiform yaws Thanks to a mass penicillin-based eradication campaign, yaws is now a relatively rare disease in the humid tropics. This Papuan child shows classical framboesiform lesions, caused by *Treponema pertenue*. Secondary lesions are frequent also at muco-cutaneous junctions.

653 Plantar hyperkeratosis Hyperkeratosis of feet and hands is a common secondary phenomenon in yaws. This man's feet were seriously eroded.

654 Gangosa The most advanced and destructive lesions affect the maxillary bones and hard palate, resulting in a condition known as 'gangosa'.

655 X-ray of forearm with yaws osteitis Focal cortical rarefaction and periosteal changes are seen especially in the tibia ('sabre tibia'), but also in other long bones.

652



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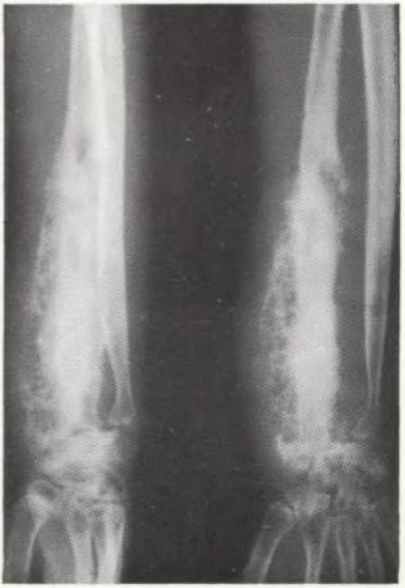

Syphilis

656 Primary syphilitic chancre Syphilis, the venereally transmitted treponematosis caused by *T. pallidum*, is widespread in many parts of the tropics, but does not occur where yaws is endemic. The figure shows a typical primary chancre with associated secondary lesions of the scrotal and abdominal skin.

Non-venereal Treponematoses

657 Depigmentation lesions of Pinta Pinta is endemic in the New World from Mexico to the Amazon. 'Pintids' start as small papules and develop into plaques with actively growing edges which become confluent. In the late stages the 'pintids' become depigmented. The causative organism of Pinta, *T. carateum*, is morphologically indistinguishable from that of syphilis and Bejel.

658 Secondary rash in endemic syphilis These non-venereal spirochaetoses ('endemic syphilis') occur mainly in dry parts of Africa, the Balkans, and Australia. A florid secondary maculopapular eruption and associated adenitis is usually the first sign. Tertiary complications including gangosa may develop. In the Middle East the condition is known as Bejel. Other forms of non-venereal 'endemic syphiles' are njovera (Rhodesia), skerlievo (Borneo), dichuchwa (Botswana) and siti (Gambia).

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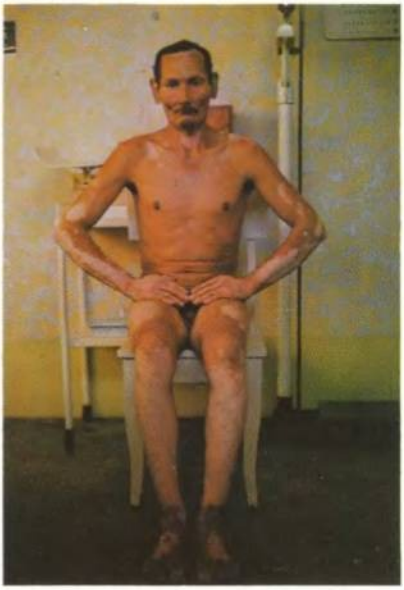

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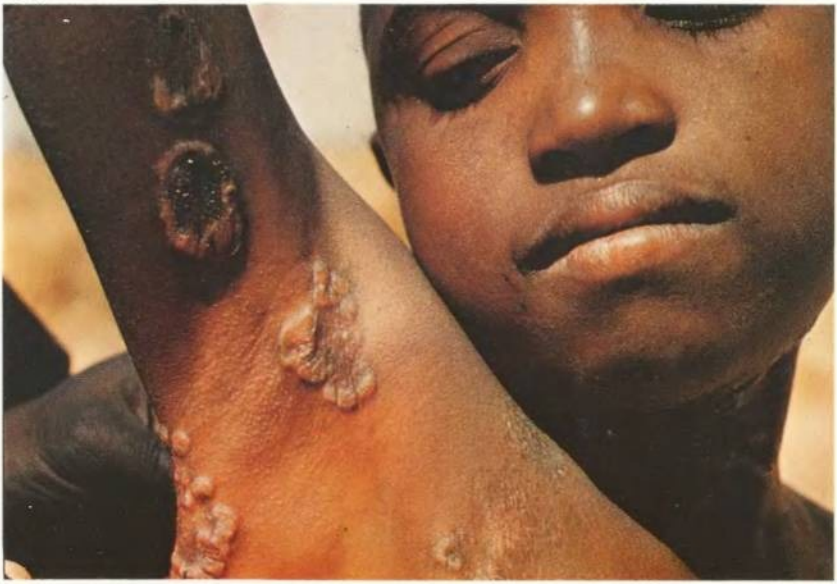
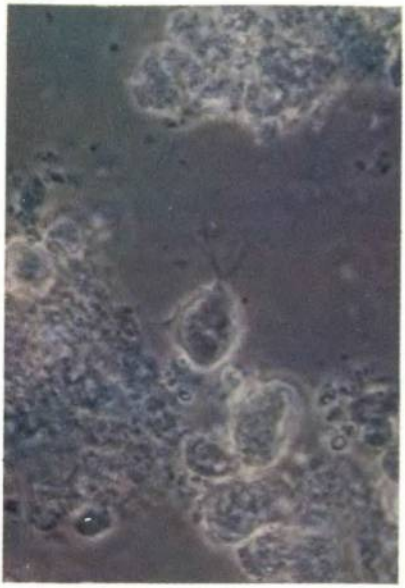


Trichomoniasis

659 & 660 Trophozoites of *Trichomonas vaginalis* The motile flagellate is found readily in the foamy vaginal discharge of trichomonal vaginitis. *(× 900)*

661 Trichomonal vaginitis The typical appearance of vaginitis as seen through a vaginal speculum. Note the creamy discharge which is commonly secondarily infected with *Candida albicans*.

659



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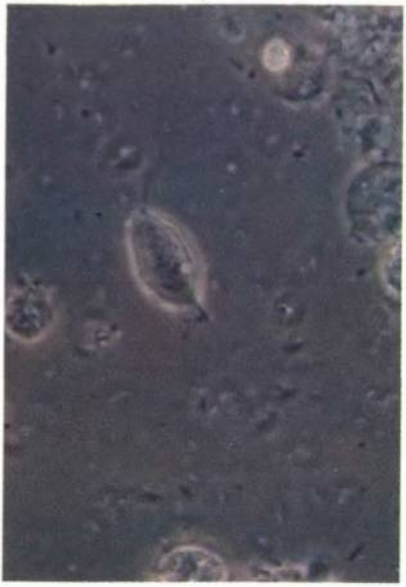

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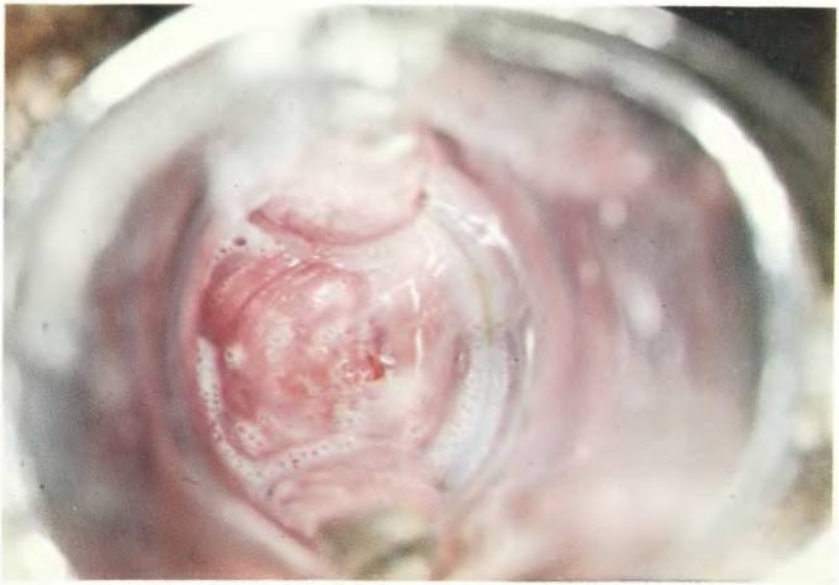
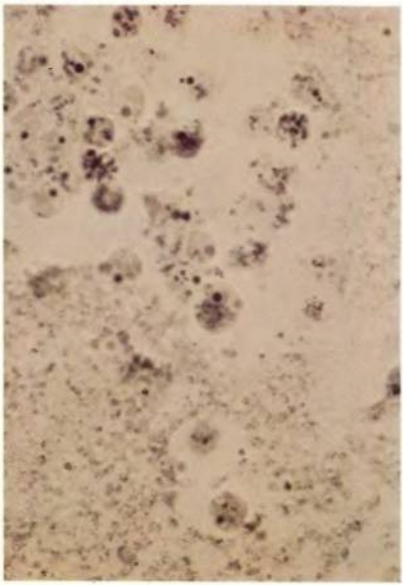


Primary Amoebic Meningo-Encephalitis

662 Section of brain containing amoebae Primary amoebic infection of the brain with free-living *limax* amoebae is rare. Infection appears to be acquired through the cribriform plate of the nasal cavity following immersion in contaminated water. *(× 900)*

663 & 664 Living trophozoites of *Naegleria fowleri* Several species of *Naegleria* (Vahlkampfiidae) may be associated with this condition. They develop a biflagellate form in water. *(× 900)*

662



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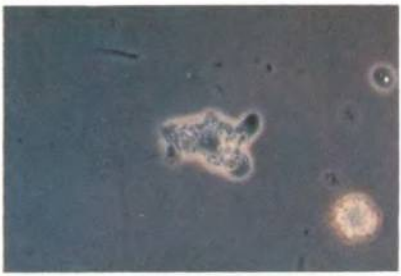

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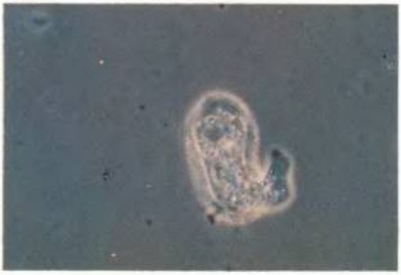

THE SUPERFICIAL MYCOSES*

665 Tinea imbricata *Trichophyton concentricum* produces characteristic superficial scaly lesions in parallel lines and concentric circles. It is common in the South Pacific and parts of the Far East, and is occasionally seen in other hot humid areas.

666 Pityriasis versicolor This condition due to infection with *Malassezia furfur* is a common cause of hypopigmentation in dark skinned young adults. Fluorescence of the patches in Wood's light helps to differentiate the condition from vitiligo and other depigmenting conditions.

*(*See* **Table XIV**)

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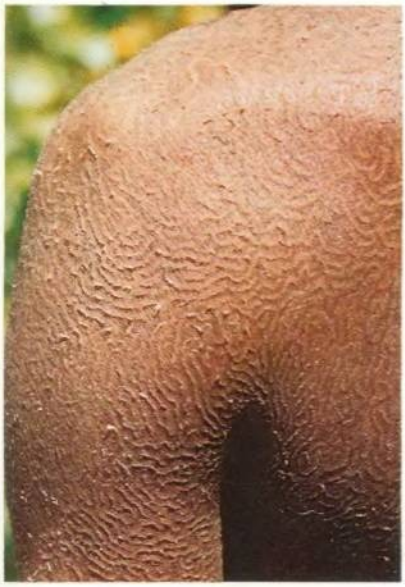

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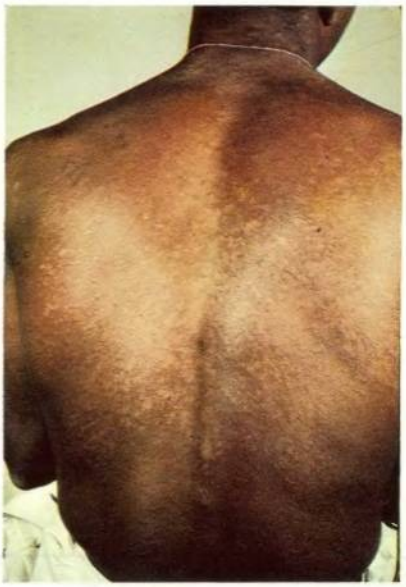

THE SYSTEMIC MYCOSES*

667 Madura foot This chronic and disabling condition may be caused by a wide variety of organisms ranging from *Actinomycetes* to various *Fungi imperfecti*.

*(*See* **Table XIV**)

668 X-ray of Madura foot Infiltration of the tarsals and metatarsals occurs in late cases.

669 Serological diagnosis Fungal species identification can be made serologically. This serum contains antibodies to *Madurella mycetomae*.

670 & 671 Culture diagnosis *M. mycetomae* (**670**) and *Streptomyces pellietieri* (**671**) on Sabouraud medium show typically shaped and pigmented colonies.

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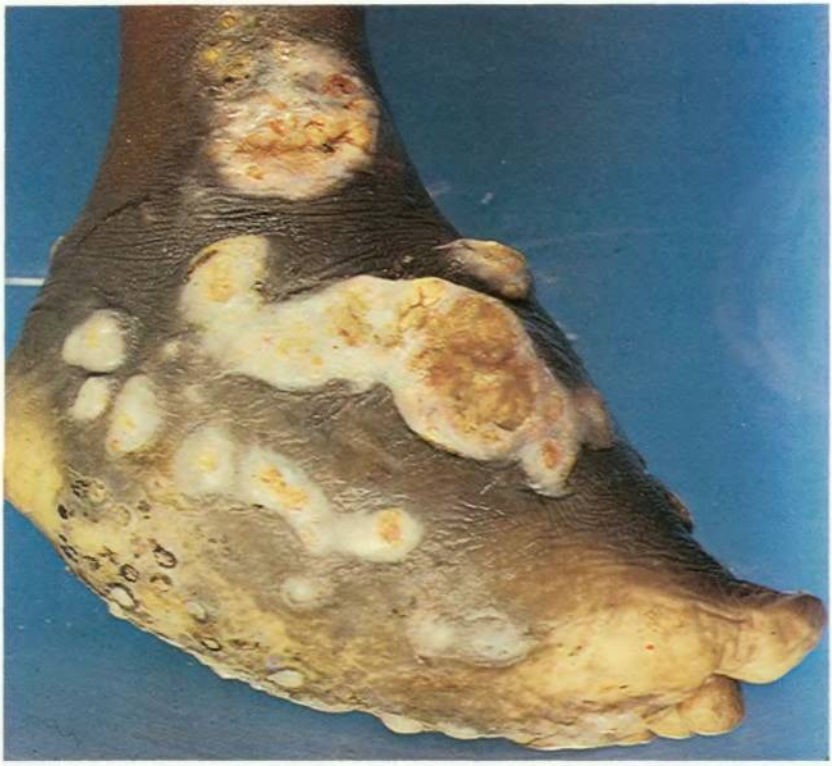

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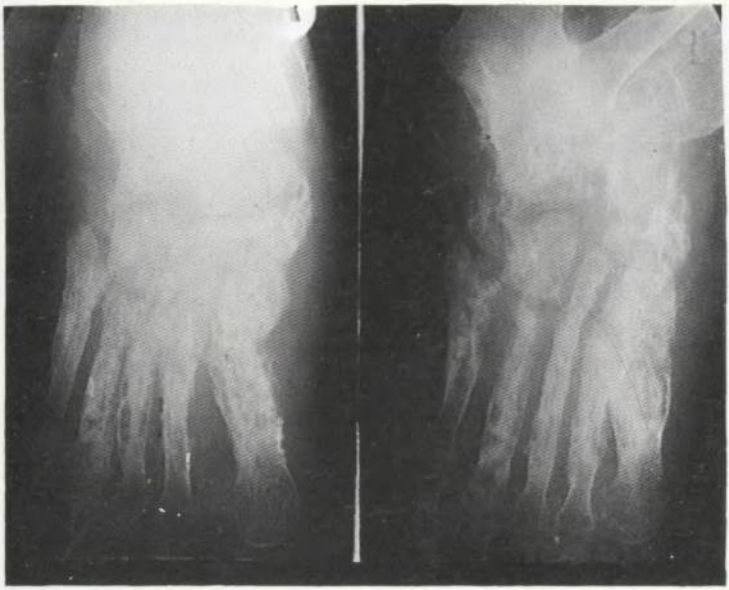

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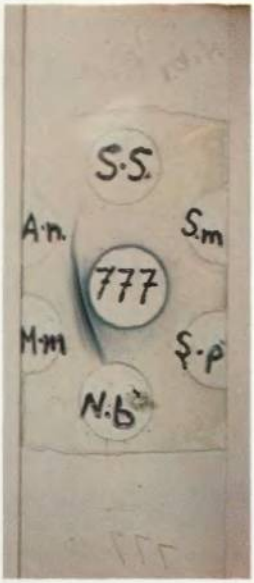

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671

672 Chronic maduromycosis due to *S. somaliensis* infection Extensive sinus formation and osteitis have led to gross disfigurement in this Sudanese man.

673 Early chromoblastomycosis The early lesions show a violet discoloration. The primary ulcer spreads slowly and is followed by verrucous lesions. Several species of fungi may cause the disease.

674 Verrucous dermatitis This is the late stage of chromoblastomycosis. The lesions are very chronic, usually painless but irritating. Lymphoedema follows lymphatic stasis.

675 Lôbo's disease This condition usually presenting with shiny, keloid-like lesions, produces a general picture similar to late chromoblastomycosis and occurs in the north east of Brazil. It is caused by *Lôboa lôboi*.

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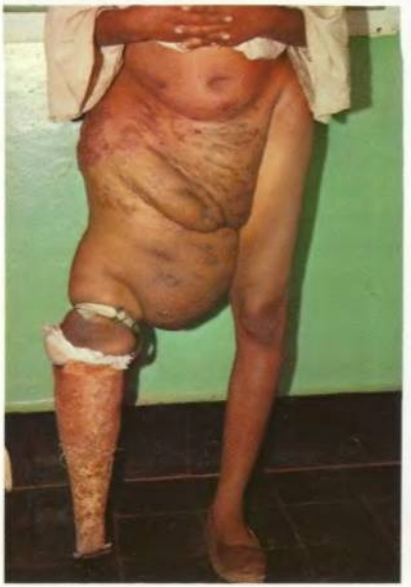

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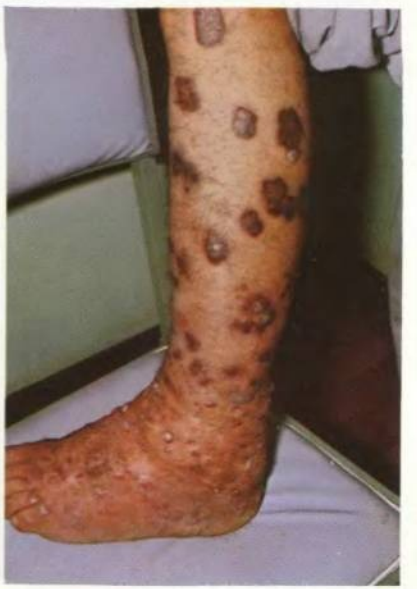

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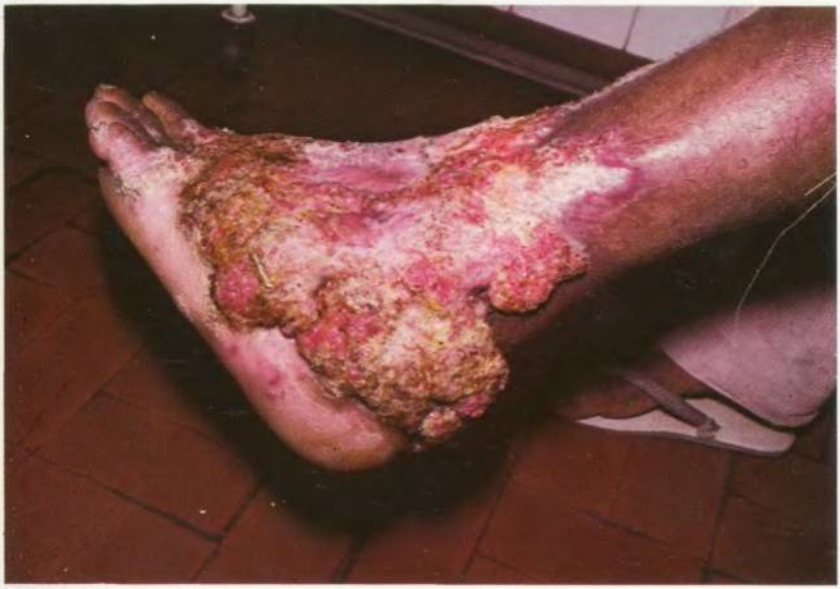

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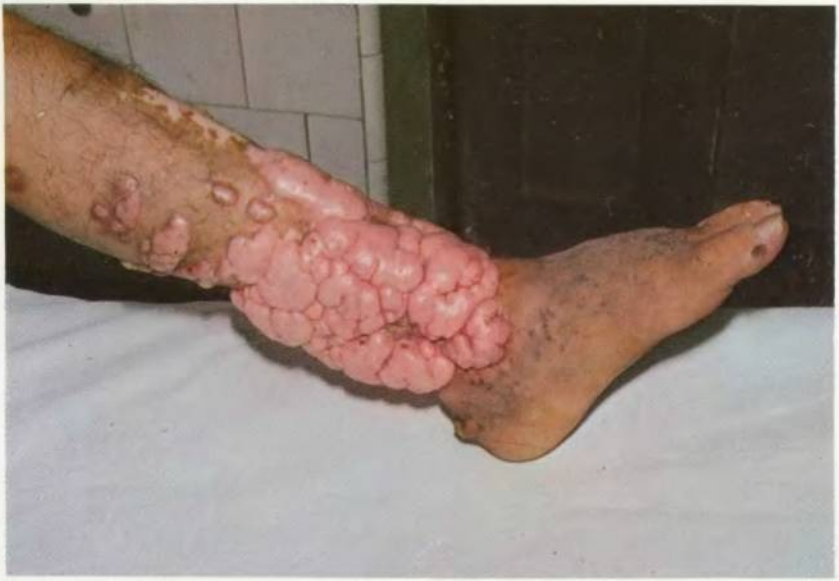

675 ***Lôboa lôboi*** The typical spores of this fungus are seen in this biopsy specimen from the keloidal lesions. *(× 350)*

677 Mucocutaneous lesions in South American blastomycosis Gross infiltration of mucocutaneous and mucous surfaces by *Paracoccidioides brasiliensis* may spread to the pharynx and larynx.

678 X-ray of chest in South American blastomycosis Chronic pulmonary infiltration may result in fibrosis and eventual death from respiratory insufficiency.

679 Meningitis due to *Cryptococcus neoformans* The organisms are readily visualised in Indian ink preparations of infected cerebrospinal fluid from patients with meningitic infection.

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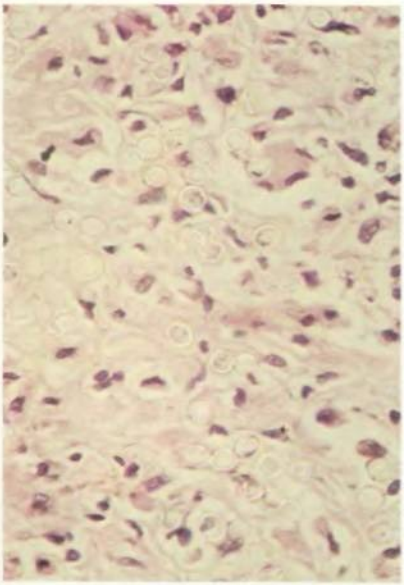

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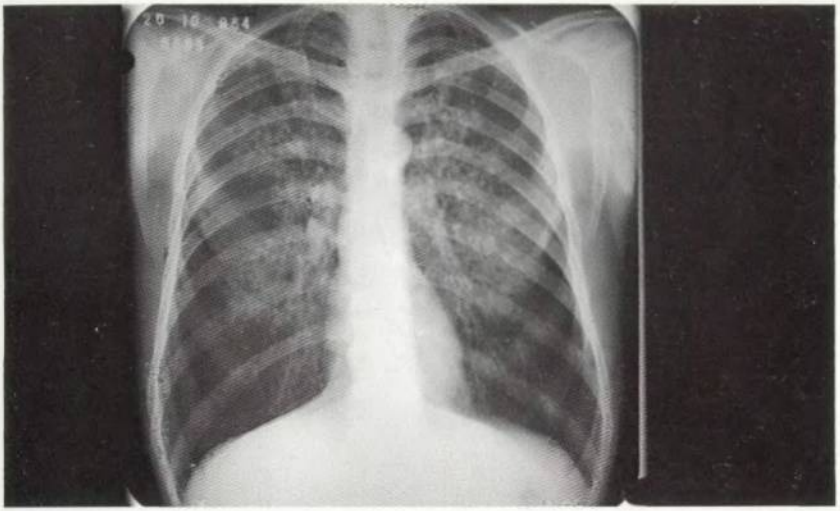

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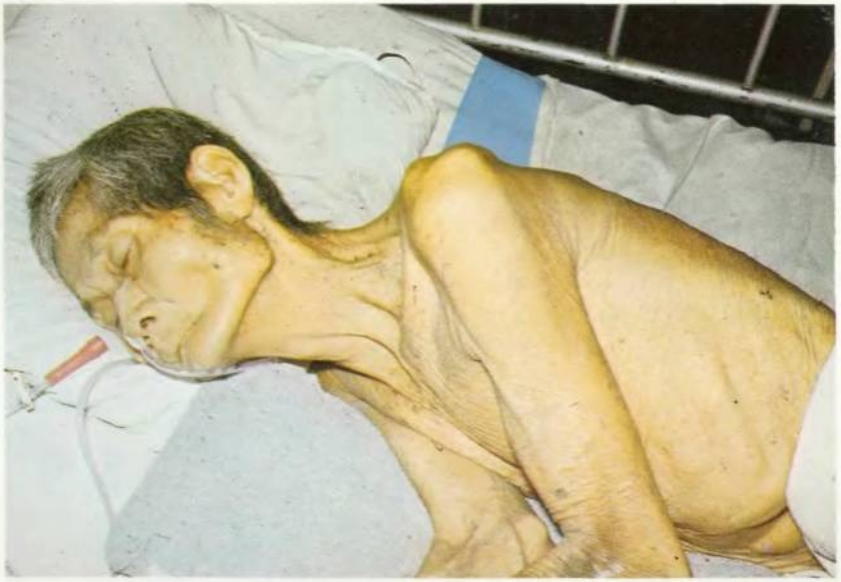
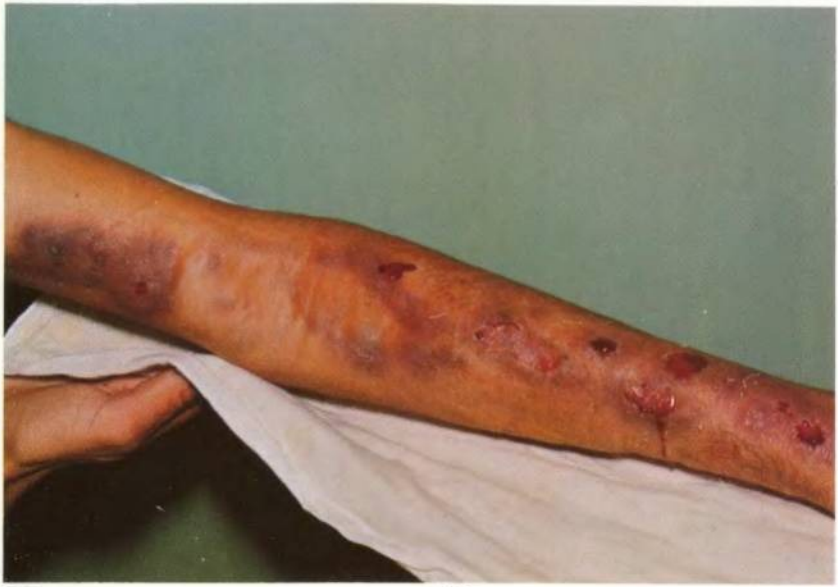


680 Lesions of sporotrichosis The typical lymphatic spread with ulceration of secondary nodules is well shown in this figure of a patient seen in Belo Horizonte, Brazil.

681 African histoplasmosis This type of histoplasmosis caused by *Histoplasma duboisii* commonly produces large destructive lesions of the skin and subcutaneous tissues. Bones are often involved in the invasive process.

682 *H. duboisii* in biopsy The typical giant cell reaction to the presence of *H. duboisii* spores is seen in this figure. *(× 350)*

680



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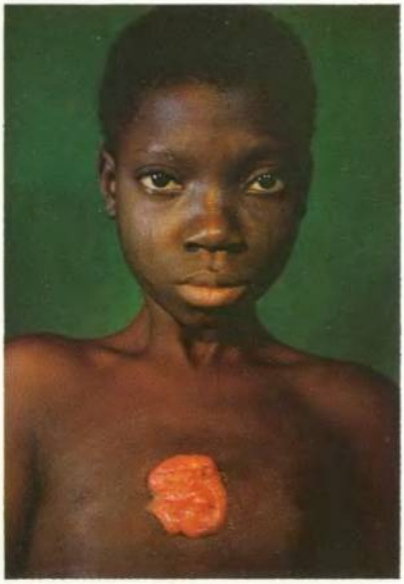

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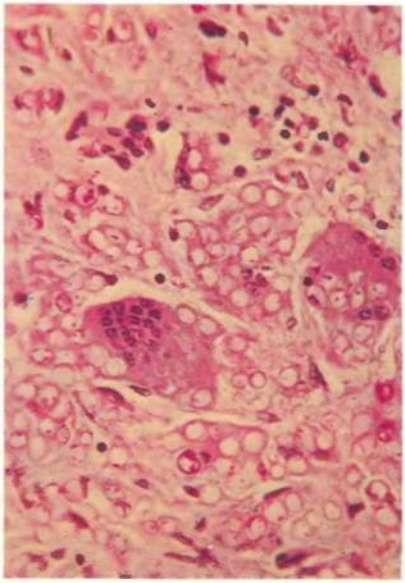

683 *Larval Trombicula autumnalis* (Ventral view) These 'harvest mites' are common in grassland. The larvae which normally feed on small mammals and birds also attack man causing intense irritation. *(× 90)* *(See also* **34**)

Scabies

684 Female scabies mite (Ventral view) The gravid female *Sarcoptes scabiei* burrows into the epidermis, lays its eggs and dies at the end of the tunnel. It is cosmopolitan in distribution. *(× 90)*

685 Infected scabies in a Papuan boy Intense local pruritis and dermatitis appear within a few days of infection. The tortuous tunnels may extend for several centimetres.

686 Secondary erythema in scabies Secondary infection is common and erythema may be associated with bacterial invasion of the sarcoptic tracks.

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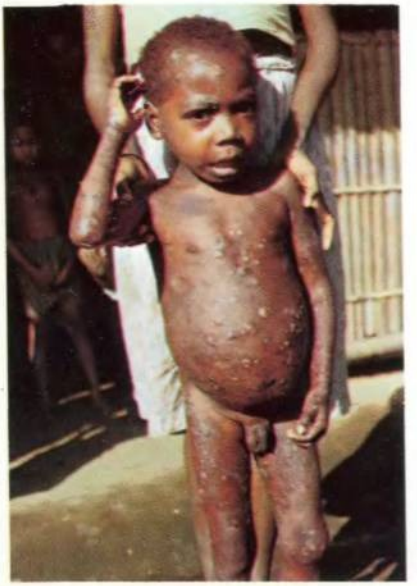

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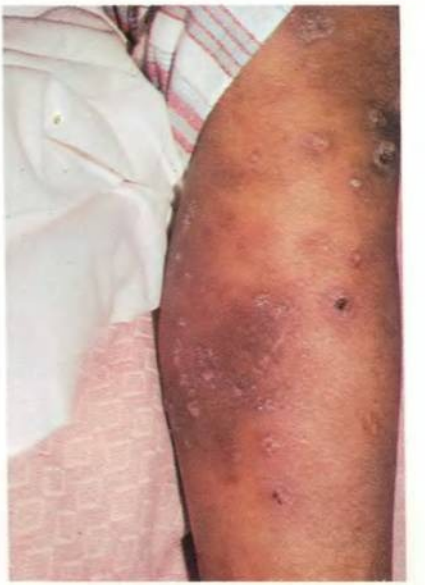

Tungiasis

687 a & b Male and female *Tunga penetrans* The 'jigger flea' occurs in tropical areas of South America and Africa. The gravid female buries itself in the skin, often under the toenails, and swells up to the size of a small pea. Eggs are laid through the entry hole. *(× 20)*

687a

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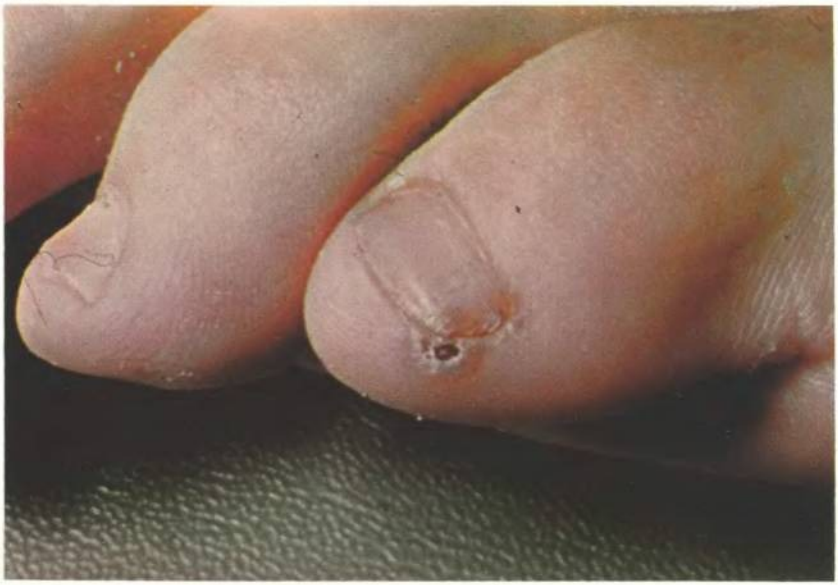

688 Jigger fleas in toe Habitual sufferers shell the gravid females out of the skin with a pin or sliver of bamboo, usually scattering eggs in the process. Tetanus is a common sequel to this type of self-treatment. The larvae mature to adulthood on the ground.

Myiasis*

689 Larva of Tumbu fly The larva of the tumbu fly, *Cordylobia anthropophaga*, produces painful boils in which the insects grow to maturity. The figure shows the powerful hooks with which they feed inside the skin. *(×20)*

690 Tumbu fly lesions Multiple infections with tumbu fly larvae cause painful boils on the trunk.

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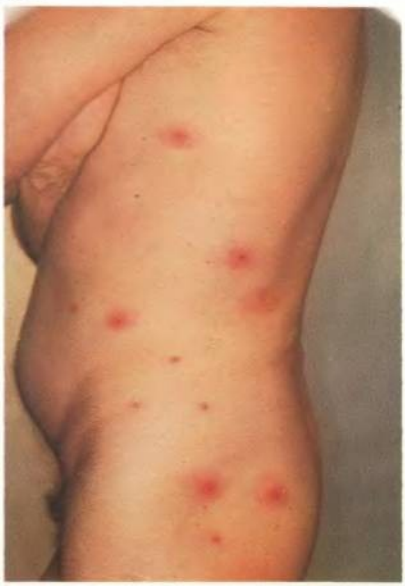

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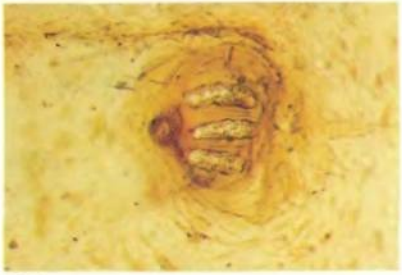

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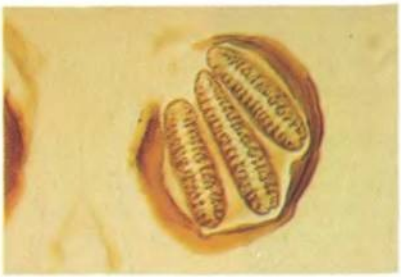

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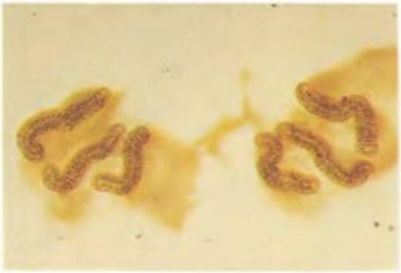

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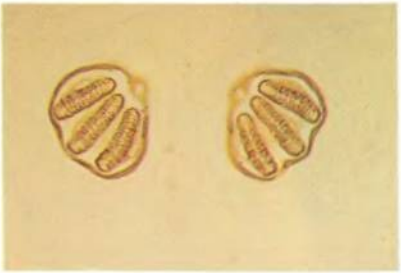

695

696

691–694 Larval spiracles of *Auchmeromyia luteola* and other calliphorid flies *Chrysomyia* (**692**), *Cordylobia* (**693**), *Lucilia* or *Calliphora* (**694**). The 'Congo floor maggot' feeds on man by sucking blood nocturnally but does not remain attached. The adult is similar to that of the Tumbu fly. The larva is separated from that of other Calliphorid flies by the distinctive posterior spiracles. *(× 75)*

695 *Dermatobia hominis* The larvae of this fly are a cause of serious cutaneous myiasis in Brazil and other tropical areas of the New World. *(Natural size)*

696 Larval and pupal stages of *D. hominis* The immature stages of this fly are characteristic.

*(*See* **Table XV**)

Part VI Airborne Infections

Infections of the upper respiratory tract are acquired mainly by the inhalation of pathogenic organisms. Man is the reservoir of most of these infections. Carriers play an important role and may represent the major part of the reservoir, eg meningococcal meningitis. There are three main mechanisms for the transmission of airborne infections – droplets, droplet nuclei and dust.

Measles tends to be a severe disease in malnourished children, and in some epidemics in the rural tropics the mortality has been as high as 50%. The infection not infrequently precipitates 'kwashiorkor'. Whooping cough is an important cause of infantile mortality in some areas of the tropics, while tuberculosis remains one of the major health problems in many tropical countries where it is being aggravated by dense overcrowding in urban slums. Tuberculosis presents a wide variety of clinical forms, but pulmonary involvement is common and is most important epidemiologically, since it is mostly responsible for the transmission of the infection.

Massive epidemics of meningococcal meningitis occur periodically in the so-called 'meningitis belt' of tropical Africa (*see* **700**). In this zone, the epidemics come in waves followed by periods of respite.

MEASLES

697 Koplik's spots Koplik's spots are pathognomonic of measles. They are found on mucous membranes during the prodromal stage and are easily detected on the mucosa of the cheeks opposite the molar teeth, where they resemble coarse grains of salt on the surface of the inflamed membrane. Histologically the spots consist of small necrotic patches in the basal layers of the mucosa with exudation of serum and infiltration by mononuclear cells.

698 Measles in twins Measles is one of the most important causes of childhood mortality in the tropics. The twin on the left shows typical post-measles desquamation but is otherwise recovering. The other twin has post-measles encephalitis.

699 Lung in giant cell pneumonia Mortality is commonly associated with giant cell pneumonia during the prodromal stage. *(H&E × 500)*

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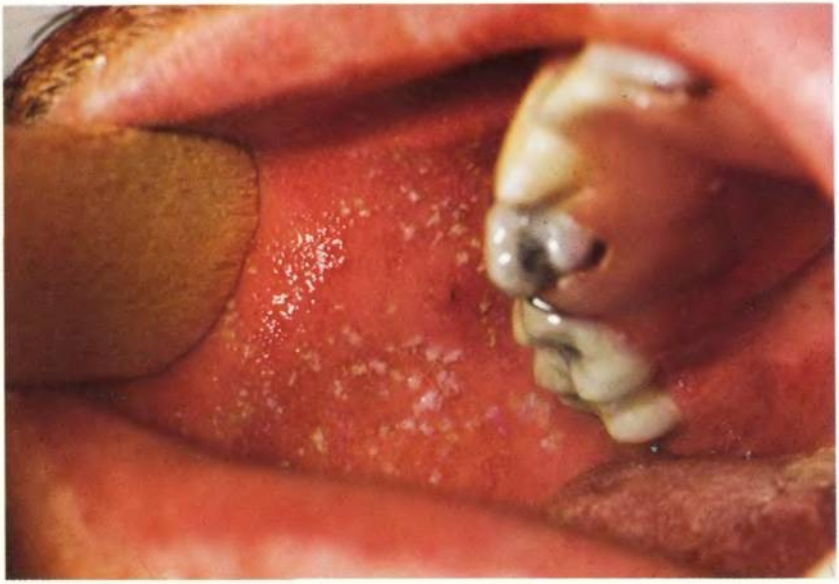

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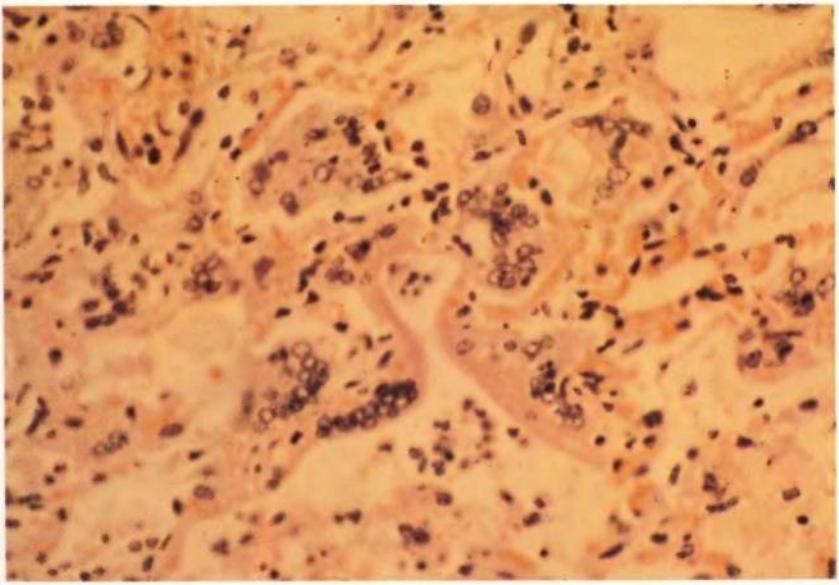

MENINGOCOCCAL MENINGITIS

700 Distribution The 'meningitis belt' of tropical Africa is the zone lying between 5° and 15° N of the Equator, and characterised by an annual rainfall between 30 and 110 cm. The disease however is not limited to Africa. Overcrowding enhances the risk of acquiring the infection. Nearly 3000 people died from meningitis in two Brazilian cities in 1974. Effective vaccines against types A and C are now available.

701 Rash of meningococcal meningitis The rash consists typically of irregular, scattered petechiae.

702 Smear of cerebrospinal fluid *Neisseria meningitidis* is a gram-negative, bean-shaped diplococcal organism. In the 'meningitis belt' of Africa, type A is the causative agent of epidemics. *(× 900)*

703 Latex agglutination test This sensitive serological test is valuable for the differentiation of meningococcal from pneumococcal and other types of meningitis. (A – meningococcus antigen; B – pneumococcus antigen; C – *Haemophilus influenzae* antigen; D – negative control)

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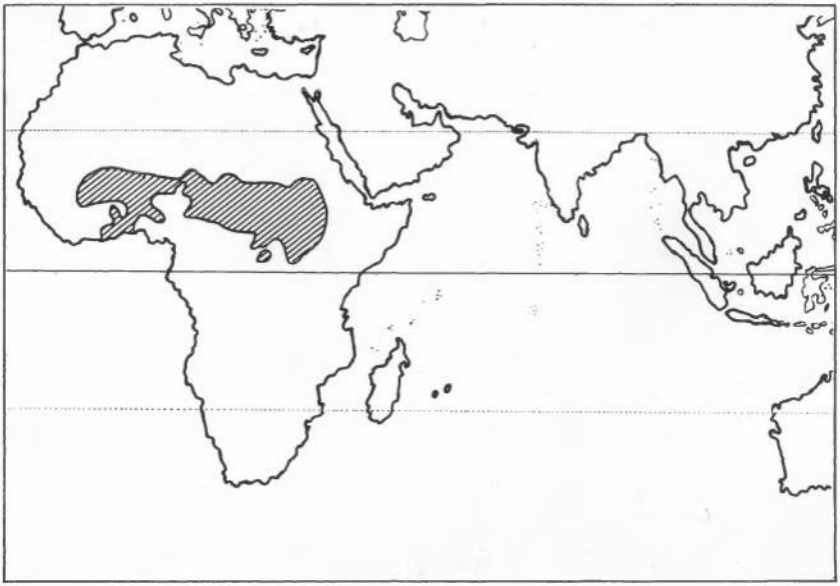

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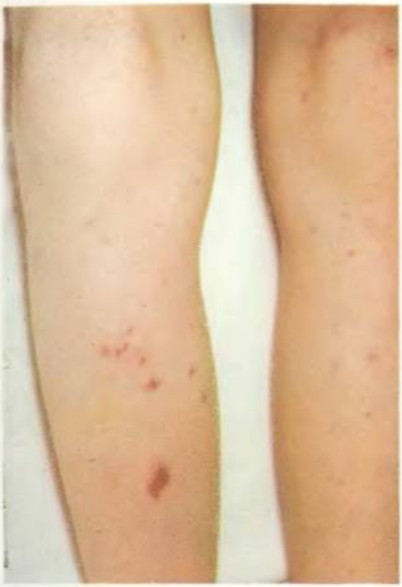

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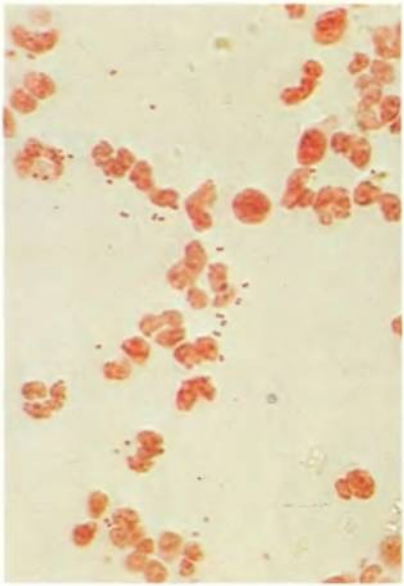

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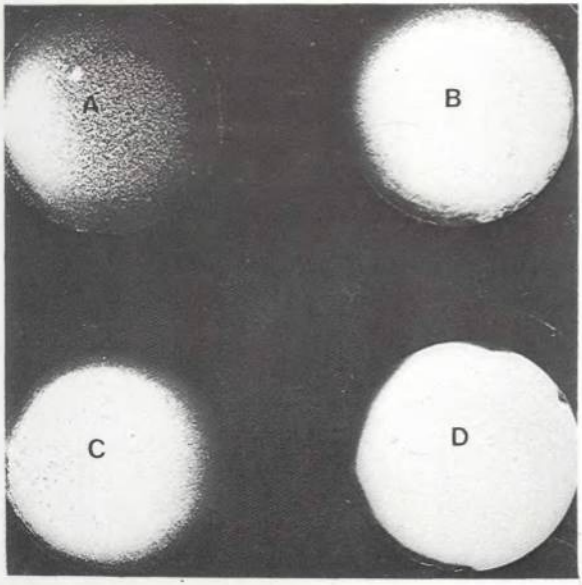

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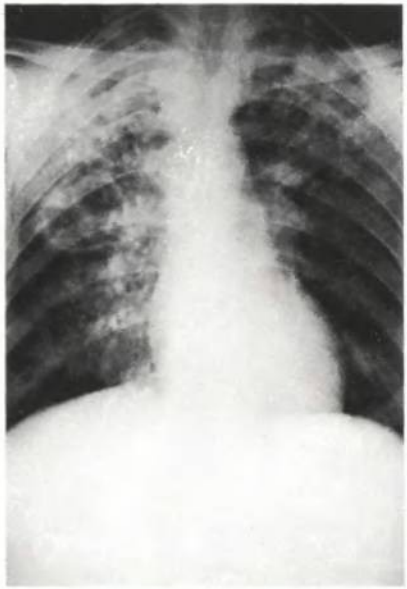

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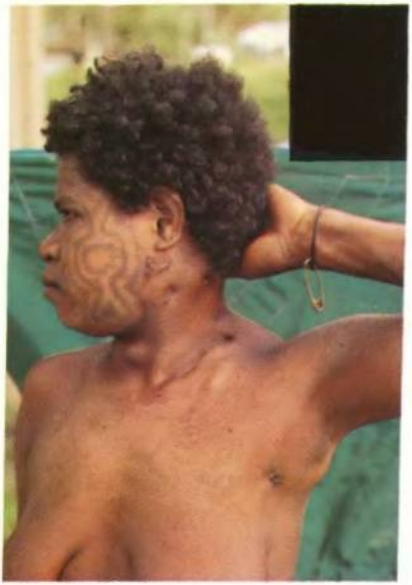


706

TUBERCULOSIS

704 Acute pulmonary tuberculosis with cavitation Tuberculosis remains one of the major health problems in the tropics. Pulmonary involvement is common and is most important epidemiologically, since it is responsible for the transmission of the infection.

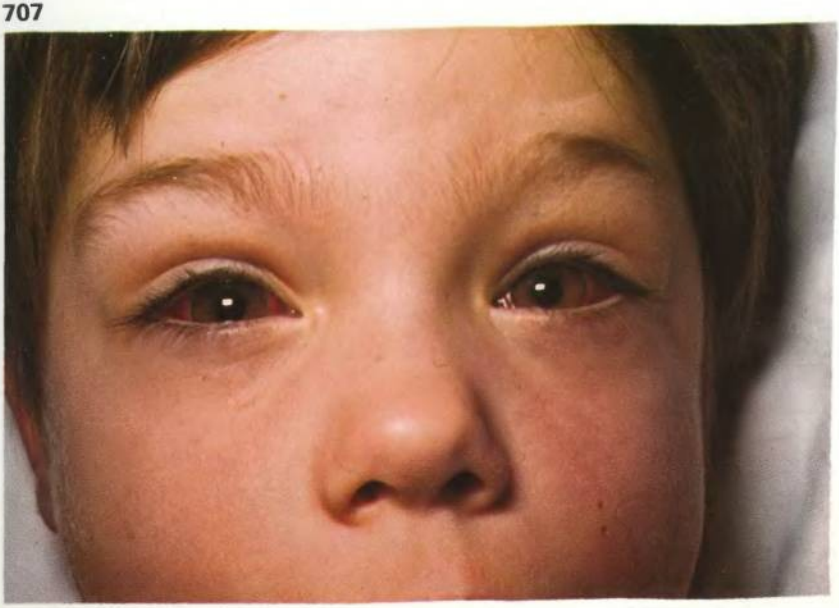
705 Tubercular glands Glandular enlargement due to human tuberculosis is not uncommon. In tropical areas in Africa it is often seen. This woman is Fijian.

706 Spinal tuberculosis Bony lesions in the spine have the usual features as in the developed countries.

WHOOPING COUGH

707 Child with whooping cough Whooping cough is an important cause of infantile mortality in the tropics. Subconjunctival haemorrhages are a common accompaniment of the severe coughing spasms.

707



Part VII
Nutritional Disorders

In large areas of the tropics, malnutrition, especially that affecting young children, is one of the principal causes of morbidity and mortality. The problem of feeding the populations of the world, and therefore maintaining an adequate status of nutritional health, is a serious one. Its magnitude and severity have only recently received attention, and there is no completely reliable assessment of it in quantitative terms. Hunger, as manifest through famines or chronic undernutrition, has been recognised from prehistoric times. However, the problems related to the absence of specific nutrients have begun to be understood only relatively recently.

Human malnutrition is an ecological problem and the following intimately related factors may be involved in its pathogenesis: (1) food production and distribution; (2) food storage and processing; (3) demographical problems related to food, eg the rate of increase of the population in most developing countries is over 2% and yet the rate of increase of food production, in most areas, has not kept up with the population increase; (4) education and socio-cultural factors; (5) food preparation and consumption; (6) the role of infection.

The United Nations Agencies have reckoned that about one third of the world's population goes to bed hungry every day, mostly in the countries of Asia, Africa and Latin America. The most 'vulnerable' groups are infants, pre-school children, pregnant and nursing mothers. Protein-calorie malnutrition is the name accepted now for a disease syndrome which includes Kwashiorkor, believed to be largely due to protein-deficiency, and nutritional marasmus which is due to a general deficiency of all nutrients, especially calories. In tropical communities one sees cases ranging from one extreme to the other.

Among adults, acute periods of undernutrition may occur in large populations because of failure of food crops, or catastrophes of one kind or another, eg floods, earthquakes, wars and failure of the rains.

The background of nutritional deficiency conditions is very wide and can be seen to be more dependent on the socio-economic level of the

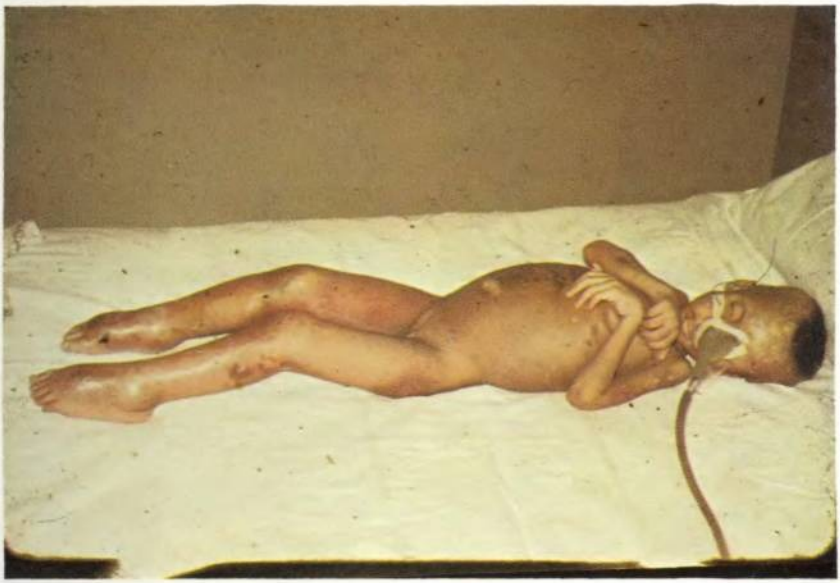
society than practically any other disease. Protein-calorie malnutrition is the most important nutrition problem of the whole world, though deficiencies of Vitamins A, B and D are also quite common. The nutritional deficiencies in many cases are complicated further by additional stress imposed by multiple parasitic infections such as intestinal helminthiases and malaria.

Many tropical diets are based on some staple carbohydrate foodstuff to which other substances are added fortuitously. These diets consist mainly of yams, cassava, rice, plantains, breadfruit and maize. Maize is a poor staple food at any time but when, in addition, the crop fails because of insect pests, famine may result. Such diets are badly balanced and lack total protein and other essential substances. They result in quantitative and/or qualitative deficiencies which are injurious to health.

KWASHIORKOR AND MARASMUS

708 Oedema and hypopigmentation The presence of oedema may give a false impression that a kwashiorkor infant is well nourished. General hypopigmentation together with some haemorrhagic skin lesions are seen in this infant who died shortly after the photograph was taken.

708



709 Kwashiorkor and marasmus in brothers Compare the miserable expression, pale hair, generalised oedema and skin changes in the child on the left with the marasmic wasting of his older brother. Kwashiorkor frequently follows acute infection and/or diarrhoea in a child during the weaning period.

710 Skin changes in kwashiorkor Serious skin changes including erythema, followed by hyperpigmentation, 'black enamel skin' and peeling, may terminate in serious ulceration and gangrene. Note the ulceration where desquamation has occurred in this child.

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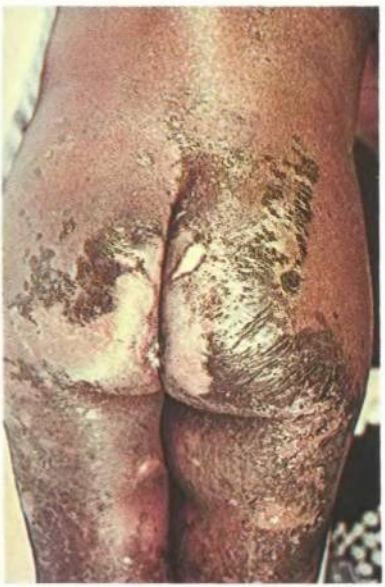

711 Papuan child with nutritional marasmus Note the obvious wasting and dehydration in this marasmic infant, an all too common picture in times of famine when the total calorie intake is grossly insufficient. (The mother has tinea imbricata of the skin; *see* **665**.)

711

AVITAMINOSES

Vitamin A

712 Xerophthalmia Vitamin A deficiency is a common cause of blindness among pre-school children in the tropics, especially in Asia. The dryness of the cornea and conjunctiva give the eye a dull, hazy appearance.

713 Bitot's spots These are silver-grey foamy spots, usually external to the cornea and often bilateral. They are thought to be due to avitaminosis A.

714 Keratomalacia A softening or coagulative necrosis of the cornea occurs in chronic, severe vitamin A deficiency. As for kwashiorkor, vitamin A deficiency may be precipitated by acute infections in undernourished children.

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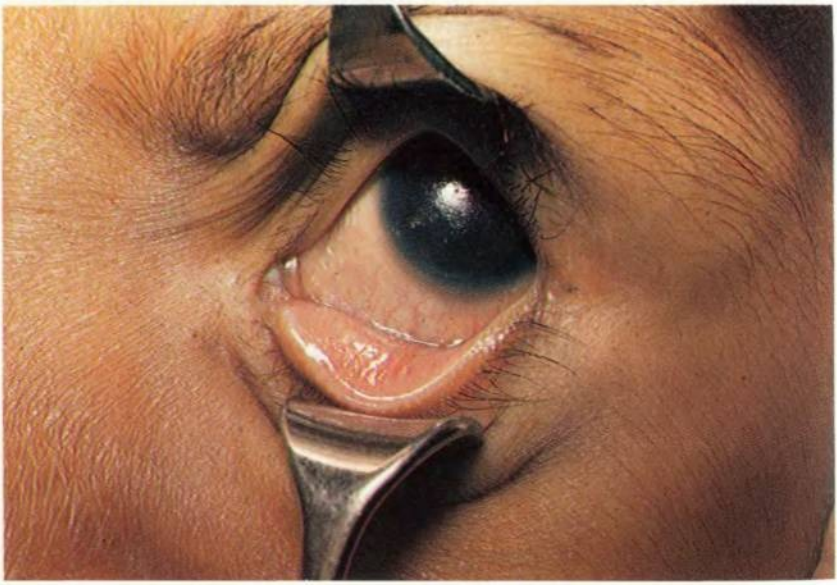

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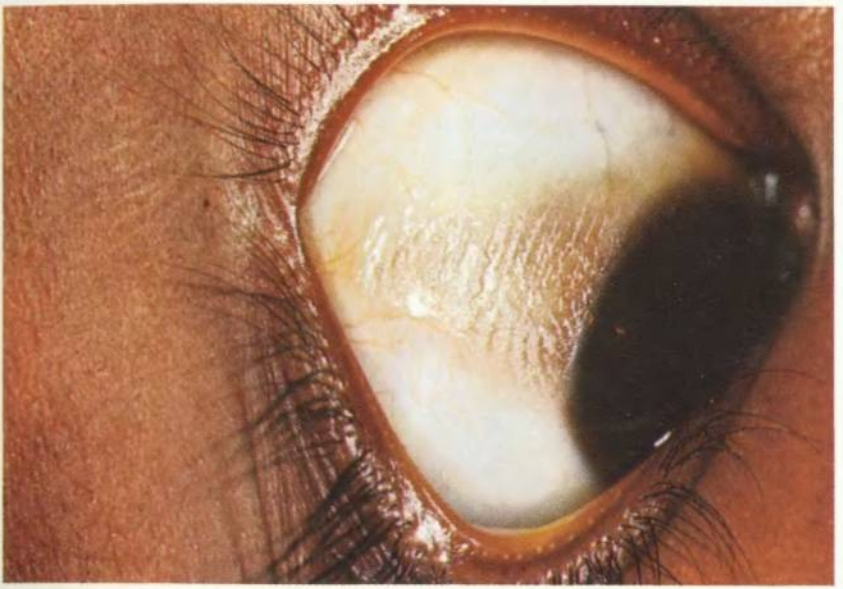

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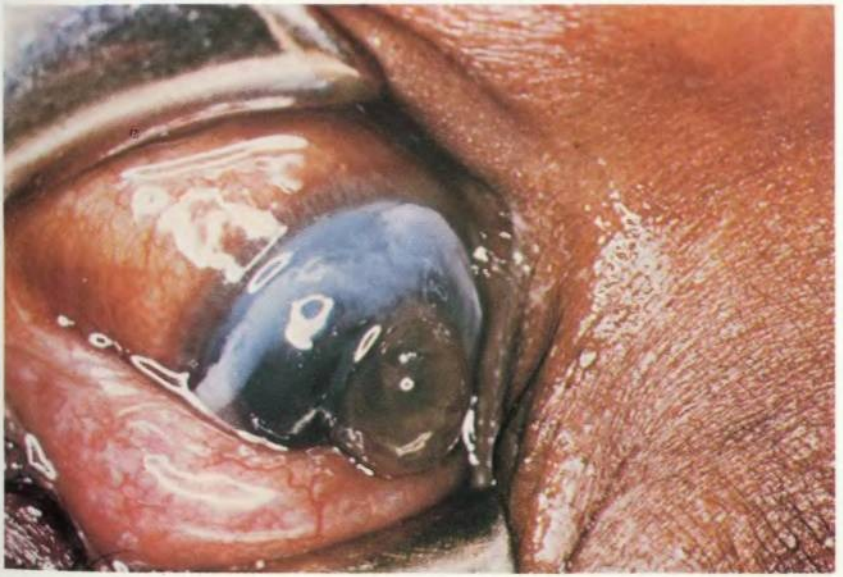


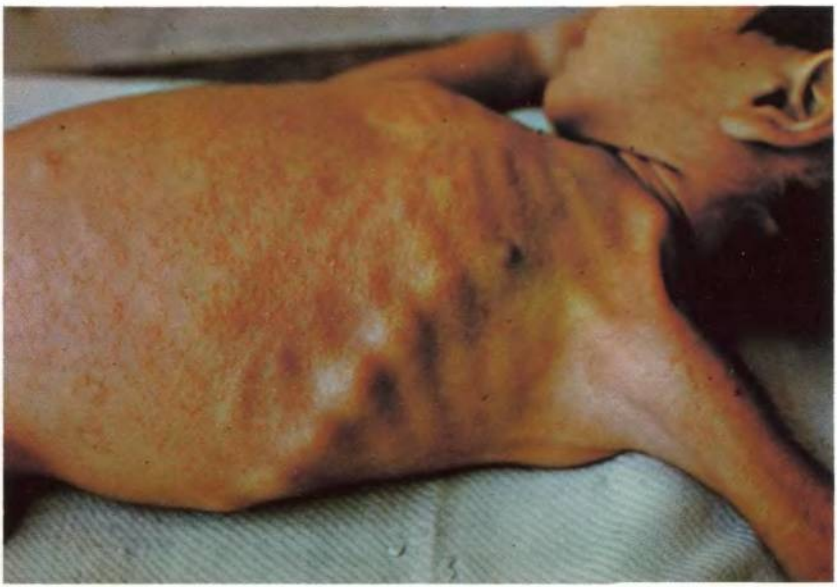
Vitamin D

715 Infantile rickets–'Rickety rosary' This is a disease of infants and children due to insufficient vitamin D. Infants are sometimes over-protected from the sun by their mothers to avoid too rapid pigmentation of the skin, and rickets occurs in this situation when it could easily be avoided. Rounded swellings appear over the costochondrial junctions near the sternum, and are known as 'rickety rosary'.

716 Infantile rickets Note the gross deformity of the legs and pigeon chest of this boy.

717 Skull of two-year-old infant with rickets The anterior fontanelle remains open and its edges soft.

715



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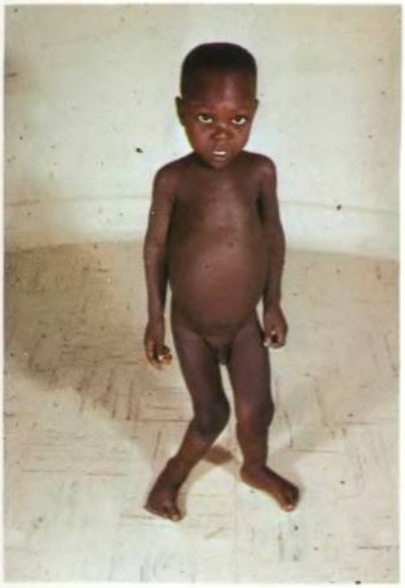

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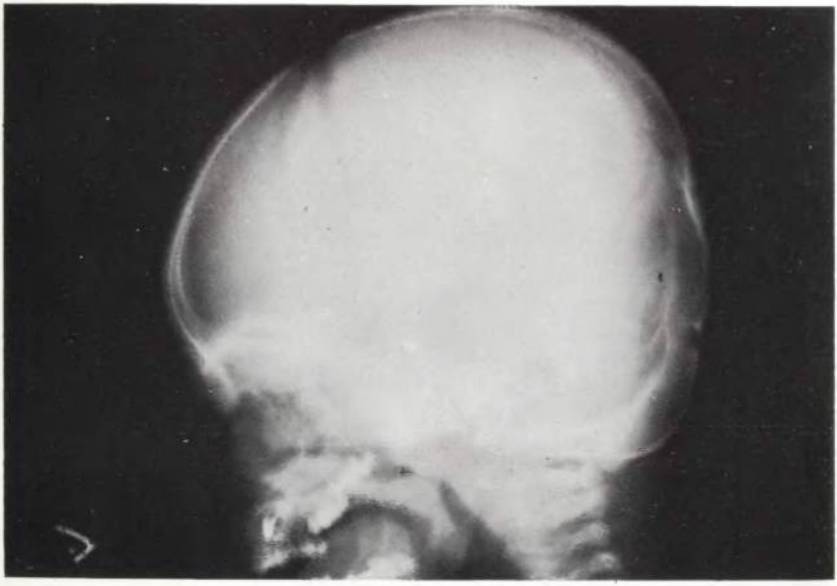
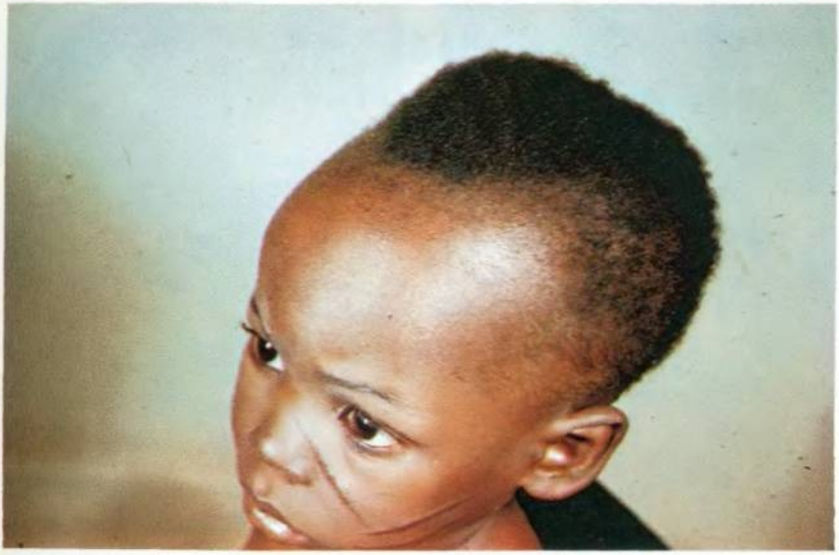


718 Bossing of the skull Bossing of the frontal and parietal eminences occurs.

719 Osteomalacia The increased demands of pregnancy may result in gross deformity of the pelvis. This occurs for example in mothers kept in purdah.

718



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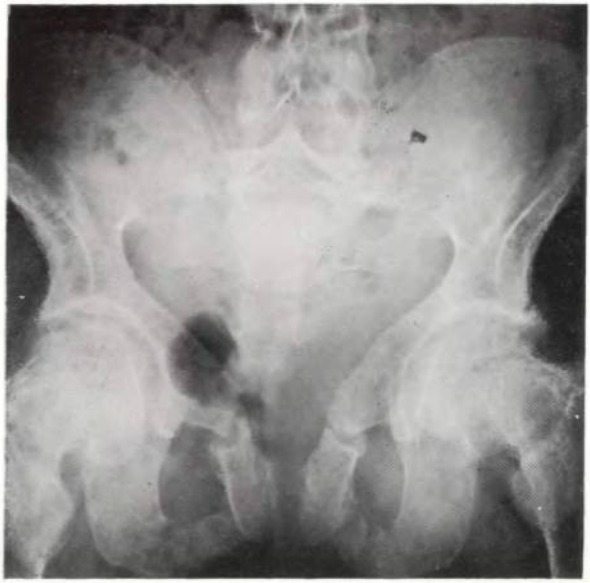

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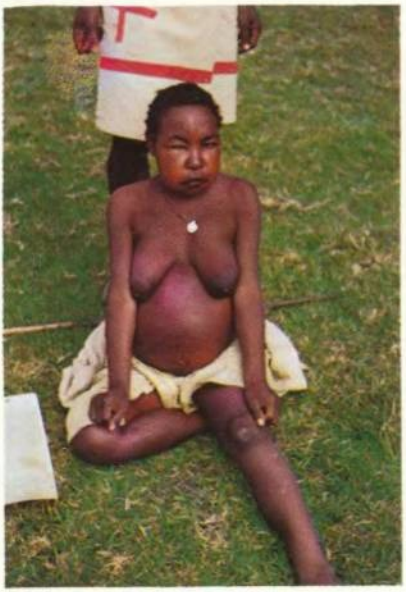

721

The B Vitamins

Thiamine (B1)

720 Beri beri oedema Beri beri is a disease characterised by generalised oedema, peripheral neuropathy and sometimes heart failure, associated with thiamine deficiency.

721 Peripheral neuropathy Wrist drop and marked wasting of the lower extremities occur in some patients.

Riboflavine (B2)

722 Angular stomatitis This consists of grey-white fissures at both angles of the mouth due to riboflavine deficiency.

723 Cheilosis A sore, cracked condition of the lips occurs in association with riboflavine deficiency.

724 Glossitis The tongue is sore and an abnormally deep red colour.

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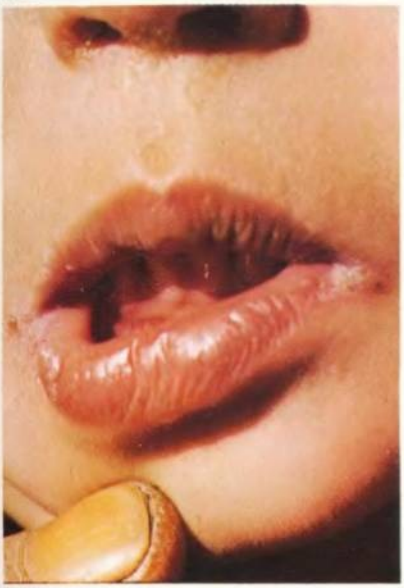

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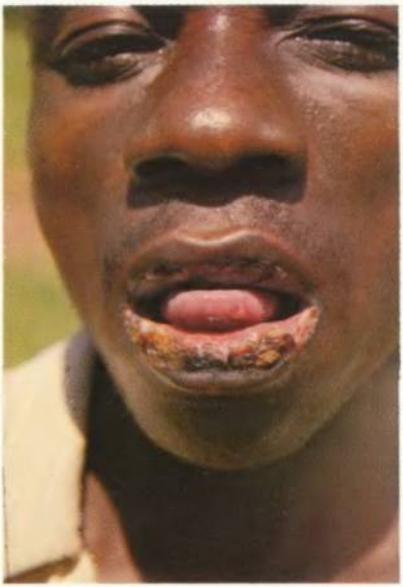

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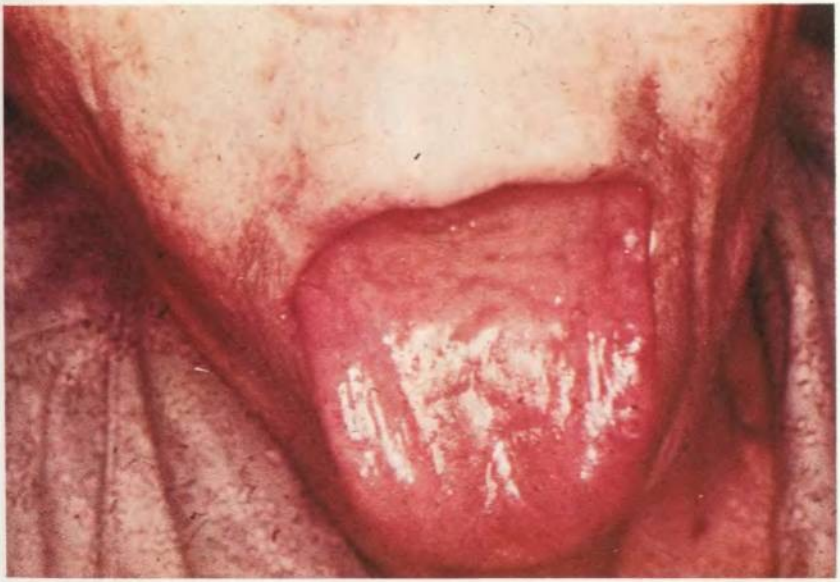
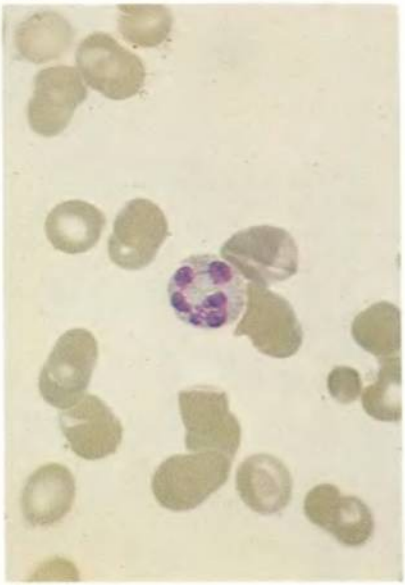


Folic acid

725 Anaemia of folic acid deficiency Folic acid deficiency results in a macrocytic megaloblastic anaemia. *(× 900)*

726 Bone marrow in folic acid deficiency The appearance in this marrow is characteristic of folic acid deficiency. *(× 900)*

725



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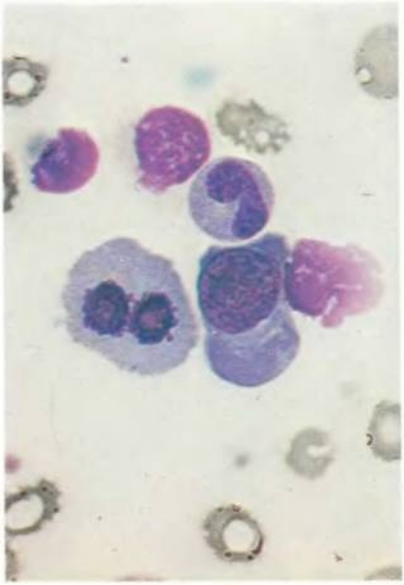


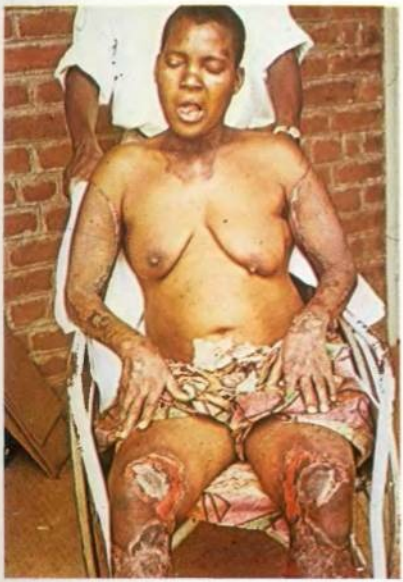
Nicotinic acid (PP)

727 Pellagra The diagnostic triad of the rash of pellagra is a combination of symmetrical skin lesions, sharp demarcation, and distribution in parts exposed to the sun. It is relatively common in people whose diet is composed predominantly of maize.

Vitamin C

728 Scurvy Vitamin C deficiency is rare in the tropics. Severe gingivitis and loosening of the teeth occur in scurvy.

727



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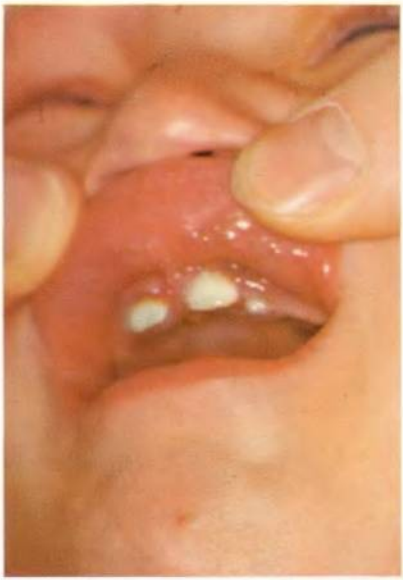


Part VIII
Miscellaneous Disorders

In this section are grouped some common conditions that occur almost exclusively in the tropics, as well as a number of exotic curiosities. Diseases such as Burkitt's tumour, endomyocardial fibrosis and the abnormal haemoglobin syndromes are so well documented elsewhere that we could not do them justice without repeating many of the illustrations already available. We felt, however, that one or two points should be included here to highlight their importance, rather than to omit them altogether from this atlas. Curiosities such as ainhum, kuru, and iatrogenic parasitoses are interesting entities of limited geographical distribution, while conditions such as endemic goitre, hepatoma, and venomous bites are important in many areas of the tropics.

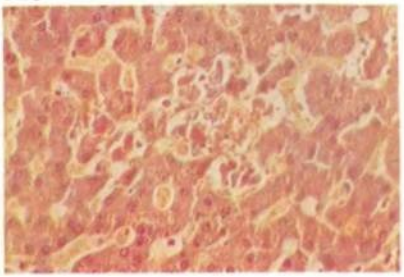
ZOONOTIC VIRAL INFECTIONS

Lassa Fever

729 Liver in Lassa fever Acellular liver necrosis is a marked feature of the hepatic lesion. *(× 250)*

730 Subintimal lymphocytic infiltration of spleen Another pathological feature seen in this disease at post mortem. *(× 60)*

729



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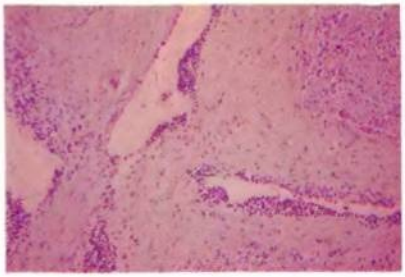
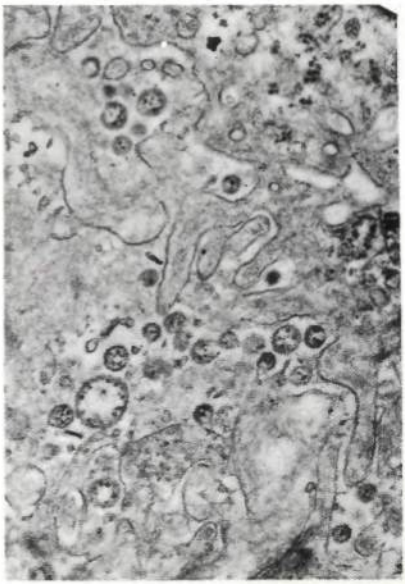


731 Rodent reservoir, *Mastomys natalensis* Lassa fever is an acute infectious disease occurring in the rural areas of West Africa. During the second week of infection toxic or vascular symptoms appear; pharyngitis, serous effusions, facial oedema, haemorrhagic diathesis, disorders of the central nervous system and a state of shock. The case fatality rate is high among severe cases but benign, febrile cases of the disease also occur as well as asymptomatic carriers. A rodent reservoir has been implicated in the epidemiology of Lassa fever.

732 Virus of Lassa fever Virus particles are seen in the perisinusoidal space in this biopsy of liver from a fatal human case. *(×27500)* (From Winn, W C Jr, Monath, T P, Murphy, F A, Whitfield, S G (1975) 'Lassa Virus Hepatitis: Observations on a fatal case from the 1972 Sierra Leone epidemic', *Archives of Pathology* **99**, 599–604.) The virus of lassa fever belongs to the arbovirus group. Its mode of transmission to man is not fully understood, but person to person infections seem common.

731

732



Marburg and Ebola Haemorrhagic Fevers

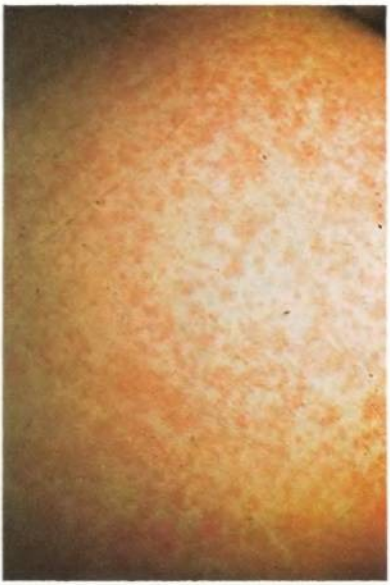
733a Reservoir of Marburg virus The African Green monkey or Vervet was the source of lethal infections contracted by laboratory workers in Europe. Sporadic cases have also occurred in Africa. The causative virus closely resembles that of Ebola haemorrhagic fever (**773c**).

773b Ebola haemorrhagic fever Outbreaks have occurred in Zaire and the southern Sudan with a high case fatality rate in severely ill patients. However, mild cases as well as asymptomatic carriers also occur. Haemorrhagic manifestations in the skin and internal organs are common.

733c Virus of Ebola haemorrhagic fever This and the Marburg virus which is morphologically almost identical are the only branched viruses so far known. The virus shown here is from human liver. *(× 30 000)*

733a

733b



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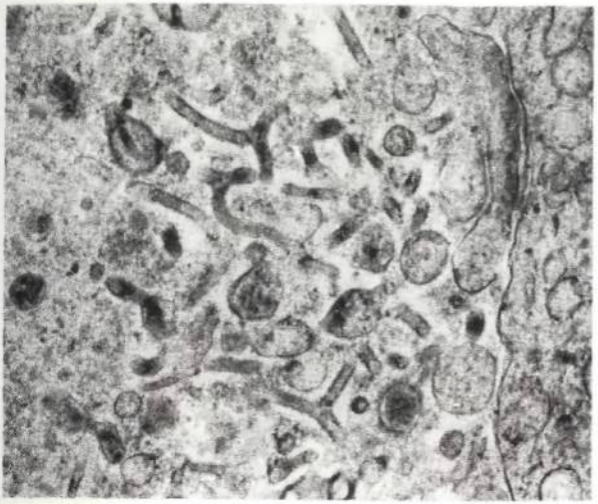


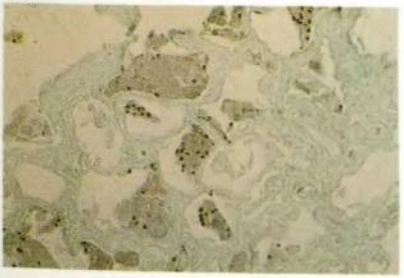
IATROGENIC PARASITOSES

Pneumocystis carinii

734 Silver stain of section of lung Pneumocystosis occurs in infants with immunity deficiency states, or older individuals receiving immunosuppressive therapy. The organisms are seen in the foamy exudate that fills the alveoli. *(× 250)*

735 The organism in lung smear *P. carinii* is an opportunistic parasite that produces eight-nucleated cysts. Its taxonomic status is still undetermined but it is probably a protozoan. *(Giemsa × 600)*

734



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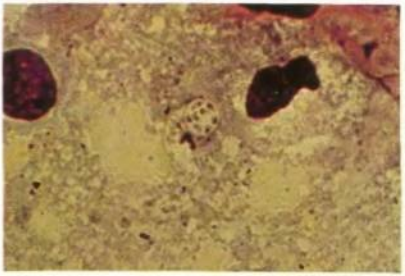

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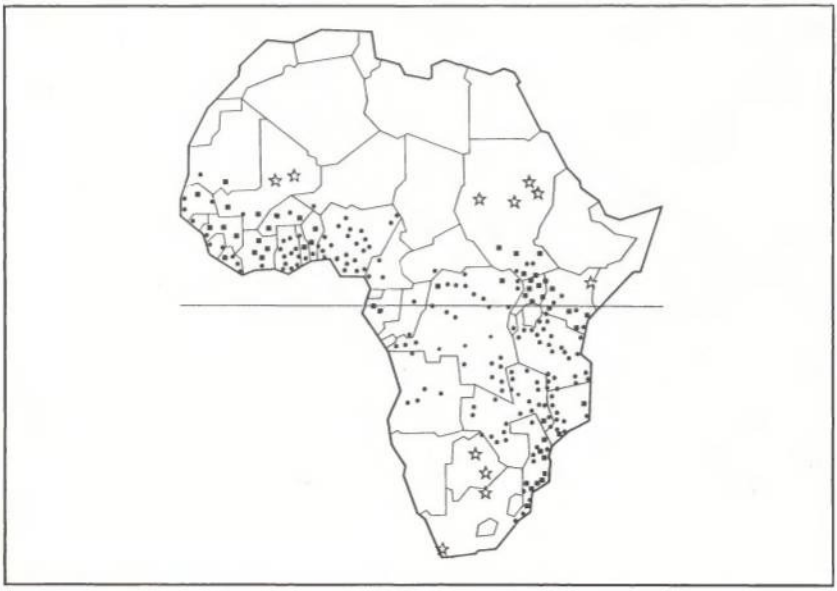

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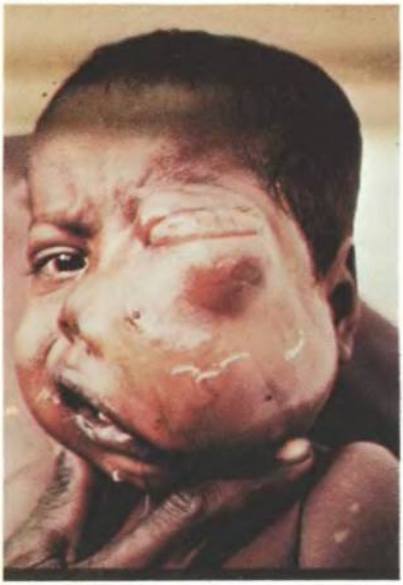

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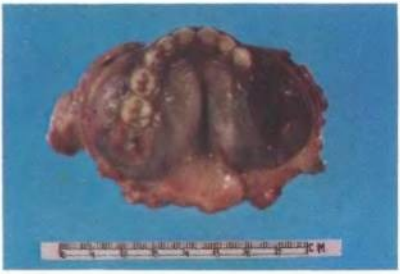

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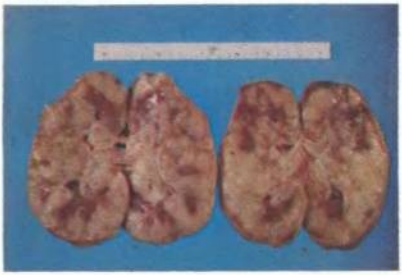


NEOPLASTIC CONDITIONS

Burkitt's Tumour

736 Distribution in Africa The geographical distribution of the tumour in high incidence is mainly controlled by two climatic parameters, temperature and humidity. In tropical Africa as well as other continents the distribution of Burkitt's tumour is roughly the same as that of malaria. Circles indicate several cases, squares show areas of unspecified documentation and stars indicate occasional cases.

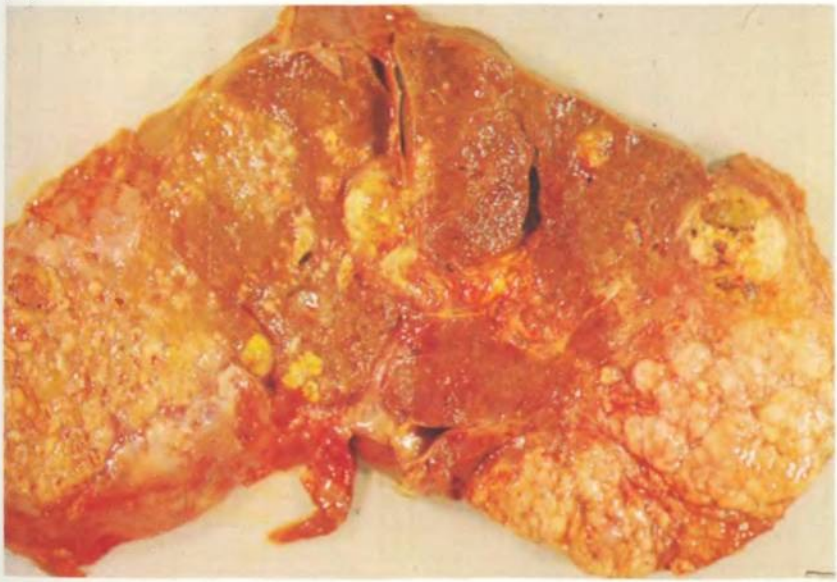
737 & 738 Maxillary tumour One of the most common forms of clinical presentation is that of facial swelling. The jaws, one or more quadrants, are most frequently affected.

739 Kidneys with Burkitt's tumour Massive replacement of both kidneys with tumour cells is apparent in this figure. Any organ can be affected.

Hepatoma

740 Macroscopic appearance of liver Primary carcinoma of the liver is unusually common in the tropics. Coarse nodular changes can be seen in this figure. Aflatoxins in certain diets appear to be an important contributory factor.

740



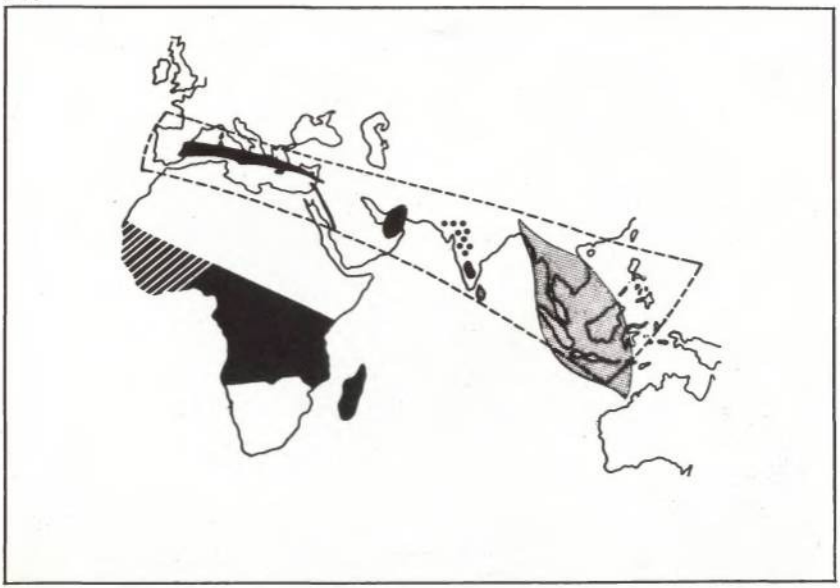
GENETIC BLOOD DYSCRASIAS

741 Distribution map of haemoglobinopathies The most important abnormal haemoglobins in the tropics are Hb S, C. E. Thalassaemia which is a failure of foetal haemoglobin synthesis is also widespread. Hb D has a limited distribution and is clinically mild. The abnormal haemoglobins also occur in Central and South America and among blacks in the United States.

742 Dactylitis due to sickle cell disease Severe bilateral dactylitis is a common presentation of sickle cell disease in children.

743 X-ray of hands in sickle cell disease Note destructive changes in small bones, the result of multiple infarction complicated by infection (in this case by a *Salmonella* species).

741



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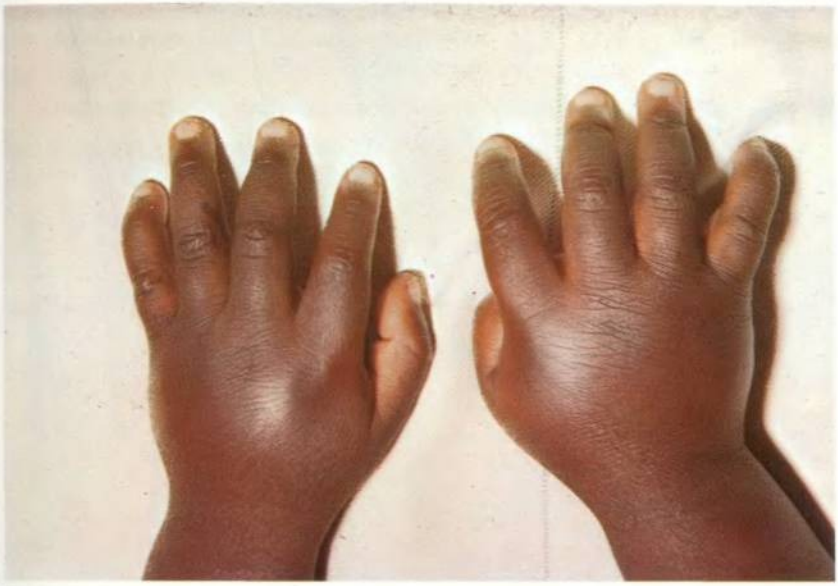

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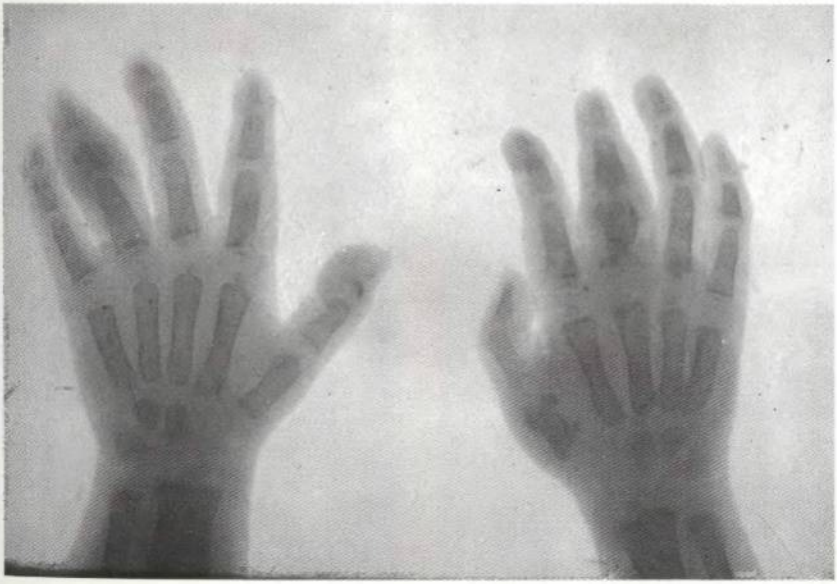
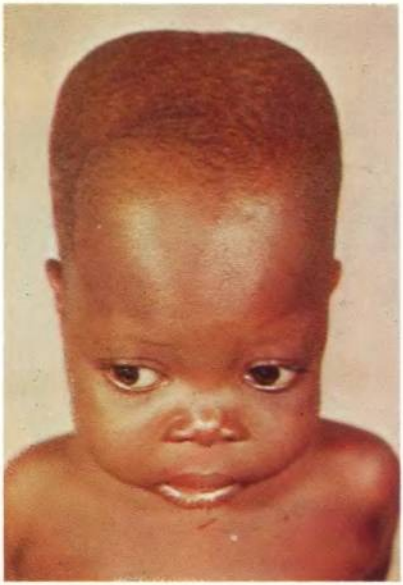


744 Bossing of skull Bossing of the skull due to hyperplasia of the marrow is another feature of sickle cell disease. Similar appearances may be seen in thalassaemia and in any other severe congenital haemolytic anaemia.

745 X-ray of skull in thalassaemia Haemoglobin E disease produces this typical 'hair-on-end' appearance of the skull in X-rays.

744



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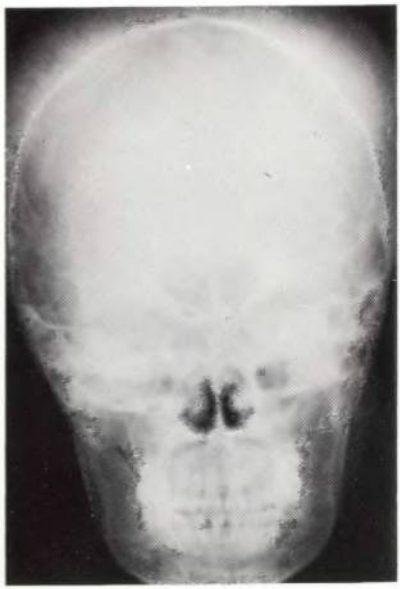

VENOMOUS BITES AND STINGS

Snake Poisoning

Venomous snakes include members of three families, sea snakes (*Hydrophiidae*), cobras and mambas (*Elapidae*) and vipers (*Viperidae*).

746 Elapidae – the cobra *Naja naja* Cobras such as that shown here, and other Elapids produce both neurotoxins and tissue necrotoxins.

747 Viperidae – venom emerging from poison fang The *Viperinae*, and *Crotalinae* (pit vipers) mainly produce toxins affecting the blood and blood vessels.

746

747

748 Myoglobinuria following sea snake bite The figure shows rapid clearance of myoglobinuria following administration of life-saving sea snake antivenom.

749 Ptosis due to elapid bite Elapid venom produces a neuromuscular block especially of cranial nerves, thus affecting vision, swallowing and respiration.

750 Extensive necrosis after cobra bite on leg This patient was bitten several days previously by a cobra.

751 Viper bite causing shock Severe shock associated with serious local tissue damage and haemorrhage.

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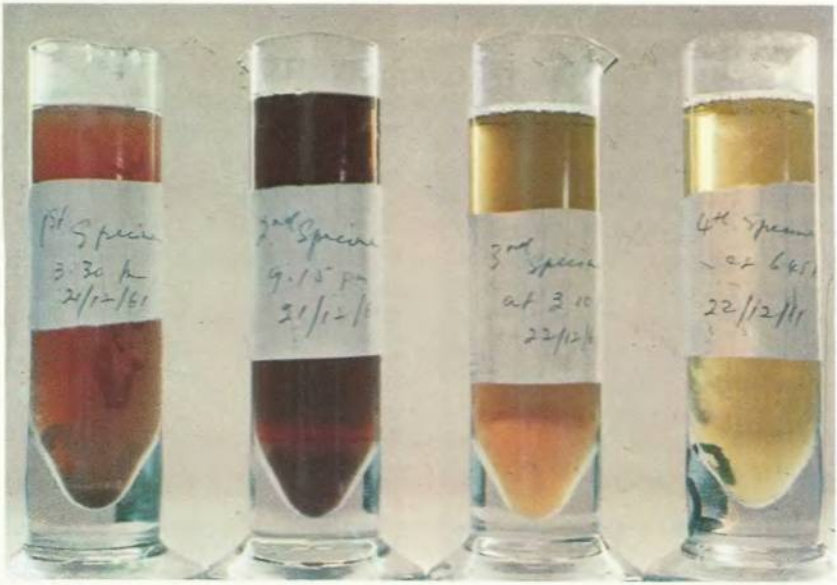

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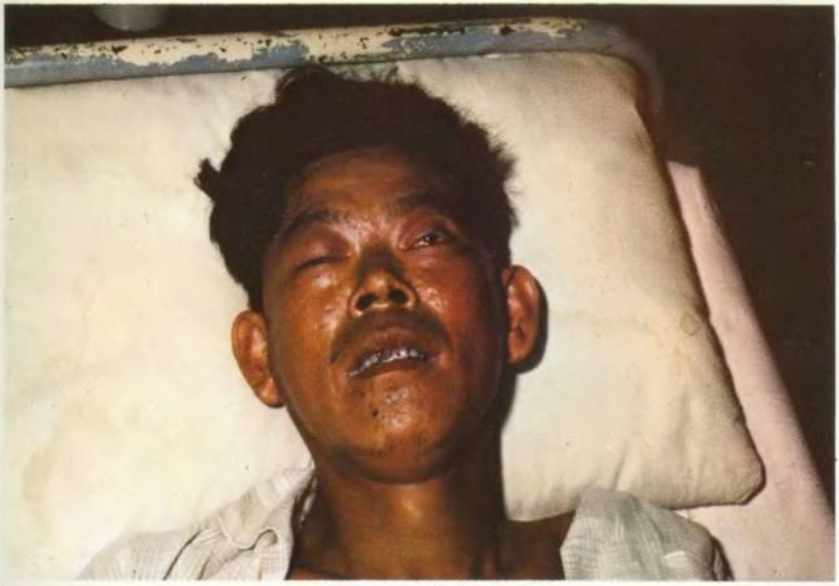

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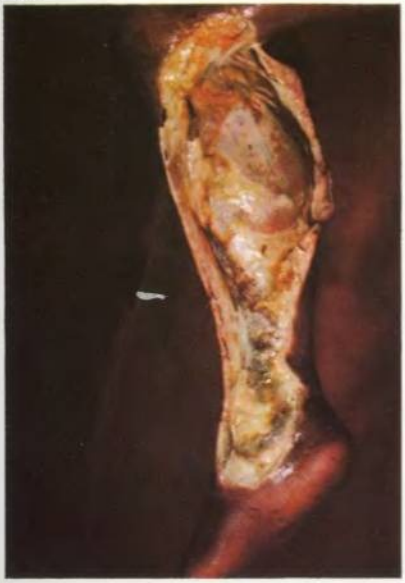

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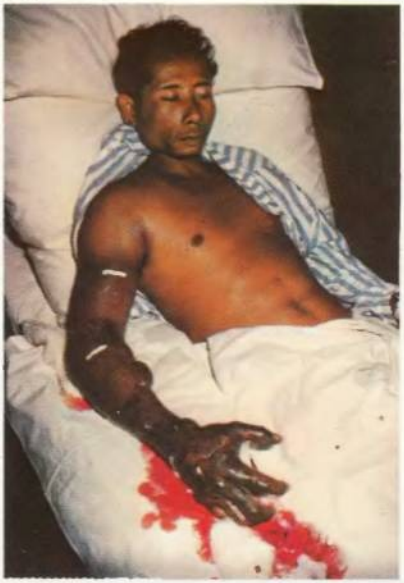

Spider Bites

752 'Black Widow' spider The best known poisonous spider is the 'Black Widow' *Latrodectus mactans*. Humans are bitten only by the females.

753 Sloughing of skin following spider bite This Chilean child was bitten by a *Loxosceles laeta*. Necrosis and sloughing followed severe blistering at the site of the bite and generalised facial oedema.

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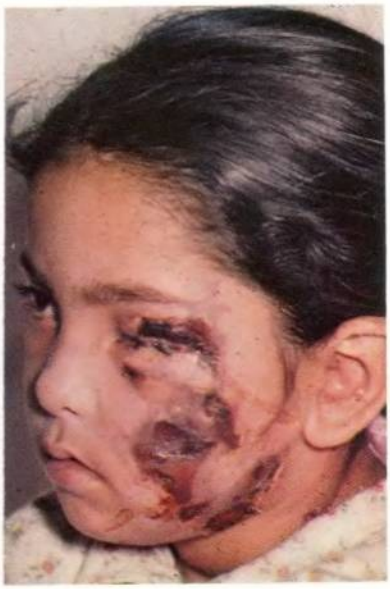

DISEASES OF UNCERTAIN AETIOLOGY OR EPIDEMIOLOGY

Endomyocardial Fibrosis

754 Fibrotic changes in heart of African Endomyocardial fibrosis is a common cause of heart disease in the tropics. It presents in three clinical forms: mainly left-sided as mitral incompetence; right-sided with features suggestive of constrictive pericarditis, and the third form involving both sides of the heart presenting as congestive cardiac failure.

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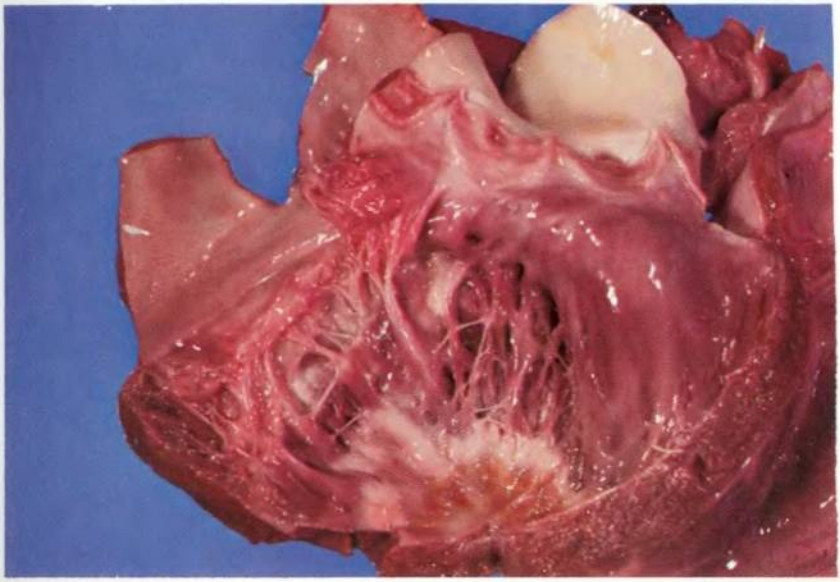

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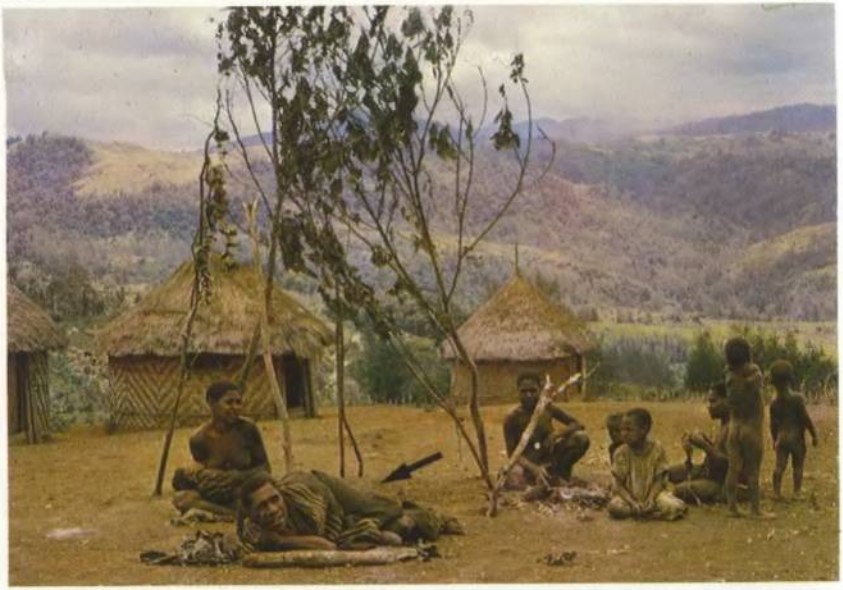

755 Patient with late kuru paralysis Kuru is a fatal disease occurring in the Fore area of New Guinea. It is due to degeneration in the central nervous system, particularly the cerebellum and its connections, resulting in a severe ataxic neuropathy, caused by a 'slow' virus. This was transmitted as a result of the cannibalistic rite of eating the brains of human relatives. The disease has now almost disappeared.

Endemic Goitre

756 Nepalese woman with goitre Endemic goitre is common in many parts of the tropics, especially in mountainous areas, and is probably due directly or indirectly to iodine deficiency.

757 Cretin Endemic cretinism is often seen in areas where endemic goitre occurs.

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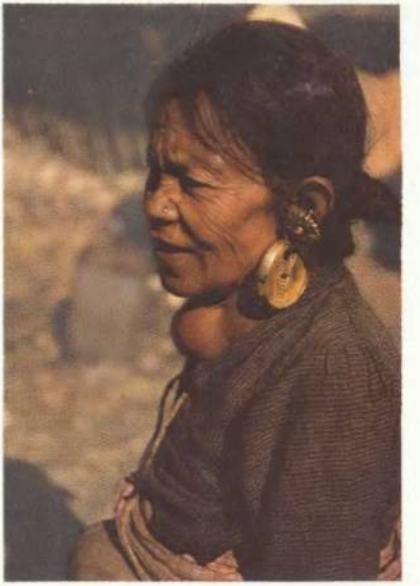

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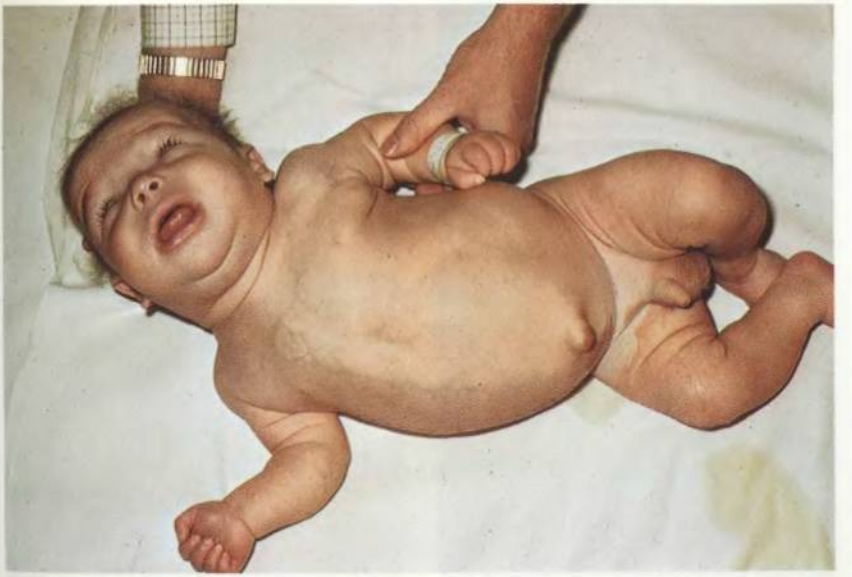

Ainhum

758 & 759 Bilateral ainhum of small toe This condition of unknown aetiology is a progressive encircling fibrosis of a toe, usually the fifth.

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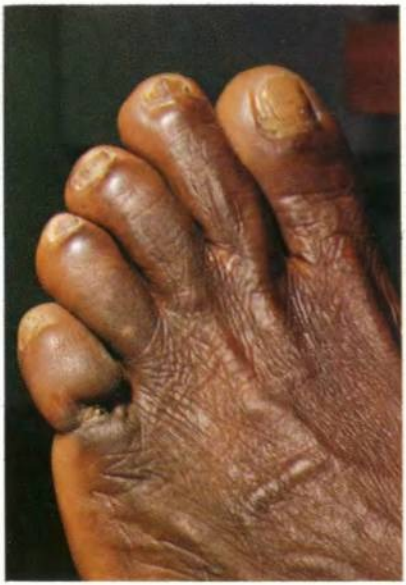

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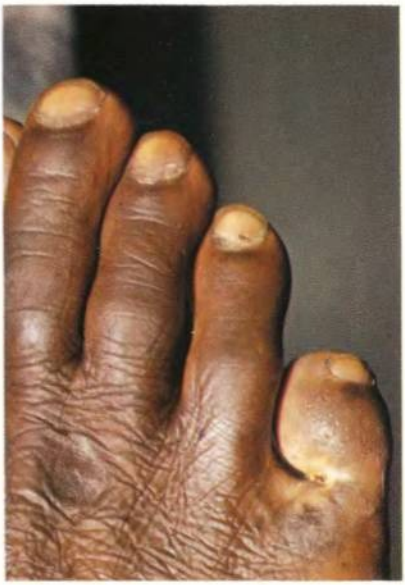

Brazilian Foliaceus Pemphigus

760 Adult Brazilian with 'fogo selvagem' A chronic form of pemphigus is endemic in a number of localities in Central Brazil. The causative agent and mode of transmission are unknown.

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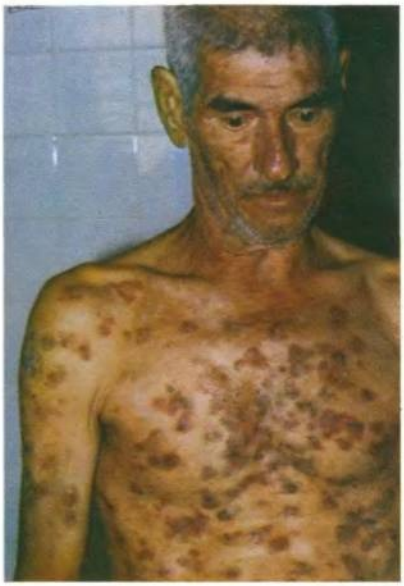

Sprue

761 & 762 X-rays of patient with sprue Sprue is characterised by chronic steatorrhoea with associated abdominal symptoms, glossitis and anaemia. The figures show loss of normal intestinal pattern before (**761**) and recovery after three months of treatment (**762**).

Vesical Calculi

763 Collection of bladder stones Bladder stones are very common in children in North East Thailand and parts of the Middle East. The aetiology is unknown but nutritional factors are implicated.

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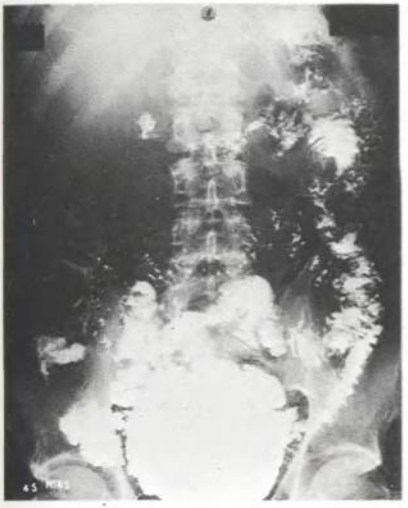

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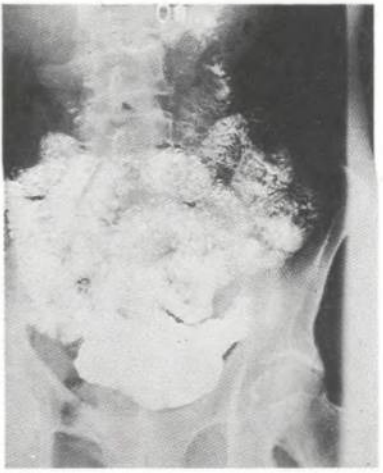

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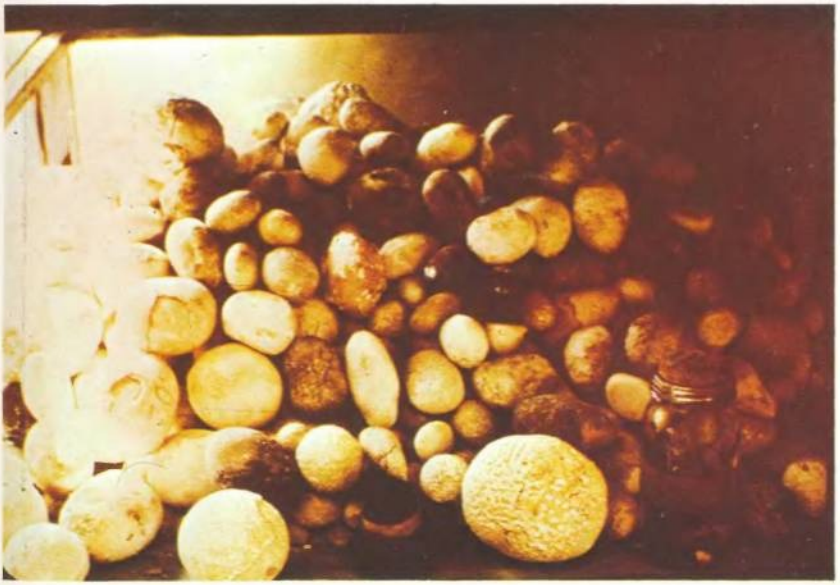

Life Cycles

MALARIA

764 Generalised life cycle The figure is based on the life cycle of *P. vivax* and *P. ovale*. Sporozoites (2) injected by the mosquito enter liver parenchyma cells where they grow into the first generation of pre-erythrocytic schizonts (3). These give rise to cryptozoites which invade red blood cells, to develop into the asexual erythrocytic cycle (4). Some sporozoites in the hepatocytes stay dormant (H = hypnozoite) to mature after an interval of weeks or months into secondary exo-erythrocytic schizonts (3a, 3b). The successive waves of cryptozoites emerging from these give rise to relapse infections in the blood after months or years. Some blood stages mature to form sexual forms, the macro- and microgametocytes (5). These enter the mosquito where the males exflagellate to fertilise the females (6). The ookinete thus produced forms oocysts on the outside of the midgut (7). Sporozoites (8 & 9) develop in the oocysts. The sporozoites enter the mosquito salivary glands (2) where they are ready to infect a new host. (In *P. falciparum* and *P. malariae* stages 3a, 3b do not exist and true relapses do not occur.) (*See also* **63–65**, **66–69**, **94–98**.)

AFRICAN TRYPANOSOMIASIS

765 Cycle in vertebrate and invertebrate hosts A. Trypomastigote stages (2) of *Trypanosoma brucei gambiense* or *rhodesiense* are produced in the blood and tissue spaces of man when he is bitten by a tsetse fly. The parasites may be found in various glands (3) as well as in the blood. B. Trypomastigotes ingested by the tsetse fly transform into epimastigotes (4) which divide (5) during a complicated migration in the fly, eventually forming metacyclic trypomastigotes (6). These can infect another man or reservoir animals (C) in which the blood and tissues are invaded as in man (2). (*See also* **124–129**, **132**, **137**.)

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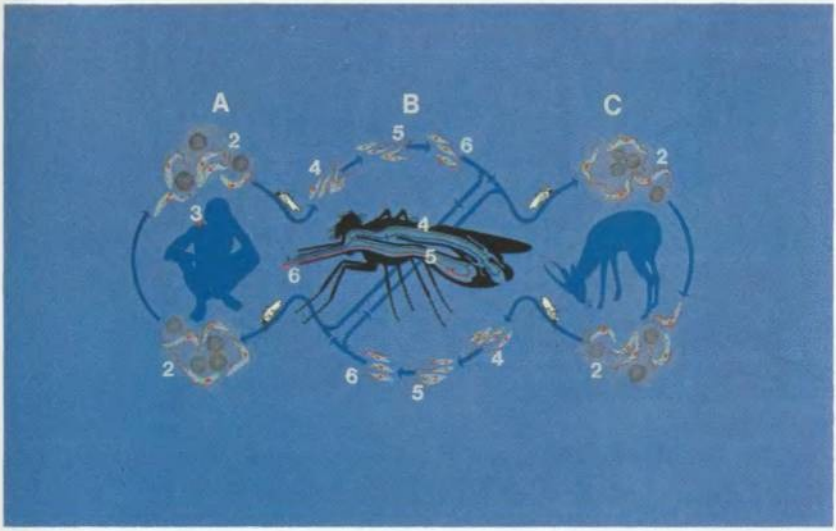

CHAGAS' DISEASE

766 Cycle in man and triatomid bugs Metacyclic trypanosomes (4) of *Trypanosomi cruzi* passed in the faeces of infected triatomid bugs (A) penetrate the skin or mucous membranes to reach the blood (5). They enter various muscular tissues, eg cardiac muscle (B), smooth muscle of the gut (C) or skeletal muscle (D). Here they transform to amastigotes which divide, producing pseudocysts. Subsequently, the daughter amastigotes transform back to trypomastigotes (6, 7). These enter the blood from which they may reinvade muscular tissues, or be picked up by another triatomid bug when it feeds. In the bug the parasites transform to epimastigotes (1, 2, 3) which divide in the gut. Finally some epimastigotes pass to the hindgut where they transform back to infective, metacyclic trypomastigotes (4) which are passed in the faeces as the bug next feeds. (*See also* **145–150**, **154**, **155**, **161**.)

LEISHMANIASIS

767 Cycle in man, animals and sandflies Promastigotes (1) of various *Leishmania* species enter the skin of a vertebrate host (B) when the sandfly (A) bites, and transform into amastigotes which are phagocytosed by macrophages (II). In the macrophages the amastigotes divide (III), finally rupturing the host cell. They then enter either macrophages of the reticuloendothelial organs (IV) or of the skin (V) where they continue to divide. Some parasites circulate in mononuclear macrophages of the blood, and are picked up in these, or with skin macrophages (VI) when another sandfly bites. In the fly (A) they transform into promastigotes in the midgut, then migrate forward or backward (depending upon the species of *Leishmania*) to attach to the gut wall and multiply as promastigotes. Finally, they migrate forward to the pharynx and proboscis (VIII) from which they enter the skin of a new vertebrate host when the fly takes another meal. (*See also* **166–168**, **173**, **180**, **184**, **186**, **212**.)

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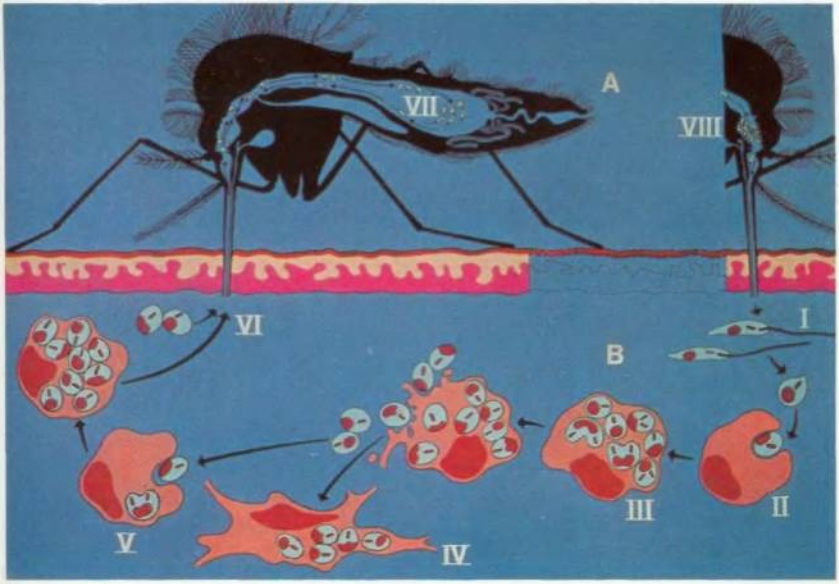

FILARIASIS

768 ***Wuchereria bancrofti*** **and** ***Brugia malayi*** Third-stage infective larvae (a) enter the mouthparts of the mosquito from which they penetrate the skin when the insect bites (b). They enter the lymphatics where the worms mature into thread-like adults (c) which can live for many years. Blockage of lymphatic vessels by the adult worms leads to elephantiasis (d) in some individuals. Larvae (e) are produced in the female worms and transform to microfilariae (f) which enter the peripheral circulation. From there they are picked up by mosquitoes with a blood meal (g). The microfilariae (h) penetrate the insect's gut wall to develop in the thoracic muscles (ij) in which they mature to the third stage larvae. (*See also* **214**, **215**, **225**, **226**, **234**, **245**.)

769 ***Loa Loa*** When the *Chrysops* fly bites, the infective third-stage larvae (a) enter the vertebrate host (b) where they mature into adults (c) within about one year. The adults live for 4 to 12 years. The females (about 7 cm long) migrate through the subcutaneous tissues and may cross the front of the eye under the conjunctiva (d). Microfilariae (f) develop from larvae (e) in the female and circulate in the blood with which they are picked up by another fly (g). In the gut of the fly the microfilariae (h) exsheath and enter the fat bodies in which they mature first to 'sausage-like' forms (ij), then infective third-stage larvae (a). The larvae infect a new host when the *Chrysops* takes another blood meal. (*See also* **256–260**.)

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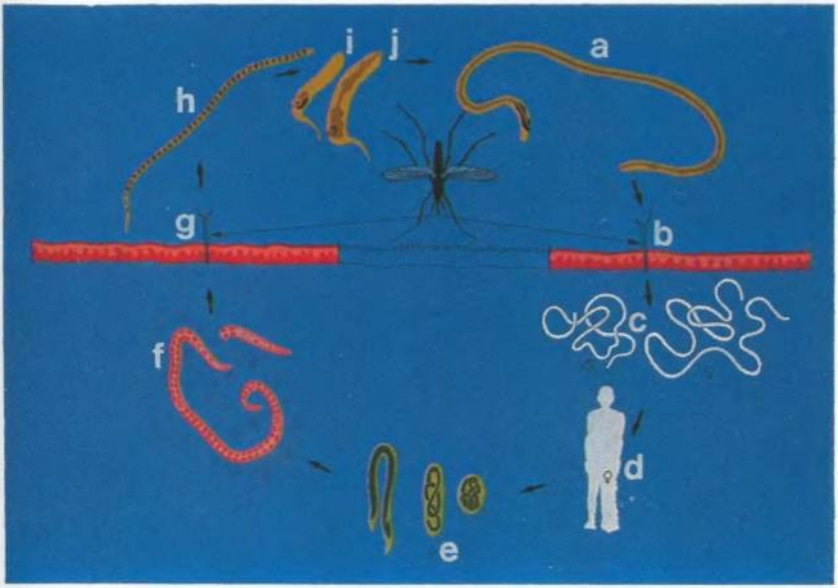

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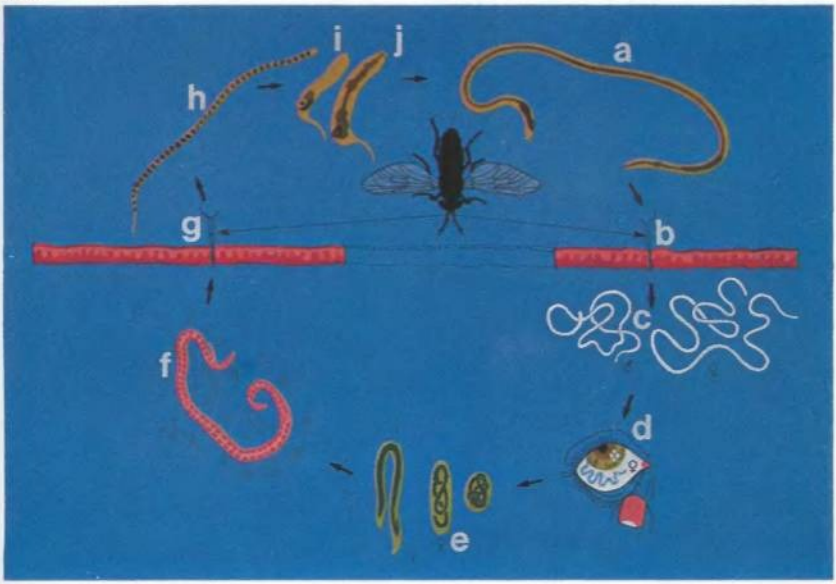

770 ***Onchocerca volvulus*** The infective, third-stage larvae (a) enter the skin following the bite of the blackfly vector, *Simulium* (b). The larvae migrate to the subcutaneous tissues where they mature into thread-like adult males and females (c) in about one year, enclosed in fibrous nodules (d). Larvae (e) developing in the females form unsheathed microfilariae (f) which live in the skin (g) and eye (h). When microfilariae are picked up from the skin by another *Simulium* (i) they develop through several stages (jk) in the thoracic muscles to become infective, third-stage larvae (a) in about 6 to 12 days. (*See also* **264**, **269–271**, **285–287**, **289**.)

HOOKWORM

771 ***Necator americanus*** **and** ***Ankylostoma duodenale*** Adults are attached to the walls of the jejunum (A) by the buccal capsule. Females lay large numbers of eggs which are passed out with the faeces (B). They mature through 4- and 8-segmented stages (C) to larvae which hatch in the soil (D). There they feed on bacteria and undergo two moults to produce filariform, infective larvae (E). These penetrate the skin of a new host (F) usually on the feet. They migrate into venules, entering the right heart (G) and lungs (H). Here they grow before penetrating from the capillaries into the alveoli. They enter the trachea (I), then the pharynx, are swallowed and pass into the small intestine (A) where they mature. (*See also* **301**, **309–318**.)

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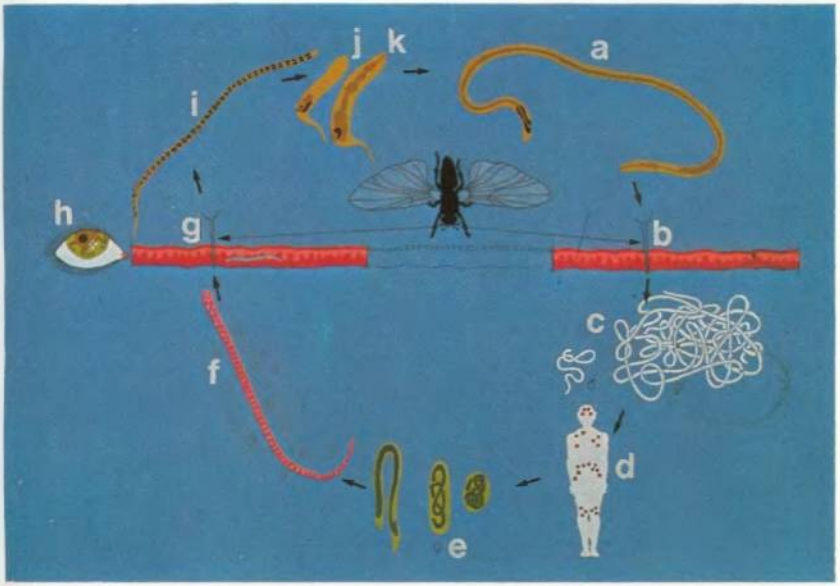

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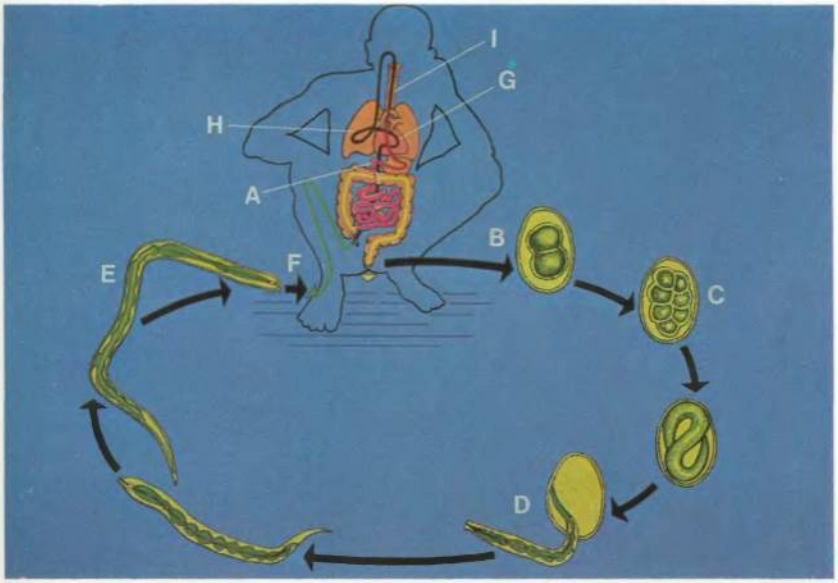

STRONGYLOIDIASIS

772 ***Strongyloides stercoralis*** has two generations, one free-living and the other parasitic. I. *The parasitic generation*. Males are rare and the females are probably parthenogenetic, living in the mucosal glands of the small intestine (A). Eggs usually hatch in these glands into larvae which are passed in the faeces (B). These rhabditiform larvae can develop into filariform larvae (C) in the ground, the infective filariform larvae then penetrating the skin of a new host (D). The rest of the cycle in man is as in hookworm infections. II. *Free-living generation*. Rhabditiform larvae (B) may develop into free-living males and females (E) which lay eggs (F) that hatch in the soil into rhabditiform (G), then infective filariform larvae (H). These too can penetrate the skin (D) and recommence a parasitic cycle in man. III. *Auto-infection*. Rhabditiform larvae (B) can mature into filariform larvae in the intestine, and these can directly penetrate the peri-anal skin, causing auto-infection. IV. *Hyperinfection*. Rhabditiform larvae may penetrate the intestinal mucosa causing massive auto-infection, usually in people with diminished immune responsiveness (cycle not illustrated here). (*See also* **322**, **323**.)

ASCARIASIS

773 ***Ascaris lumbricoides*** adult worms live in the small intestine (A) where they lay large numbers of eggs (B) that are passed out with the faeces. In the soil they can readily contaminate vegetables, for example when nightsoil (C) is used as fertiliser. The larvae develop inside the eggs (D) which are swallowed when they are present in uncooked food (D). The eggs enter the jejunum where the larvae hatch, penetrate the mucosa, and are carried through the hepatic circulation to the heart and lungs (G). There they grow, moulting twice before escaping from the capillaries into the alveoli. They again enter the stomach (H) *via* the trachea and oesophagus, and thence pass to the small intestine where they grow to adulthood. (*See also* **297**, **298**, **329–332**.)

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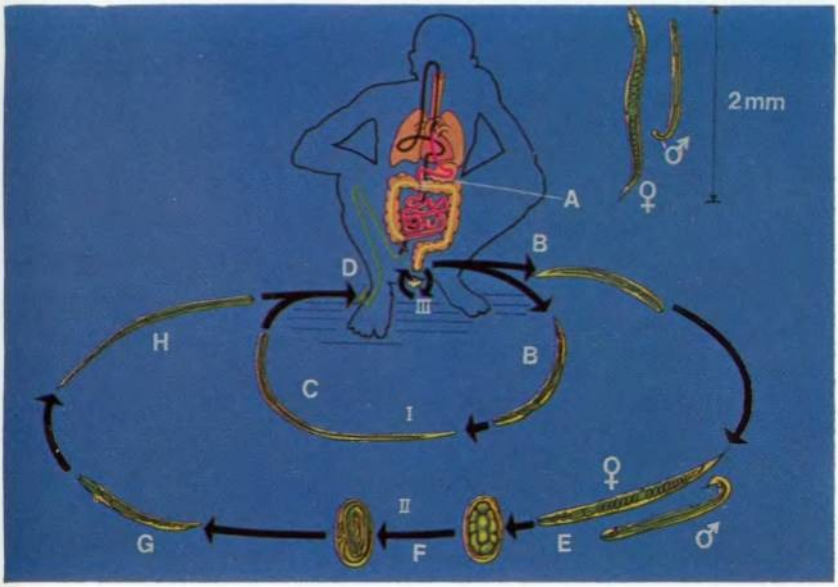

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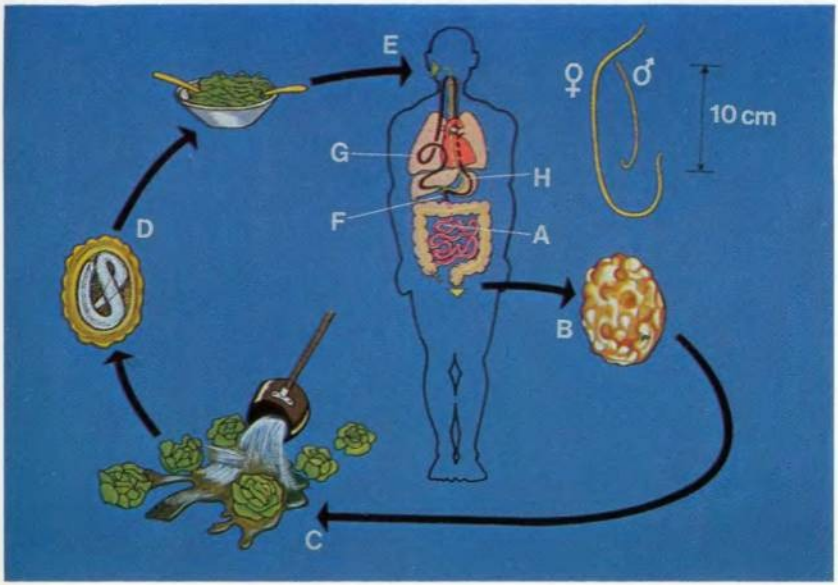

TRICHURIASIS

774 The whipworm, *Trichuris trichiura* adults inhabit the caecum (A) and sometimes the colon and rectum where they are attached to the mucosa. Eggs passed with the faeces (B) mature in the soil to larvae (C, D) which remain in the eggs. Eggs can readily contaminate vegetables, eg when nightsoil is used as fertiliser (E) or sanitary habits are otherwise primitive, and are then swallowed in uncooked food (G). The eggs hatch in the small intestine and the developing larvae pass directly to their attachment sites in the large intestine (A). Females commence egg-laying after about 3 months. (*See also* **302**, **341–343**.)

SCHISTOSOMIASIS

775 Cycle of human schistosomes The three common parasites of man, *Schistosoma haematobium*, *S. japonicum* and *S. mansoni* have a similar life cycle. Eggs passed in urine (*S. haematobium* A) or faeces (*S. japonicum* B, *S. mansoni* C), hatch in aggregations of water such as ponds, lake edges, streams and canals. From the eggs miracidia (D) hatch into the water where they penetrate into suitable snails. In the snails they develop two generations of sporocysts (not shown here) the second of which produces fork-tailed cercariae (E). These penetrate the skin (F) when a new host comes into contact with contaminated water. Once through the skin the cercariae shed their tails and become schistosomulae which migrate through the tissues until they reach the portal venous system of the liver (G). There males and females (H) copulate before settling down in pairs in the venous system of the liver. From there they migrate to the venous plexus of the bladder (I) or rectum (J) where spiny eggs are laid. These penetrate into the bladder or rectum from which they reach the exterior. Eggs laid by worms in the liver itself lead to local fibrotic changes and cirrhosis. (*See also* **293–295**, **347–363**, **366**, **377**, **382**, **397–399**.)

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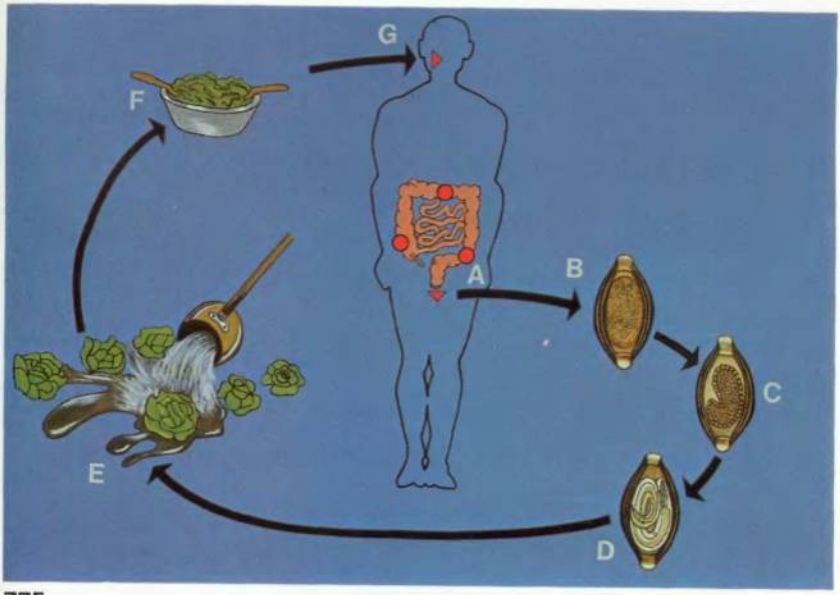

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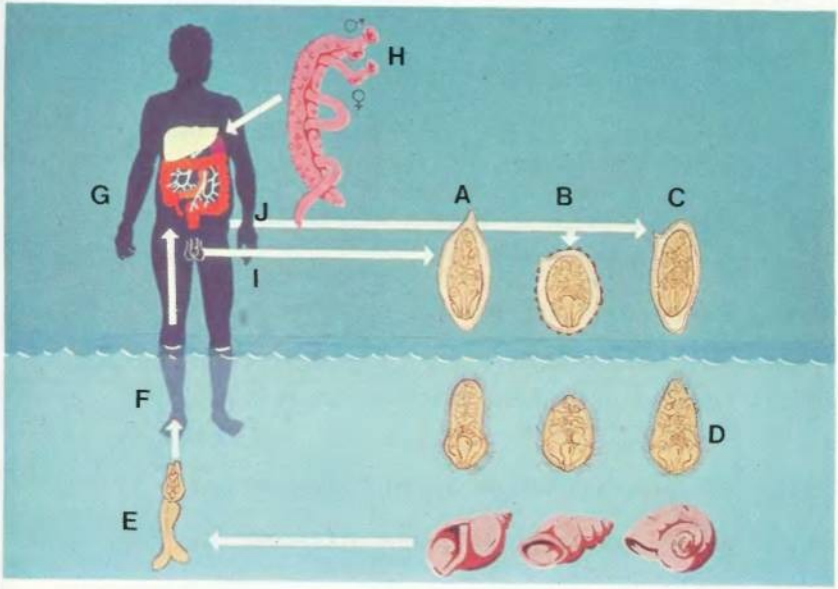

List of Tables

I	Arthropod Vectors of Disease	369
II	The Clinically Recognised Arboviruses of Man	370
III	The Rickettsial Diseases	371
IV	The Protozoa (Subphylum Apicomplexa) of Medical Importance	372
V	The Protozoa (Class Zoomastigophora) of Medical Importance	373
VI	Trypanosomes of Medical and Veterinary Importance	374
VII	The Genus *Leishmania* and the Leishmaniases	375
VIII	The Nematodes of Medical Importance and their Prevalence	377
IX	The Microfilaria Occurring in Man	378
X	The Digenetic Trematodes of Medical Importance and their Prevalence	379
XI	Snails and Other Molluscs of Medical Importance (Class Gastropoda)	380
XII	The Protozoa (Subphyla Ciliophora and Rhizopodea) of Medical Importance	381
XIII	The Cestodes of Medical Importance and their Prevalence in Man	382
XIV	The Superficial and the Systemic Mycoses	383
XV	The Myiasis Producing Diptera of Medical Importance	384

Table I Arthropod Vectors of Disease

Class	Order	Vectors	Disease Transmitted
CRUSTACEA	COPEPODA	*Cyclops* spp. (water fleas)	guinea worm fish tapeworm
	DECAPODA	crayfish, freshwater crabs	paragonimiasis
ARACHNIDA	ACARINA	hard ticks	spotted and Q fevers, virus encephalitides
		Ornithodorus spp. (soft ticks)	endemic relapsing fever
		mites	scrub typhus, Rickettsial pox, scabies
INSECTA	ANOPLURA	*Pediculus humanus* (body louse)	epidemic typhus, epidemic relapsing fever
	MALLOPHAGA	chewing lice	*Dipylidium caninum* (dog tapeworm)
	BLATTARIA	cockroaches	*Hymenolepis diminuta* (rat tapeworm)
	HEMIPTERA	*Reduviidae* (assassin bugs)	Chagas' disease
	DIPTERA	*Anopheles*	malaria, filariasis *(W. bancrofti, B. malayi)*
		culicines	filariasis *(W. bancrofti, B. malayi)*, arboviruses (including yellow fever, dengue)
		Culicoides	filariasis *(M. ozzardi, T. perstans, T. streptocerca)*
		Simulium	filarial blindness *(O. volvulus)*
		Chrysops	filariasis *(Loa loa)*
		Phlebotomus *Lutzomyia*	sandfly fever, leishmaniasis, Bartonellosis
		Glossina	African trypanosomiasis
	SIPHONAPTERA	fleas	plague, murine typhus, rat and dog tapeworms
	COLEOPTERA	beetles	rat tapeworms
	LEPIDOPTERA	grain moths	rat tapeworms

(see also Part V, Ectoparasites)

Table II The Clinically Recognised Arboviruses of Man

Group	Virus	Vector	Reservoir	Amplifier
Alphavirus (= Group A)	Chikungunya	*Aedes* spp.; *Culex* spp.	Baboons	
	O'Nyong-Nyong	***A. gambiae; A. funestus***	*n/k*	
	Mayaro	*Aedes* spp.; *Anopheles* spp.	*n/k*	
	Ross River	*Aedes* spp.	*n/k*	
	Venezuelan equine encephalitis	*Aedes* spp.	Rodents and birds	Horses
	Western equine encephalitis	*Culex* spp.	Birds	Horses
	Eastern equine encephalitis	*Aedes* spp.	Birds	Horses
Flavivirus (= Group B)	Yellow fever	*Aedes aegypti*	Monkeys	
	Dengue fevers: types 1, 2, 3, 4	*Aedes* spp.	*n/k*	?Monkey
	Southeast Asian haemorrhagic fever	*Aedes aegypti*	*n/k*	
	Japanese B encephalitis	*Culex* spp.	Birds	Pigs
	West Nile	*Culex* spp.	Birds	
	Kyasanur Forest disease	Ticks	Rodents, ground-living birds	
Phlebotomus (includes Group C)	Sandfly fever	*Phlebotomus* spp.	*n/k*	
	Rift Valley fever	*Aedes* spp.; *Culex* spp. *Eratmapodites*	Domestic animals	
Bunyaviridae	Congo-Crimean haemorrhagic fever	Ticks	Domestic animals	

(n/k indicates 'not known')

Table III The Rickettsial Diseases*

Group	Disease	Causative Agent	Vector	Animal Reservoir
TYPHUS	Epidemic	*Rickettsia prowazeki*	*Pediculus humanus*	None
	Brill-Zinsser disease	*R. prowazeki*	*P. humanus*	None
	Murine	*R. mooseri*	*Xenopsylla cheopis*	Rat
SPOTTED FEVER	American spotted fevers	*R. rickettsi*	various species of ticks	many species of small mammals
	Fièvre boutonneuse	*R. conori*		
	Siberian tick typhus	*R. siberica*		
	Queensland tick typhus	*R. australis*		
	Rickettsial pox	*R. akari*	Mites	Mice
	Scrub typhus	*R. tsutsugamushi*	Trombiculid mites	Small mammals, mice, and field rats
	Trench fever	*R. quintana*	*P. humanus*	None
	Q fever	*Coxiella burneti*	None (ticks)	Cattle, sheep, goats and wild animals

*(*Identification is made on the basis of serological typing – eg Weil Felix reaction*)

Table IV The Protozoa (Phylum Apicomplexa) of Medical Importance

Phylum etc.	Family	Genus and Species
SARCOMASTIGOPHORA (*see* Tables V and XII)		
CILIOPHORA (*see* Table XII)		
APICOMPLEXA		
Subclass PIROPLASMIA	BABESIIDAE*	*Babesia microtus* *B. bovis* *Babesia* spp.
Subclass COCCIDIA		
Order EUCOCCIDIIDA		
Suborder HAEMOSPORINA	PLASMODIDAE	*Plasmodium vivax* *P. ovale* *P. malariae* *P. (Laverania) falciparum*
Suborder EIMERIINA	SARCOCYSTIDAE	*Toxoplasma gondii* *Sarcocystis hominis*** *S. suihominis**** *Sarcocystis* spp.****
	EIMERIIDAE	*Isospora belli*

*All rare in man
**Man definitive, cattle intermediate host
***Man definitive, pig intermediate host
****'*Sarcocystis lindemanni*' is a number of unidentified species for which man is an intermediate host

Table V The Protozoa (Class Zoomastigophorea) of Medical Importance

Phylum	Subphylum	Order	Genus and Species
APICOMPLEXA (*see* Table IV)			
CILIOPHORA (*see* Table XII)			
SARCOMASTIGOPHORA	SARCODINA (*see* Table XII)		
	MASTIGOPHORA		
	Class ZOOMASTIGOPHOREA	RETORTAMONADIDA	*Chilomastix mesnili*
	(No vector)	DIPLOMONADIDA	*Giardia lamblia*
		TRICHOMONADIDA	*Trichomonas vaginalis* *T. hominis* *Dientamoeba fragilis**
	(Invertebrate vector)	KINETOPLASTIDA	*Leishmania* spp. (*see* Table VII) *Trypanosoma* spp. (*see* Table VI)

*This parasite is now considered to be a flagellate and not an amoeba.

Table VI Trypanosomes of Medical and Veterinary Importance

Genus & Subgenus		Species	Host Species	Disease
in Africa				
SALIVARIA	*Trypanosoma (Duttonella)*	*vivax*	antelopes, ruminants, equines, dogs	Souma
		uniforme	antelopes, ruminants	(pathogenic)
	T. (Nannomonas)	*congolense*	antelopes, ruminants, equines, pigs, dogs	(pathogenic)
		simiae	pigs, warthogs, camels	(pathogenic)
	T. (Trypanozoon)	*brucei brucei*	antelopes, domestic mammals	Nagana
		b. rhodesiense	antelopes, man	Sleeping sickness (acute form)
		b. gambiense	man, pigs	Sleeping sickness (chronic form)
		*evansi**	bovines, equines, camels, dogs, etc.	Surra
		*equiperdum***	equines	Dourine
	T. (Pycnomonas)	*suis*	domestic and wild pigs	(pathogenic)
in South America				
SALIVARIA	*Trypanosoma (Duttonella)*	*vivax****	bovines	(pathogenic)
	T. (Herpetosoma)	*rangeli*****	many wild animals, man	(non-pathogenic)
STERCORARIA	*T. (Schizotrypanum)*	*cruzi*****	man, armadillos, opposums, dogs, etc.	Chagas' disease

All species transmitted by tsetse flies except
*by tabanid flies
**by coitus
***by various biting flies
****by reduviid bugs

Table VII The Genus Leishmania and the Leishmaniases

Type of Disease	Species	Localities	Main Vectors	Main Reservoirs
VISCERAL LEISHMANIASIS (KALA-AZAR) 60% between 10 and 20 years of age	*L. donovani*	India	*P. argentipes*	man
	L. infantum complex	China (N. of Yangtze)	*P. chinensis*	dog, ? fox
		USSR	*P. major*	
		Iraq	*P. major*	? jackal
		Sudan	*P. langeroni orientalis*	rodents, serval cat, genet
		Kenya	*P. martini*	rodents
		Uganda		
INFANTILE KALA-AZAR 80–90% under 10 years old	*L. infantum* complex	France, Mediterranean basin	*P. ariasi*, *P. perniciosus*, *P. major*	fox, dog
	'L. *chagasi*'	Brazil, Paraguay, Venezuela	*L. longipalpis*	fox, dog
POST K-A DERMAL LEISHMANOID	*L. donovani*, *L. infantum* complex	(as classical kala-azar)		
ORONASAL K-A	*L. infantum* complex	Sudan, Ethiopia		may lead to man-man spread
CUTANEOUS LEISHMANIASIS (OLD WORLD)	*L. tropica tropica* (= *minor*)	India	*P. sergenti*	dog
		Mediterranean basin, Middle East	*P. papatasi*	rodents, ? dog, gerbils
		Iran	*P. ansarii*	dog
		USSR (urban)	*P. mongolensis*	rodents, gerbils
ORIENTAL SORE	*L. major*	Iran	*P. caucasicus*	gerbils, merions
		USSR (rural)	*P. papatasi*	
		Saudi Arabia	?	
		Libya	?	
	L. tropica complex	Tanzania	?	?
		Senegal	*P. duboscqi*	rodents
		Namibia	*P. rossi*	hyrax spp.
SINGLE SORE AND DIFFUSA	*L. aethiopica*	Ethiopia	*P. longipes*	hyrax spp.
		Kenya	?	?

Table VII (continued)

Type of Disease	Species	Localities	Main vectors	Main Reservoirs
CUTANEOUS AND MUCO-CUTANEOUS (NEW WORLD)	*L. mexicana mexicana*	Mexico Guatemala Honduras	*L. olmeca*	forest rodents
SINGLE SORE AND DIFFUSA	*L. m. pifanoi*	Venezuela	? *L. panamensis* ? *L. flaviscutellata*	forest rodents
	L. m. amazonensis	Amazon basin ? Trinidad	*L. flaviscutellata*	forest rodents, opossums
	L. mexicana complex	Costa Rica Panama Matto Grosso	?	forest rodents
ESPUNDIA	*L. braziliensis braziliensis*	Brazil* Peru Ecuador Bolivia Venezuela Paraguay Colombia	*L. pessoai* *L. intermedia* *L. wellcomei* *L. paraensis* ? *L. migonei* ? *L. whitmani* ? *L. anduzei*	forest rodents forest rodents ? forest rodents, ? paca
PIAN BOIS	*L. braziliensis guyanensis*	Guyanas Brazil (Northern states) ? Venezuela	*L. umbratilis* *L. whitmani*	sloths and other arboreal mammals
USUALLY SINGLE SORES, SOME LYMPHATIC SPREAD	*L. braziliensis panamensis*	Panama ? Central America to Colombia	*L. trapidoi* *L. ylephiletor* *L. gomezi* *L. panamensis*	sloths marmoset kinkajou olingo
UTA	*L. peruviana*	Peru (West of Andes)	? *L. noguchii* *L. verrucarum* *L. peruensis*	dog

*Forested areas East of Andean chain

Table VIII The Nematodes of Medical Importance and Their Prevalence*

Subclass	Order (Suborder)	Superfamily	Genus and Species	Probable Prevalence in Man
ADENOPHOREA	ENOPLIDA	TRICHUROIDEA	*Trichinella spiralis*	39 million
			Trichuris trichiura	536 million
			Capillaria hepatica	rare
			C. philippensis	thousands
SECERNENTEA	RHABDITIDA	RHABDITOIDEA	*Strongyloides stercoralis*	56 million
	STRONGYLIDA	ANKYLOSTOMATOIDEA	*Ankylostoma duodenale* / *Necator americanus*	716 million
			A. caninum	thousands
			A. braziliense	thousands
			A. ceylanicum	rare
			Ternidens deminutus	thousands
			Oesophagostomum apiostomum	rare
			Syngamus laryngeus	rare
		TRICHOSTRONGYLOIDEA	*Trichostrongylus* spp.	8 million
		METASTRONGYLOIDEA	*Metastrongylus elongatus*	rare
			Angiostrongylus cantonensis	thousands
	OXYURIDA	OXYUROIDEA	*Enterobius vermicularis*	291 million
	ASCARIDIDA	ASCARIDOIDEA	*Ascaris lumbricoides*	986 million
			Toxocara canis	thousands
			Toxocara cati	thousands
			Lagochilascaris minor	rare
			Anisakis spp.	rare
	SPIRURIDA (SPIRURINA)	SPIRUROIDEA	*Gongylonema pulchrum*	rare
		GNATHOSTOMATOIDEA	*Gnathostoma spinigerum*	rare
		THELAZOIDEA	*Thelazia callipaeda*	rare
		FILARIOIDEA	*Wuchereria bancrofti* / *Brugia malayi*	296 million
			Loa loa	26 million
			Onchocerca volvulus	39 million
			T. petalonema perstans	52 million
			T. streptocerca	? million
			Mansonella ozzardi	12 million
			Dirofilaria spp.	rare
	SPIRURIDA (CAMALLANINA)	DRACUNCULOIDEA	*Dracunculus medinensis*	79 million

*Updated in part from Le Riche (1967) in *Health of Mankind*, CIBA Symposium (Churchill, London) p. 38.

Table IX The Microfilariae Occurring in Man

in Blood	Sheathed		TAIL	nuclei not to tip	*Wuchereria bancrofti*
			HEAD	nuclei almost to tip	
			BODY	244–296μm smooth curves	
			SHEATH	unstained with Giemsa	
			TAIL	2 tiny nuclei in terminal thread	*Brugia malayi*
			HEAD	clear cephalic space	
			BODY	177–230μm kinked	
			SHEATH	pink stain with Giemsa	
			TAIL	nuclei to tip often bent on body	*Loa loa*
			BODY	250–300μm kinked	
			SHEATH	unstained with Giemsa	
	Unsheathed		TAIL	nuclei not to tip pointed	*Mansonella ozzardi*
			BODY	200μm × 5μm (New World and Caribbean)	
			TAIL	nuclei to tip rounded	*Tetrapetalonema perstans*
			BODY	200μm × 5μm (All tropics)	
in Skin	Unsheathed		TAIL	nuclei not to tip	*Onchocerca volvulus*
			HEAD	spatulate	
			BODY	285–368μm 150–287μm × 8μm	
			TAIL	nuclei to tip crooked	*Tetrapetalonema streptocerca*
			HEAD	single column 10–12 nuclei, then double column	
			BODY	180–240μm × 5μm	

Table X The Digenetic Trematodes of Medical Importance and Their Prevalence

Superorder	Order	Superfamily	Genus & Species	Estimated Cases (minimum)	No.
ANEPITHELIO-CYSTIDA	STRIGEATOIDEA	SCHISTOSO-MATOIDEA	*Schistosoma mansoni*	57 million	1
			S. haematobium	78 million	2
			S. japonicum	69 million	3
			S. intercalatum	thousands	4
			S. mattheei	rare	5
			S. bovis	rare	6
			S. rodhaini	rare	7
	ECHINOSTOMIDA	PARAMPHISTO-MATOIDEA	*Gastrodiscoides hominis*	rare*	8
			Watsonius watsoni	rare	9
		ECHINOSTO-MATOIDEA	*Fasciola hepatica*	thousands	10
			F. gigantica	rare	11
			Fasciolopsis buski	15 million	12
			Echinostomum ilocanum	rare**	13
EPITHELIO-CYSTIDA	PLAGIORCHIIDA	PLAGIOR-CHIOIDEA	*Dicrocoelium dendriticum*	rare	14
			Troglotrema salmincola	rare	15
			Paragonimus westermani	5 million	16
			P. africanus	rare	17
			P. uterobilateralis	thousands	18
	OPISTHORCHIIDA	OPISTHOR-CHIOIDEA	*Opisthorchis felineus*	1 million	19
			O. viverrini	2 million	20
			Clonorchis sinensis ***	28 million	21
			Heterophyes heterophyes	thousands	22
			Metagonimus yokogawai	thousands	23

(Note: The numbers in the right-hand column refer to Table XI in which are indicated the molluscs that serve as intermediate hosts for these trematodes.)

*Said to be common in Assam
**Said to be common in Philippines
***Now referred to as *Opisthorcis sinensis*

Table XI Snails and Other Molluscs of Medical Importance (class Gastropoda)

(The relation of the genera to the helminths that infect them is indicated by reference to the numbers in Table X. The most important helminths are in bold type.)

Subclass	Order	(Suborder)	Family	Genera & Species	Helminths
STREPTO-NEURA	PROSOBRAN-CHIATA	fresh water	AMNICOLIDAE	*Oncomelania* spp.	**3**
				Parafossarulus spp.	**21**
				Bithynia spp.	**19**, **20**, **21**
			THIARIDAE	*Thiara granifera*	**16**, 23
			PLEUROCERIDAE	*Semisulcospira libertina*	**16**, 23
			PILIDAE	*Pila conica*	13
		brackish and sea water	POTAMIDAE	*Cerithidia congulata*	22
				Pirenella conica	22
EUTHY-NEURA	PULMONATA	BASOM-MATOPHORA fresh water	PLANORBIDAE	*Biomphalaria* spp.	**1**, 7
				Bulinus (Bulinus) spp.	**2**, 6
				Bulinus (Physopsis) spp.	**2**, 4, 5, 6
				Segmentina hemisphaerula	**12**
				S. trochoideus	**12**
				Hippeutis cantori	**12**
				H. umbilicalis	13
				Gyraulus convexiusculus	13
				G. prashadi	13
				Indoplanorbis exutus?	8
			ANCYLIDAE	*Ferrissia tenuis*	**2**
			LYMNAEIDAE	*Fossaria* spp.	10, 11
				Lymnaea spp.	10, 11
				Stagnicola bulimoides	10
				Pseudosuccinia columella	10, 11
		STYLOM-MATOPHORA land snails	HELICIDAE	*Helicella candidula*	14
			ENIDAE	*Zebrina detrita*	14
			CIONELLIDAE	*Cionella lubrica*	14
				Achatina fulica	**
				Bradybaena similaris	**
				Subulina octona	**
		SYSTELLOM-MATOPHORA	VERONICELLIDAE	*Veronica leydigi*	**

Angiostrongylus cantonensis. The land planarian **Geoplana septemlineata** has also been found infected with this nematode.

Table XII The Protozoa (Phyla Ciliophora and Sarcomastigophora) of Medical Importance

Phylum etc.	Subphylum etc.	Order	Genus and Species
APICOMPLEXA (*see* Table IV)			
CILIOPHORA	CILIATEA	TRICHOSTOMATIDA	*Balantidium coli*
SARCOMASTIGO-PHORA	MASTIGOPHORA (*see* Tables V, VI, VII)		
	SARCODINA Superclass RHIZOPODA	AMOEBIDA	*Entamoeba coli* *E. histolytica* *E. gingivalis* *Endolimax nana* *Iodamoeba bütschlii* *Acanthamoeba culbertsoni** *Acanthamoeba* spp.*
		SCHIZOPYRENIDA	*Naegleria fowleri**

*Pathogenic 'free-living' species. The other species are obligatory parasites, some of which may be non-pathogenic.

Table XIII The Cestodes of Medical Importance and Their Prevalence in Man*

Order	Family	Genus & Species	Estimated Cases (minimum)
PSEUDOPHYLLIDEA	DIPHYLLOBOTHRIIDAE	*Diphyllobothrium latum*	13 million
		Spirometra spp.	rare
		'Sparganum' spp.	rare
	ANOPLOCEPHALIDAE	*Bertiella* spp.	rare
	DAVAINEIDAE	*Raillietina* spp.	rare
	LINSTOWIIDAE	*Inermicapsifer* spp.	rare
CYCLOPHYLLIDEA	MESOCESTOIDIDAE	*Mesocestoides* spp.	rare
	DILEPIDIDAE	*Dipylidium caninum*	rare
	HYMENOLEPIDIDAE	*Hymenolepis nana*	29 million
		H. diminuta	rare
	TAENIIDAE	*Taenia solium*	4 million
		T. saginata	61 million
		Multiceps multiceps	rare
		Echinococcus granulosus	thousands
		E. multilocularis	rare

*See footnote to Table VIII.

Table XIV The Superficial and the Systemic Mycoses

Group	Clinical Syndrome	Causative Agents
SUPERFICIAL (*dermatomycoses*)		
Mainly hair affected	Black piedra	*Piedraia hortae*
	White piedra	*Trichosporon beigelii*
	Favus	*Trichophyton schoenleinii*
	Trichophytosis (Tinea barbae)	*Trichophyton* spp.
	Microsporosis (Tinea capitis)	*Microsporon* spp.
Hair not affected	Tinea cruris	*Epidermophyton* spp.
	Tinea pedis	*Trichophyton* spp.
	Tinea unguium	*Candida albicans*
	Tinea corporis	*Microsporum* spp.
	Pityriasis versicolor	*Malassezia furfur*
	Tinea imbricata	*Trichophyton concentricum*
	Otomycosis	various genera
SYSTEMIC	Actinomycosis	*Actinomyces bovis*, *Nocardia* spp., wide variety of organisms including *Streptomyces* spp.
	Madura foot (Mycetoma)	*Madurella* spp., *Indiella* spp. etc.
	Chromoblastomycosis (Mossy foot)	*Phialophora* spp., *Cladosporium carrionii*
	Keloidal blastomycosis (Lôbo's disease)	*Lôboa lôboi*
	North American blastomycosis	*Blastomyces dermatitidis*
	South American blastomycosis	*Paracoccidioides brasiliensis*
	Coccidioidomycosis	*Coccidioides immitis*
	Cryptococcosis (Torulosis)	*Cryptococcus neoformans*
	Systemic candidiasis	*Candida* spp.
	Sporotrichosis	*Sporothrix schenckii*
	Histoplasmosis	*Histoplasma capsulatum*
	African histoplasmosis	*H. duboisii*
	Phycomycosis	various genera
	Rhinosporidiosis	*Rhinosporidium seeberi*
	Aspergillosis	*Aspergillus fumigatus*

Table XV The Myiasis Producing Diptera of Medical Importance

Family	Subfamily	Genus & Species	Other Names
CALLIPHORIDAE	CALLIPHORINAE	'metallic group'	
		Chrysomyia bezziana	Old-world screw-worm
		Callitroga hominivorax	New-world screw-worm
		Lucilia spp.	green bottles
		Calliphora	blue bottles
		'non-metallic group'	
		Auchmeromyia luteola	Congo floor maggot
		Cordylobia anthropophaga	Tumbu or mango fly
	SARCOPHAGINAE	*Wohlfahrtia* spp.	flesh flies
		Sarcophaga spp.	
OESTRIDAE		*Dermatobia hominis*	
		Hypoderma spp.	'larva migrans'
		Gasterophilus spp.	

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Index

References in bold are to captions and picture numbers, those in light type to page numbers, and those in Roman numerals to Table numbers (on pages 368–384).

Achatina fulica, **457**, **460**
Actinomyces, **667**
Aedes, **3**, **6**, **14**, **16**, **17**, **24**, **216**, **220**
African green monkey, **733A**
Ainhum, **758**, **759**
Aleppo button, *see* Dermal leishmaniasis
Amblyomma hebraeum, **37**
Amoebae, *Dientamoeba fragilis*, **491**
–differentiation, **487–495**
–*Entamoeba coli*, **487**, **492**
–*Entamoeba histolytica*, **448**, **493**
–*Entamoeba nana*, **489**, **494**
–*Iodamoeba bütschlii*, **495**
Amoebiasis, diagnosis, **496**, **497** **500–505**, **509–511**
–epidemiology, **234**
–extraintestinal, **498**, **508–510**, **662–664**
–immunity, **511**
–liver abscess, **501–507**
Anaemia, **71**, **84**, **319**, **320**, **567**, **725**
Angiostrongyliasis, **455–461**
Angiostrongylus cantonensis, **455**, **456**, **460**, **461**
Angular stomatitis, **722**
Anisakiasis, **551**, **552**
Anisakis, **551**
Ankylostoma spp., **313**, **315**, **321** *see also* Hookworm
Ankylostoma duodenale, **301**, **308–312**, **314**, **317**, **318**, **321**, **771**
Anopheles, **4**, **9**, **15**, **59–63**, **65**, **216**
Arboviruses, II
Armadillo, **152**
Armillifer armillatus, **608**, **609**
Arthropod vectors, *see* I, Assassin bugs, *Chrysops*, *Culicoides*, Ectoparasites, Fleas, Kissing bugs, Lice, Mosquitoes, Reduviid bugs, Sandflies, *Simulium*, Ticks, Triatomid bugs, Trombiculid mites, Tsetse flies
Ascariasis, **329**, **332–335**, **773**
Ascaris lumbricoides, **297**, **298**, **329–331**, **773**
Assassin bugs, **147**
Auchmeromyia luteola, **691**

Babesiasis, **121**, **122**
Bacteria, *Bartonella bacilliformis*, **57**
–*Brucella* spp., 246
–*Clostridium welchii*, **482**
–coliforms, 242
–*Donovania granulomatosis*, **647**
–*Mycobacterium leprae*, **631**, **632**
–*Mycobacterium ulcerans*, **641**, **642**
–*Neisseria meningitidis*, **702**
–*Pseudomonas pseudomallei*, **484**
–*Vibrio cholera*, **477**
–*Yersinia pestis*, **41**
Baghdad boil, *see* Dermal leishmaniasis
Balanitis, **510**
Balantidiasis, **533**
Balantidium coli, **532**
Bartonella bacilliformis, **57**
Bartonellosis, **57**, **58**
Bat, **624**
Bedsonia, **618**
Beef tapeworm, **587–590**
Bejel, **658**
Beri beri, **720**, **721**
Big spleen disease, **118–120**

Biomphalaria spp., **345**, **346**, **361**, **364**, **402**, **403**
Bithynia spp., **419**, **426**
Bitot's spots, **713**
Black enamel skin, **709**
Black flies, *see Simulium*
Black sickness, *see* Visceral leishmaniasis
Black vomit, **19**
Blackwater fever, **84**, **85**
Black widow spider, **752**
Bladder, calculi, **763**
–schistosomiasis, **381–386**, **389**, **390**
–worm, **599**
Blood films in, babesiasis, **121**, **122**
–Bartonellosis, **57**
–Chagas' disease, **145**, **146**
–filariasis, **234–246**, **254**
–hookworm, **320**
–kala-azar, **180**
–malaria, *see Plasmodium*
–preparation, **74–77**
–relapsing fever, **52**, **55**, **56**
–trypanosomiasis, **124**, **125**, **165**
Blood pathology in, babesiasis, **121**, **122**
–diphyllobothriasis, **567**
–folic acid deficiency, **725**, **726**
–haemoglobinopathies, **92**, **741–745**
–kala-azar, **180**
–malaria, **71**, **92**
–snakebite, **748**
Bone, **644**, **655**, **668**, **672**, **682**, **706**, **715–719**, **737**, **738**, **742–745**
Bone marrow, **184**, **726**
Borrelia duttoni, **52–54**
Borrelia recurrentis, **55**, **56**
Borrelia vincenti, **643**
Bossing of skull, **718**, **744**
Bottle feeding, **212**
Bradybaena similaris, **459**
Brain pathology in, African trypanosomiasis, **142**, **143**
–amoebiasis, **498**, **662–664**
–angiostrongyliasis, **461**
–cysticercosis, **586**
–hydatidosis, **595**
–Japanese B encephalitis, **28**
–malaria, **87**, **88**
–meningitis, **702**
–paragonimiasis, **454**
–rabies, **624**
–schistosomiasis, **393**
–toxoplasmosis, **524–527**, **530**
Bromeliads, **60**
Brucella spp., 246
Brucellosis, **481**
Brugia malayi, **213**, **229**, **235**, **236**, **768**
Buffalo gnats, *see Simulium*
Bulinus spp., **362**, **404**, **405**
Burkitt's tumour, **736–739**
Buruli ulcer, **641**, **642**
Bushbuck, **132**

Calabar swelling, **257**
Calliphora, **694**
Cancrum oris, **645**
Capillaria hepatica, **546**
Capillaria philippensis, **547–550**
Capillariasis, **546–550**
Carcinomata, **389**, **390**, **425**, **597**, **740**
Carriòn's disease, **57**, **58**
Cats, 218, **399**, **422**, **521**, **523**, **533**, **570–573**, **624**
Cattle, **398**, **444**, **481**, **589**, **591**
Cercaria, **352**, **420**, **437**, **442**
Cercarial dermatitis, **365**
Cercocyst, **575**
Cerebral malaria, **85**, **87**, **88**
Cestodes, *see* Tapeworms XIII
Chagas' disease, **144–165**, **766**
–distribution, **144**

–diagnosis, **153**, **156**, **162–164**
–immunology, **163**, **164**
–epidemiology, **151**, **152**
–pathology, **154–161**
Chancre, **133**, **656**
Cheilomastix mesnili, **515**, **517**
Cheilosis, **723**
Chiclero's ulcer, **200**
Chinese liver fluke, **418–425**
Chlamydia, **618**, **622**
Chloroquine resistance, **93**
Cholera, **476–480**
Chromoblastomycosis, **673**, **674**
Chrysomyia, **692**
Chrysops spp., **256**, **769**
Chyluria, **233**
Cipangopaludina chinensis, **458**
Circadian rhythm, **247**
Circulatory system, schistosomiasis, **370–372**
Clonorchis sinensis, **307**, **391**, **415**, **420–424**, **432**
Clonorchiasis, **391**, **418–425**
Cobra, **746**, **750**
Coccidial infections, **518–520**, *see also* Toxoplasmosis
Coenurus cerebralis, **599**, **600**
Congo floor maggot, **691**
Cordylobia anthropophaga, **689**, **693**
Cor pulmonale, **379**
Coxiella burneti, **39**
Crab, **267**, **448–450**
Crawcraw, **272**
Crayfish, **448**
Creeping eruption, **257**, **321**, **324**
Cretin, **757**
Crotalinae, **747**
Cryptococcus neoformans, **679**
Ctenopharyngodon idellus, **419**
Culex, **2**, **5**, **8**, **10–13**, **216**, **217**, **221**, **222**, **224**
Culicoides spp., **250–253**, **288**, **290**
Cyclops spp., **554**, **557**, **565**, **568**
Cyprinus carpio, **429**
Cysticercoid, **572**, **573**, **575**, **576**, **578**
Cysticercosis, **581**, **584–586**, **589**
Cysticercus cellulosae, **585**
Cysts, protozoal, *see* Protozoa, Flagellates
Cystozoites, **530**

Dactylitis, **742**, **743**
DDT, **29**, **59**
Dehydration, **474**, **475**, **479**, **480**, **711**
Delhi sore, *see* Dermal leishmaniasis
Dengue, **22**, **23**
Dermal leishmaniasis, diagnosis, **191–195**, **197–201**
–distribution, **189**, **196**
–epidemiology, **190**, **202**, **203**
–immunology, **195**, **199**
–pathology, **210**
Dermatobia hominis, **695**, **696**
Dichuchwa, **658**
Dicrocoelium dendriticum, **434**
Dientamoeba fragilis, **491**
Diffuse cutaneous leishmaniasis, **210–212**
Diphyllobothriasis, **563–567**
Diphyllobothrium latum, **300**, **563–566**
Dipylidium caninum, **570–573**
Dirofilaria immitis, **248**, **249**
Dog, **177**, **311**, **321**, **399**, **451**, **553**, **570–573**, **591**, **597–599**, **624**
Dog sarcoma, **167**
Dog tapeworm, **570–573**, **591**
Donovanosis, **647–650**
Dracontiasis, diagnosis, **559–562**
–epidemiology, **556**, **558**
–pathology, **560**
Dracunculus medinensis, **557**, **561**, **562**
Dum-dum fever, *see* Visceral leishmaniasis

Dwarf tapeworm, **574–577**
Dysentery, *see* Amoebiasis, Enteritis, Gastroenteritis, Typhoid

Ebola fever, **733B, C**
Echinococcus granulosus, **591–596**
Echinococcus multilocularis, **597, 598**
Ectoparasitic arthropods,
Auchmeromyia luteola, **691**
–*Calliphora*, **694**
–*Chrysomyia*, **692**
–*Cordylobia anthropophaga*, **689, 693**
–*Dermatobia hominis*, **695, 696**
–*Lucilia*, **694**
–*Sarcoptes scabiei*, **683, 684**
–*Tunga penetrans*, **687**
Egyptian splenomegaly, **369**
Elapidae, **746**
Electronmicrographs, *Ascaris lumbricoides*, **330**
–*Aedes*, **220**
–*Borrelia recurrentis*, **55**
–*Coxiella burnetti*, **39**
–*Culex*, **221**
–Ebola fever virus, **733C**
–*Giardia lamblia*, **512**
–hepatitis viruses, **464, 465**
–hookworms, **313–317**
–Lassa fever virus, **732**
–*Leishmania*, **168**
–*Plasmodium*, **65, 115**
–poliomyelitis virus, **462**
–*Rickettsia prowazeki*, **30**
–smallpox virus, **616**
–*Trypanosoma gambiense*, **126**
–yellow fever virus, **21**
Elephantiasis, **229, 231, 278, 279**
Elephant skin, **273**
Endemic syphilis, **658**
Endodyogeny, **523**
Endomyocardial fibrosis, **754**
Endozoites, **523**
Entamoeba coli, **487, 492**
Entamoeba histolytica, **488, 493, 497, 507, 511**
Entamoeba nana, **489, 494**
Enteric fever, *see* Typhoid
Enteritis necroticans, **482, 483**
Enterobiasis, **541–543**
Enterobius vermicularis, **303, 309, 541, 544, 545**
Entropion, **620**
Eosinophilia, **257, 291, 292**
Eosinophilic lung, **291, 292**
Eschars, **35, 38**
Espundia, **196, 204–207, 209**
Exoerythrocytic schizonts, **67**
Eye pathology, avitaminosis A, **712–714**
–Chagas' disease, **153**
–*Coenurus*, **600**
–leprosy, **640**
–leptospirosis, **485**
–loaiasis, **258, 259**
–onchocerciasis, **271, 280–282, 286**
–pentastomiasis, **610**
–sparganosis, **569**
–toxocariasis, **337, 338**
–toxoplasmosis, **525, 528**
–trachoma, **619–621**
–trichinosis, **538**
–whooping cough, **707**

Faeces, helminth eggs in, **294–307**
–helminth larvae in, **322, 456**
–in amoebiasis, **496**
–in capillariasis, **549, 550**
Faeco-oral transmission, 234
Fascioliasis, **435–444**
Fasciola gigantica, **444**
Fasciola hepatica, **296, 408, 437–443**

Fasciolopsis buski, **408**, **410**, **411**, **413**
Fasciolopsiasis, **409**, **414**
Field's stain, **74–77**
Filariform larvae, **310**, **322**
Filariasis, *see Brugia malayi*
–Eosinophilic lung
–Loaiasis
–*Mansonella ozzardi*
–Onchocerciasis
–*Tipetalonema perstans*
–*Tipetalonema streptocerca*
–*Wuchereria bancrofti*
Fish, **415–417**, **418–421**, **427–429**, **553**, **563**, **564**, **566**
Fish tapeworm, *see* Diphyllobothriasis

Fleas, **42–48**, **572**
Flagellates, in genitalia, **659**, **660**
–in intestine, **512–517**
Flies, *see* Myiasis
Fogo selvagem, **760**
Foliaceus pemphigus, **760**
Folic acid, **725**, **726**
Framboesiform yaws, **652**
Frei test, **623**
Frog, **568**, **569**
Fungi, XIV, *see* Mycoses
Fungi imperfecti, **667**

Gametes, **63**
Gametocytes, **81**, **83**, **97**, **98**, **105**
Gangosa, **654**
Gangrene, **32**
Gastro-intestinal tract, amoebiasis, **496–500**
–anisakiasis, **551**, **552**
–ascariasis, **332–335**
–capillariasis, **546–550**
–Chagas' disease, **159–161**
–enterobiasis, **541**
–enteritis necroticans, **482**
–giardiasis, **517**
–hookworm, **312**
–hymenolepiasis, **575**
–schistosomiasis, **372–377**, **394**
–sprue, **761**, **762**
–strongyloidiasis, **326**
–trichuriasis, **343**, **344**
–typhoid, **473**
Gastroenteritis, non-specific, **474**, **475**
Genitalia, **510**, **648–651**, **656**, **661**
Giant cell pneumonia, **699**
Giardiasis, **517**
Giardia lamblia, **512**, **513**, **515**
Giemsa stain, **74–77**
Gingivitis, **728**
Gingivostomatitis, herpetic, **625**
Gland puncture, **137**, **138**
Glossina spp., **127–131**
Glossitis, **724**
Gnathostoma spinigerum, **553**, **554**
Gnathostomiasis, **553–555**
Goat, **481**
Goitre, **756**, **757**
Gonorrhoea, **651**
Granuloma inguinale, **647**, **648–650**
Guinea worm, *see* Dracontiasis

Haemagogus, **16**
Haematuria, **381**
Haemoglobinopathies, **92**, **741–745**
Hair-on-end, **745**
Halzoun syndrome, **602**
Hamster, **185**, **207**, **208**
Hanging groin, **277**, **278**
Heart pathology, Chagas' disease, **154**, **156–158**
–endomyocardial fibrosis, **754**
–schistosomiasis, **379**
Helminth eggs, differentiation, **293–307**, **547**, **571**

Helminthiases, epidemiology, 157, 158, VII, X, XIII
Hepatoma, **740**
Herpes simplex, **625**, **626**
Heterophyes heterophyes, **307**, **408**, **415–417**
Hexacanth embryos, **565**, **574**
Hippeutis sp., **410**
Histoplasma duboisii, **681**, **682**
Histoplasmosis, **681**
Hookworm, diagnosis, **301**, **309**, **319–321**, **328**
–distribution, **308**
–epidemiology, **310**, **311**
–identification, **313–317**
–life cycle, **310–312**, **771**
–pathology, **318–321**
–*see also Ankylostoma*, *Necator*
Hydatid sand, **594**
Hydatidosis, **591–598**
Hydrocoele, **228**
Hydronephrosis, **387**, **388**
Hymenolepis diminuta, **305**, **578**, **579**
Hymenolepis nana, **304**, **574–577**
Hyperkeratosis, **653**
Hyrax, **190**

Iatrogenic parasitoses, **734**, **735**
Immune response in, African trypanosomiasis, **141**, **143**
–amoebiasis, **511**
–Chagas' disease, **163**, **164**
–filariasis, **248**, **249**
–infective hepatitis, **469**
–leishmaniasis, **187**, **188**, **195**, **199**, **210**
–leprosy, **633**
–lymphogranuloma venereum, **623**
–malaria, **73**, **117**, **118–120**
–meningitis, **703**
–mycoses, **669**
–schistosomiasis, **395**, **396**
–smallpox, **617**
–toxocariasis, **340**
–toxoplasmosis, **531**
–trichinosis, **539**
Immunoglobulins, *see* Immune response
Infant mortality, **212**, **475**
Infantile kala-azar, **181**
Infective hepatitis, **464–469**
Insect, host of helminth, **576**, **579**
Iodamoeba bütschlii, **495**
Isospora belli, **520**

Japanese B encephalitis, **27**, **28**
Jaundice, **466**
Jigger flea, **687**, **688**

Kala-azar, **175–188**
–distribution, **175**
–diagnosis, **178**, **179**, **184–188**
–epidemiology, **176**, **177**
–immunology, **187**, **188**
–infantile, **181**
–pathology, **178**, **180**
–post k.a. dermal leishmanoid, **182**, **183**
Keratomalacia, **714**
Kerteszia, **60**
Kidney pathology, Burkitt's tumour, **739**
–malaria, **84**, **85**, **116**, **117**
–schistosomiasis, **381**, **386–388**
Kissing bugs, **147**
Koplik's spots, **698**
Kuru, **755**
Kwashiorkor, **708–710**

Larva migrans, **257**, **258**, **292**, **321**, **324**, **337–339**, **555**

Lassa fever, **729–732**
Latex agglutination test, **703**
Latrodectus mactans, **752**
Leishman-Donovan bodies, **154**, **166**
Leishman stain, **74–77**
Leishmania, aethiopica, **190**, **211**
–*braziliensis*, **207**, **208**
–*chagasi*, **175**
–classification, VII
–*donovani*, **175**, **186**, **188**
–*infantum*, **175**, **186**
–life cycle, **166**, **167**, **173**, **767**
–*mexicana*, **167**, **198**, **202**, **208**, **210**
–*minor*, **190**
–*panamensis*, **197**, **203**
–*tropica*, **190**, **191**
Leishmaniasis, *see* Dermal leishmaniasis
–diffuse cutaneous, **210–212**
–epidemiology, VII
–immunology, **187**, **188**, **210**
–mucocutaneous, *see* Espundia
–New World cutaneous, **196–203**, **208**
–Old World cutaneous, **189–195**
–post kala-azar dermal, **182**, **183**
–visceral, *see* Kala-azar
Leonine facies, **183**, **639**
Leopard skin, **275**
Lepromin test, **633**
Leprosy, diagnosis, **628–632**, **633–640**
–distribution, **627**
–epidemiology, 307
–immunology, **633**
–indeterminate, **634**
–lepromatous, **210**, **639**, **640**
–pathology, **631**, **632**
–tuberculoid, **635–638**
Leptospira spp., **485**, **486**
Leptospirosis, **485**, **486**
Leptotrombidium, **33**, **34**
Lice, **29**
Life cycles, **764–775**
–African trypanosomiasis, **765**
–ascariasis, **773**
–Chagas' disease, **766**
–filariasis, **768–770**
–hookworm, **771**
–leishmaniasis, **767**
–malaria, **764**
–schistosomiasis, **775**
–strongyloidiasis, **772**
–trichuriasis, **774**
Limax amoebae, **662–664**
Linguatula serrata, **601–603**
Liver flukes, **418–444**
Liver pathology in, amoebiasis, **498**, **502–507**
–ascariasis, **334**
–clonorchiasis, **422**, **424**, **425**
–fascioliasis, **437–439**
–hepatoma, **740**
–*Herpes simplex*, **626**
–hydatidosis, **592–594**, **597**, **598**
–infective hepatitis, **468**
–kala-azar, **178**
–Lassa fever, **729**
–leptospirosis, **485**
–malaria, **67**, **72**, **89**, **120**
–opisthorciasis, **433**
–pentastomiasis, **608**
–plague, **41**
–schistosomiasis, **366–368**, **370**
–toxocariasis, **339**
–yellow fever, **20**
Loaiasis, diagnosis, **257–259**
–distribution, **255**
–epidemiology, **256**
Loa loa, **237**, **238**, **254**, **260**, **769**
Lôboa lôboi, **675**, **676**
Lôbo's disease, **675**
Loxosceles laeta, **753**
Lucilia, **694**
Lumbar puncture, **140**

Lung, **291**, **311**, **378**, **379**, **448**, **451–453**, **456**, **484**, **498**, **508**, **596**, **601**, **607**, **678**, **704**
Lung flukes, **445–454**
Lungworm, **607**
Lutzomyia spp., **58**, **169–172**
Lymnaea spp., **407**, **436**, **440–442**
Lymphadenopathy, **50**, **136**, **137**, **227**, **232**, **277**, **278**, **529**, **622**, **674**, **680**, **705**
Lymphogranuloma venereum, **622**, **623**, **649**

Machado-Guerreiro reaction, **163**
Madura foot, **667**, **668**
Madurella mycetomae, **669**, **670**
Maduromycosis, **667–672**
Main de predicateur, **636**
Malaria, **59–120**, **764**
–*see also* Plasmodium
Malassezia furfur, **666**
Mansonella ozzardi, **239**, **290**
Mansonia, **214**, **218**, **219**, **223**
Maps of distribution, African trypanosomiasis, **123**
–Burkitt's tumour, **736**
–Chagas' disease, **144**
–cholera, **476**
–chloroquine resistance, **93**
–clonorchiasis, **418**
–dermal leishmaniasis, **189**, **196**
–espundia, **196**
–filariasis, **213**
–haemoglobin S, **92**
–haemoglobinopathies, **741**
–hookworm, **308**
–leprosy, **627**
–loaiasis, **255**
–malaria, **59**
–meningitis, **700**
–onchocerciasis, **261**
–opisthorciasis, **418**
–paragonimiasis, **445**
–plague, **40**
–schistosomiasis, **345**, **346**
–scrub typhus, **33**
–smallpox, **612**
–Southeast Asian haemorrhagic fever, **24**
–trachoma, **618**
–yellow fever virus, **1**
Marasmus, **475**, **708**, **711**
Marburg disease, **733A**
Marmoset, **203**
Marrow, **184**
Mastomys natalensis, **731**
Maurer's dots, **79**
Measles, **697–698**
Mega syndrome, **159**, **160**
Melioidosis, **484**
Meningitis, **679**, **700–703**
Meningococcal meningitis, **700–703**
Metacercaria, **411**, **420**, **421**, **428**, **450**
Metagonimus yokagawai, **415–417**
Microfilaria, IX
–counts, **246**
–differentiation, **234–245**, **254**, **271**, **285**, **288–290**
–life cycle, **214**, **215**
Milk, 242, 246
Miracidia, **347–350**, **440**
Mitsuda reaction, **633**
Molluscan intermediate hosts, XI
–of schistosomes, **361–364**
–of trematodes, **400–407**
Monkeys, **16**, **203**, **733A**
Montenegro test, **195**
Morula cell, **143**
Mossy foot, **667**, **668**
Mosquitoes, *Aedes*, **3**, **6**, **14**, **16**, **17**, **24**, **216**, **220**
–*Anopheles*, **4**, **9**, **15**, **216**
–*Culex*, **2**, **5**, **8**, **10–13**, **216**, **217**, **221**, **222**, **224**
–*Haemagogus*, **16**
–*Kerteszia*, **60**
–*Mansonia*, **214**, **218**, **219**, **223**

–*Stegomyia*, **16**
Mott cell, **143**
Mucocutaneous leishmaniasis, *see* Espundia
Multiceps multiceps, **599**, **600**
Murine typhus, 32
Muscles, **463**, **540**, **581**, **584**, **585**, **589**, **590**, **636**, **721**
Mycobacterium leprae, **631**, **632**
Mycobacterium ulcerans, **641–645**
Mycoses, XIV
–superficial, **665**, **666**
–systemic, **667–682**
Myiasis, **689–696**, XV
Myocardium, *see* Heart
Myoglobinuria, **748**

Naegleria spp., **662–664**
Naja naja, **746**
Nacator americanus, **301**, **308**, **309–312**, **316**, **318**, **771**, *see also* Hookworm
Negri bodies, **624**
Neiseria meningitidis, **702**
Nematodes, VIII, *see* Angiostrongyliasis, Anisakiasis, Capillariasis, Dracontiasis, Enterobiasis, Filariasis, Gnathostomiasis, Soil-mediated helminthiases, Trichinosis
Neoplasia, **389**, **390**, **425**, **597**, **736–740**
Nephrosis, **116**, **117**
Nerves, in beri beri, **721**
–in leprosy, **636–638**
–in poliomyelitis, **463**
–in snake bite, **749**
Nicotinic acid, **727**
Njovera, **658**
NNN medium, **186**, **207–209**
Nodules, bladder, **383**, **384**
–subcutaneous, **268–270**, **287**
Nutritional diseases, avitaminoses, **712–728**, *see also* Vitamins
–epidemiology, 352, 353
–kwashiorkor, **708–710**
–marasmus, **475**, **708**, **711**

Onchocerca volvulus, **241**, **242**, **261**, **285**, **286**, **289**, **770**
Onchocerciasis, diagnosis, **268–286**
–distribution, **261**
–epidemiology, **262**, **263**, **265–267**
–eye lesions, **271**, **280–282**, **286**
–pathology, **269–271**
–skin changes, **271–276**, **279**, **283–285**
Oncomelania spp., **363**, **400**
Oncosphere, **574**
Oocysts, **65**, **519**, **520**, **522**
Ookinetes, **64**
Opisthorciasis, **418**, **426–433**
Opisthorcis felineus, **307**, **408**, **418**, **430**
Opisthorcis viverrini, **418**, **427**, **431**
Optic atrophy, **282**
Oriental liver fluke, **418–425**
Oriental sore, *see* Dermal leishmaniasis
Ornithodorus moubata, **53**
Oroya fever, **57**
Osteitis, **655**
Osteomalacia, **719**

Panstrongylus spp., **147**
Paracoccidioides brasiliensis, **677**
Pasteurella pestis, **41**
Pathology, *see* individual diseases and tissues
Paragonimiasis, **445–454**
Paragonimus africanus, **445**, **453**
Paragonimus heterotremus, **445**, **447**

Paragonimus uterobilateralis, **445**, **453**
Paragonimus westermani, **299**, **408**, **445**, **446**, **448**, **452–454**
Pediculus humanus, **29**
Pellagra, **727**
Pentastomiasis, **601–611**
Periodic filariasis, **213**
Periportal fibrosis, **367**, **368**
Peyer's patches, **473**
Phlebotomus spp., **169**, **767**
Physopsis, **405**
Pian bois, **197**
Pig, **481–483**, **532**, **534**, **535**, **537**, **581**, **591**
Pigbel, **482**, **483**
Pinta, **657**
Pinworm, **541–543**
Pipestem fibrosis, **368**
Pirenella spp., **415**
Pit viper, **747**
Pityriasis versicolor, **666**
Placenta in malaria, **91**
Plague, **40–51**
Planarians, **457–459**
Plasmodium, falciparum, **78–83**
–life cycle, **63–69**, **78–81**, **764**
–*malariae*, **106–115**
–*ovale*, **102–105**
–rodent, **68**, **69**
–*vivax*, **94–100**
Plerocercoid, **566**
Pneumonia, **484**, **699**, **734**, **735**
Pneumococcus, **703**
Pneumocystis carinii, **734**, **735**
Poliomyelitis, **462**, **463**
Polyposis, of bladder, **383**, **384**
–of colon, **373–377**
Pork tapeworm, **580–586**
Porocephalus crotali, **604–607**
Post kala-azar dermal leishmanoid, **182**, **183**
Potamon rathbuni, **449**
Primary amoebic meningoencephalitis, **662–664**
Procercoid, **565**, **572**
Proechimys guyanensis, **202**
Proglottids, **564**, **577**, **587**, **588**
Protozoa, IV, V, VI, VII, XII
–*see Plasmodium*
–in CNS, **662–664**
–in faeces, **487–495**, **512–517**, **532**
–in genitalia, **659**, **660**
Ptosis in snakebite, **749**
Pus, **504**, **505**

Q fever, **39**

Rabbit, **601**
Rabies, **624**
Radio-isotope scan, **391**, **502**
Rashes, African trypanosomiasis, **133**, **134**
–bartonellosis, **58**
–dengue, **23**
–hookworm, **321**
–kwashiorkor, **709–710**
–leishmaniasis, **174**, **182**, **183**
–louse-borne typhus, **31**
–meningococcal meningitis, **701**
–pellagra, **727**
–scabies, **685**, **686**
–schistosomiasis, **365**, **371**
–scrub typhus, **35**, **36**
–smallpox, **613–615**
–Southeast Asian haemorrhagic fever, **25**, **26**
–tick typhus, **38**
–trepanomatoses, **657**, **658**
–typhoid, **471**
–*see also* Leprosy
–*see also* Onchocerciasis
Rat, **48**, **49**
Rat tapeworm, **578**, **579**

Redia, **441**
Reduviid bugs, **147**
Relapsing fever, **52–56**
Rhabditiform larvae, **310**, **322**
Rhodnius spp., **147**
Riboflavine, **722–724**
Rickettsia conori, **37**
–*prowazeki*, **29**, **30**
–*tsutsugamushi*, **33**
Rickets, **715–719**
Rickettsioses, III
–epidemiology, III
–louse-borne typhus, **28–31**
–murine typhus, 32
–Q fever, **39**
–scrub typhus, **33–36**
–tick typhus, **37**, **38**
Rickety rosary, **716**
Rodents, **48**, **49**, **68**, **69**, **124**, **125**, **177**, **180**, **202**, **354**, **399**, **455**, **456**, **460**, **574**, **606**, **607**, **731**
Romaña's sign, **153**
Romanowsky stains, **74–77**
Rose spots, **471**

Sabre tibia, **655**
Salmonellosis, **472**, **743**
Sandflies, **169–171**, **173**, **174**, **182**, **767**
Sarcocystis lindemanni, **518**
Sarcocystis suihominis, **519**
Sarcocystis hominis, **519**
Sarcoptes scabiei, **684**
Schistosoma haematobium, **293**, **355**, **358**, **362**, **775**
Schistosoma intercalatum, 180, **347**, **397**
Schistosoma mattheei, **398**
Schistosoma japonicum, **295**, **357**, **360**, **363**, **775**
Schistosoma mansoni, **294**, **347–354**, **356**, **359**, **361**, **366**, **408**, **775**
Schistosoma rodhaini, **399**
Schistosomiasis, ectopic, **365**, **378**, **379**, **393**
Schistosomiasis, intestinal, diagnosis, **369–375**, **379**, **392**
–distribution, **345**, **346**
–epidemiology, **364**
–immunology, **395**, **396**
–life cycle, **347–354**, **775**
–pathology, **366–368**, **376–378**, **393**, **394**
–with salmonellosis, **472**
Schistosomiasis, vesical, diagnosis, **381**, **383–388**
–distribution, **346**
–epidemiology, **380**
–pathology, **383**, **384**, **389**, **390**
Schistosomule, **353**
Schüffner's dots, **94–98**, **102–105**
Sclerosing keratitis, **281**
Scolex, **563**, **570**, **582**, **583**, **594**
Scotch tape swab, **542**, **543**
Scrotum, **228**, **231**, **278**
Scurvy, **728**
Sea snake, **748**
Segmentina, **406**, **410**
Semisulcospira spp., **416**, **448**
Sheep, **398**, **434**, **437–439**, **591**
Sickle cell disease, **742–744**
Simulium spp., **262–267**, **770**
Siti, **658**
Skerlievo, **658**
Skin, amoebiasis, **509**, **510**
–biopsy, **209**, **212**, **271**, **283–285**, **628–631**
–tests, **195**, **249**, **395**, **623**, **633**
Sleeping sickness, **139–143**
Sloth, **203**
Slugs, **457–459**
Smallpox, **612–617**
Snails, *see* Molluscs
Snakes, **604**, **605**, **607**, **609**, **746–751**
Snowflake opacities, **280**
South American blastomycosis, **677**, **678**

Southeast Asian haemorrhagic fever, **24–26**
Sowda, **279**
Sparganosis, **568**, **569**
Sparganum spp., **566**, **569**
Spider bites, **752**, **753**
Spirometra spp., **568**
Spleen pathology, kala-azar, **178**
–Lassa fever, **730**
–malaria, **72**, **90**, **118**, **119**
–schistosomiasis, **369**, **392**
Sporocysts, **351**
Sporotrichosis, **680**
Sporozoites, **66**
Sprue, **761**, **762**
Sputum, **448**
Stegomyia, **16**, **216**
Streptomyces pellietieri, **671**
Streptomyces somaliensis, **672**
Strongyloidiasis, **324–326**
Strongyloides stercoralis, **322**, **323**, **325**, **772**
Subperiodic filariasis, **213**
Syphilis, **656**

Taenia saginata, **306**, **583**, **587**, **589**
Taenia solium, **306**, **580–582**, **585**, **588**
Taeniasis, **581**, **584–586**, **590**
Tapeworms, XIII
–*Diphyllobothrium latum*, **300**, **563–566**
–*Dipylidium caninum*, **570–573**
–*Echinococcus granulosus*, **591–596**
–*Echinococcus multilocularis*, **597**, **598**
–*Hymenolepis diminuta*, **305**, **578**, **579**
–*Hymenolepis nana*, **304**, **574–577**
–*Multiceps multiceps*, **599**, **600**
–*Spirometra* spp., **568**
–*Taenia saginata*, **306**, **583**, **587**, **589**
–*Taenia solium*, **306**, **580–582**, **585**, **588**
Temperature charts, African trypanosomiasis, **135**
–brucellosis, **481**
–dengue, **22**
–kala-azar, **179**
–malaria, **70**, **86**
–relapsing fever, **54**
–typhoid, **470**
–yellow fever, **18**
Termite hills, **176**
Ternidens deminutus, **327**, **328**
Terranova spp., **551**, **552**
Tetanus, **646**, **688**
Tetrapetalonema perstans, **240**, **250**, **254**
Tetrapetalonema streptocerca, **243**, **244**, **288**
Thalassaemia, **741**, **744**, **745**
Thiamine, **720**, **721**
Thiara granifera, **401**, **448**
Tick, **37**, **53**
Tick typhus, **37**, **38**
Tinea imbricata, **665**, **711**
Tissue culture, **155**, **167**
Tissue paper skin, **276**
Tongue worm, **601–603**
Toxocara canis, **336**
Toxocara cati, **336**
Toxocariasis, **337–340**
Toxoplasma gondii, **521–523**, **530**, **531**
Toxoplasmosis, **524–531**
Trachoma, **618–621**
Tragelaphus scriptus, **132**
Trematodes, X, 179
–*see also* Intestinal flukes, Liver flukes, Paragonimiasis, Schistosomiasis
Treponema carateum, **657**
Treponema pallidum, **656**

Treponema pertenue, **652**
Treponematoses, *see* Endemic syphilis, Leptospirosis, Pinta, Syphilis, Yaws
Triatoma spp., **147**
Triatomid bugs, **147–151**, **162**
Tribolium spp., **579**
TRIC virus, **619**
Trichiasis, **620**, **621**
Trichinella spiralis, **309**, **534–536**
Trichinosis, **534–540**
Trichomonas vaginalis, **514**, **659**, **660**
Trichophyton concentricum, **665**
Trichuriasis, **343**, **344**
Trichuris trichiura, **302**, **309** **341–343**, **546**, **774**
Trombiculid mites, **33**, **34**, **683**
Tropical splenomegaly syndrome, **118–120**
Trypanosoma, VI
–*brucei*, **124**, **125**, **765**
–*cruzi*, **145–150**, **154**, **155**, **161**, **766**
–*gambiense*, **124–126**, **131**, **136**, **765**
–life cycles, **128**, **129**, **148–150**, **765**, **766**
–*rangeli*, **165**
–*rhodesiense*, **124**, **125**, **132**, **139**, **140**, **765**
Trypanosomiasis, African, *see* Sleeping sickness
–American, *see* Chagas' disease
–diagnosis, **133–141**, **153**, **156**, **162–164**
–distribution, **123**, **144**
–epidemiology, 14, **127**, **131**, **132**, **147**, **151**, **152**
–immunology, **141**, **143**, **163**, **164**
–pathology, **133**
Tsetse fly, **127**
Tuberculosis, **704–706**
Tumbu fly, **689**, **690**
Tunga penetrans, **687**
Tungiasis, **688**
Typhoid, **470–473**
Typhus, epidemic, **29**
–epidemiology, 14
–louse-borne, **29–32**
–scrub, **33–36**

Ulcers, *see also* Chancre, Dermal leishmaniasis, Espundia
–Buruli, **641**, **642**
–cancrum oris, **645**
–kwashiorkor, **709**
–*Mycobacterium ulcerans*, **641**, **642**
–mycoses, **667**, **672**, **673**, **680**
–phagedaenic, **643**
–tropical, **643**, **644**
Ureters, **386**, **387**
Urine, blackwater fever, **84**
–helminth eggs, **293**
–hepatitis, **467**
–microfilaria, **233**
–schistosomiasis, **381**
–sea snake bite, **748**
Uta, **196**

Vaccination, **617**
Vaginitis, **661**
Vahlkampfiidae, **662–664**
Vampire bat, **624**
Venereal diseases, **622**, **623**, **647**, **648**, **656**, **661**
Venomous animals, **746–753**
Verrucous dermatitis, **674**
Verruga Peruviana, **58**
Vervet monkey, **733A**
Vibrio cholerae, **476**, **477**
Vincent's organisms, **643**, **645**
Viperidae, **747**, **751**
Virus infections, dengue, **22**, **23**
–Ebola fever, **733B**, **C**
–hepatitis, **464–469**
–Herpex simplex, **625**, **626**

–Japanese B encephalitis, **27**, **28**
–kuru, **755**
–Lassa fever, **729–732**
–lymphogranuloma venereum, **622**, **623**
–Marburg disease, **733A**
–measles, **697–699**
–poliomyelitis, **462**, **463**
–rabies, **624**
–smallpox, **612–617**
–Southeast Asian haemorrhagic fever, **24–26**
–trachoma, **618–621**
–yellow fever, **1**, **18–21**
–*see also* II
Visceral larva migrans, *see* Larva migrans
Visceral leishmaniasis, **175–188**
Vitamins, A, **712–714**
–B1, **720**, **721**
–B2, **722–724**
–C, **728**
–D, **715–719**
–folic acid, **725**, **726**
–PP, **727**
Vitiligo, **666**
Vomitus, **19**

Water caltrop, **410**, **414**
Water chestnut, **410**
Water fleas, **554**, **556**, **557**, **565**, **568**
Whitmore's bacillus, **484**
Whooping cough, **707**
Winterbottom's sign, **136**
Wuchereria bancrofti, diagnosis, **227–234**, **245**, **246**, **248**, **290**
–distribution, **213**
–epidemiology, **216**, **224**
–immunology, **248**, **249**
–life cycle, **214**, **215**, **225**, **226**, **247**, **768**
–pathology, **227–233**

Xenodiagnosis, **162**
Xenopsylla astia, **44**, **45**
–*brasiliense*, **46**, **47**
–*cheopis*, **42**, **43**, **48**
Xerophthalmia, **712**
X-rays, amoebic liver abscess, **503**
–ascariasis, **332**
–Chagas' disease, **159**
–cysticercosis, **584**
–dracontiasis, **559**
–filariasis, **232**
–hydatid cyst, **592**, **596**
–Madura foot, **668**
–melioidosis, **484**
–opisthorciasis, **433**
–paragonimiasis, **453**
–pentastomiasis, **609**
–pneumonic plague, **51**
–rickets, **717**, **719**
–schistosomiasis, **372**, **373**, **379**, **383**, **385–387**
–sickle cell disease, **743**
–South American blastomycosis, **678**
–sprue, **761**, **762**
–thalassaemia, **745**
–tuberculosis, **704**
–yaws ostetis, **665**

Yaws, **652–655**
Yellow fever, **1**, **18–21**
Yersinia pestis, **41**

Ziehl-Niellsen stain, **630**
Ziemann's dots, **106–110**
Zoonoses, 204, **121**, **122**, **132**, **152**, **177**, **189**, **190**, **202**, **203**, **729–733**