CHAPTER 2

COMMUNICABLE DISEASES

Section I - Parasitic

2-1. GENERAL. Of all the diseases that afflict mankind, many parasites, especially malaria, cause the highest morbidity and mortality worldwide.

2-2. AMEBIASIS. Caused by the one-celled parasite Entamoeba histolytica. It is present throughout the world, but is especially severe in third world countries and in tropical countries. Diarrhea is the most common presentation.

S. Recurrent bouts of diarrhea and abdominal cramps, sometimes alternating with constipation.

O. Tenderness and enlargement of the liver are frequent. Semifluid stools containing no pus and only flecks of blood-stained mucus. Stools 5-10 per day often with fever up to 105° F. Abdominal colic and vomiting. Lab findings: Entamoeba histolytica trophozoites and cysts in stool specimens are difficult to detect. Even with the best lab techniques a minimum of six separate stool specimens are needed to diagnose the disease. Trophozoites are found in liquid stools; cysts are found in formed stools.

A. Amebiasis. Differential diagnosis: Other causes of diarrhea, bacillary dysentery, emotional diarrhea, diarrhea 2° to laxative abuse, diverticulitis, drugs, pernicious anemia.

P. Collect six stool samples to look for trophozoites and cysts. Trophozoites that contain ingested red blood cells are diagnostic for invasive Entamoeba histolytica. Leukocytes and macrophages are relatively rare in the stool sample; whereas in bacillary dysentery many white blood cells are present.

Treatment: Metronidazole (Flagyl) 750 mg. t.i.d. x 10 days followed by diiodohydroxyquin 650 mg. q.i.d. x 21 days.

Follow-up care: The stool should be examined six times over one week after symptoms have disappeared. If any cysts or trophozoites are found in these specimens, initiate the treatment above until symptoms are cleared.

2-3. MALARIA. Malaria is perhaps the most debilitating illness worldwide, especially in the tropics. Four species of Plasmodium are responsible: Plasmodium vivax, falciparum, malariae, and ovale.

S. Acute episodes of chills, fever, and sweating. Occasionally delirium, coma, convulsions, gastrointestinal disorders, and jaundice. The chills last from 15 minutes to an hour; nausea, vomiting, and severe headache are common at this time. Fever that follows the chills will last several hours and will often get to 104° F. or higher. The third stage, or sweating, concludes the cycle. The fever subsides and the patient falls asleep to awaken feeling fairly well. In vivax, ovale, and falciparum infections, the episodes occur every 48 hours (tertian malaria). In malariae infections (quartan malaria) the cycle takes 72 hours.

O. The thick and thin blood film, stained with Giemsa's stain or Romanovsky stain, is the mainstay of malaria diagnosis. The thin film is used primarily for species differentiation after the presence of an infection is detected on a thick film. The level of parasites in the blood varies from hour to hour; therefore the blood should be examined several times a day for 2-3 days. Anemia may be present and is usually more severe with falciparum infections. Jaundice may develop in severe infections.

A. Malaria. Differential diagnosis: Other causes of fever in tropics, urinary tract infections, typhoid fever, infectious hepatitis, dengue, leptospirosis. Examination of the blood film is essential to differentiate the above from malaria.

P. Chloroquine is used to prophylatically suppress symptoms of malaria, but it does not prevent infection. If falciparum malaria does not respond promptly to chloroquine (within 24 hours), parasite resistance to this drug must be considered.

Give chloroquine phosphate, 1 gram as initial dose, 500 mg. in 6 hours, and 500 mg. daily for the next 2 days. If patients cannot absorb the drug rapidly because of vomiting or severe diarrhea, or if they are comatose, give 250 mg. (salt) of chloroquine hydrochloride intramuscularly. Repeat in 6 hours, if necessary, and follow with oral therapy as soon as possible. Do not use chloroquine for severely ill patients whose infections originated in an endemic region for P. falciparum.

Prophylactic (suppressive) dosage: Before leaving home, the patient should take a test dose of the medication to detect possible allergic readings. Starting about 1 week before arrival in the area of malaria risk, the patient should begin chloroquine phosphate 500 mg. (salt) weekly, or the combined tablet of chloroquine 500 mg. (salt) plus primaquine phosphate 78.9 mg. (salt) weekly. After leaving the endemic area, the chloroquine should be continued for 6 weeks or the combined tablet for 8 weeks. For those taking chloroquine dose, a 14-day course of primaquine should be given if there has been significant exposure to P. vivax or P. ovale.

Primaquine phosphate: This drug has been shown to be the most effective agent against the tissue forms of P. vivax and P. ovale. The dosage for primaquine phosphate is 26.3 mg. daily for 14 days.

Treatment of malaria due to P. falciparum strains resistant to chloroquine.

When the patient can take medication orally, give quinine sulfate 650 mg. 3 times daily for 14 days plus pyrimethamine 25 mg. twice daily for 3 days, plus either sulfadiazine 500 mg. 4 times daily for 5 days, or dapsone 25 mg. daily for 28 days.

For prophylaxis, Fansidar for nonimmune individuals (pyrimethamine, 25 mg. and sulfadoxine, 500 mg.) should be given once weekly. The medication should be continued for 6 weeks after leaving the endemic area. Although Fansidar is not available in the USA, it is usually available in countries with chloroquine-resistant malaria under the trade names of Fansidar, Falcidar, or Antemal.

2-4. AFRICAN TRYPANOSOMIASIS (sleeping sickness). Rhodesian and Gambian trypanosomiasis are caused by two morphologically similar parasites

Trypanosoma rhodesiense and Trypanosoma gambiense. Trypanosomiasis occurs throughout tropical Africa from south of the Sahara to about 20 degrees South latitude. Trypanosoma gambiense is limited to West Africa up to the western Rift Valley. Trypanosoma rhodesiense occurs to the east of the Rift Valley. Both trypanosomes are transmitted by the bites of tsetse flies.

S. The patient may complain of a local inflammatory reaction (called a trypanosoma chancre). It occurs within 48 hours after a bite. The lesions may be painful or pruritic for up to 3 weeks. The patient may have personality changes, headache, apathy, somnolence, and tremors. The patient may become severely emaciated and finally become comatose.

O. Irregular fever, tachycardia, painless lymph nodes. Multiple thick wet blood smears should be taken. Other lab findings include anemia and increased sedimentation rate.

A. Trypanosomiasis. Differential diagnosis: May be mistaken for a variety of other diseases including malaria, tuberculosis, kala-azar, and cerebral syphilis.

P. Pentamidine is the drug of choice for prophylaxis of sleeping sickness, but is effective with certainty only against the Gambian type. In Rhodesian infection, pentamidine may lead to suppression of early symptoms resulting in recognition of the disease too late in its course for effective treatment. One intramuscular injection (4 mg./kilogram, maximum 300 mg.) will protect against Gambiense infection for 6 months. The drug is potentially toxic and should be used for persons at high risk. It must be emphasized that the drugs used to treat trypanosomiasis are available only from the Parasitic Disease Drug Service, Center for Disease Control, Atlanta, GA 30333, (404) 329-3670.

Suramin sodium is the drug of choice for treatment of the early stages of trypanosomiasis. Treatment is 1 gm dosages @ 1, 3, and 7 days and then weekly until a total of 7 grams have been given.

Tryparsamide has been used for a long time for Gambiense infections of the central nervous system. It is given intravenously in a 20% solution in water. The dosage is 20-40 mg./kg. (maximum dose 2 gm) given at weekly intervals for a total of 10-12 injections.

General measures: Good nursing care and treatment of anemia, concurrent infections, and malnutrition are essential in the management.

Prognosis: If untreated, most cases of African trypanosomiasis are fatal. If treated properly, the prognosis is excellent.

2-5. AMERICAN TRYPANOSOMIASIS (Chagas' disease). Chagas' disease is caused by Trypanosoma cruzi, a one-celled parasite of the blood and tissues of humans and other animals. T. cruzi is found in wild animals from southern South America to northern Mexico, Texas, and the southwestern USA. Many species of reduviid bugs (cone-nosed or kissing bugs) transmit the infection, which results from rubbing infected bug feces, passed during feeding, into the wound.

S. Intermittent fever, swollen painful lymph nodes, and occasionally convulsions.

O. Hard, edematous, red, and painful cutaneous nodules (chagoma). Unilateral palpebral and facial edema and conjunctivitis.

A. Chagas' disease. Differential diagnosis: Can be confused with kala-azar. The chagoma may be mistaken for a variety of tropical skin diseases.

P. Establish the diagnosis by taking thick and thin blood films and finding the parasite in the smears. Trypanosomes should be looked for in the blood of all patients but will usually be seen only in the acute stage of infection. Treatment of Chagas' disease is symptomatic and supportive. The best plan of action is preventive: Living quarters should be cleaned and pesticides used to eradicate the insects that transmit the disease.

2-6. LEISHMANIASIS. The clinical manifestations of leishmaniasis may be classified as (1) visceral, (2) cutaneous, and (3) mucocutaneous. These distinctions are not rigid because in the course of illness one type may develop into another. The leishmaniases are caused by different species of leishmania transmitted by the bites of sandflies (Phlebotomus).

a. Visceral leishmaniasis (kala-azar). Visceral leishmaniasis is geographically widespread. It is caused mainly by two species: Leishmania donovani in the Indian region and Leishmania infantum in USSR, China, Middle East, Mediterranean basin, and Africa. It also occurs in South America.

S. Irregular fever, insidious and chronic; onset may be acute.

O. Progressive anemia, loss of weight, progressive darkening of the skin especially the forehead and hands, gradual enlargement of the spleen and liver. The fever may be very high and the patient sometimes does not look very ill. There is a marked decrease in the W.B.C., usually less than 3,000/ml. The diagnosis is established by demonstrating Leishman-Donovan bodies in stained blood smears.

A. Leishmaniasis. Differential diagnosis: malaria.

P. Treatment of visceral leishmaniasis is difficult--the best drugs are not available for general use. The drug that is available is highly toxic, but it should be used if necessary. Amphotericin B at a dose of 0.5 mg./kg. per day is dissolved in 500 ml. of 5% dextrose and given over 6 hours on alternate days. Patients must be closely monitored. Without treatment, kala-azar is usually fatal.

b. Cutaneous leishmaniasis. Cutaneous leishmaniasis may present as self-healing ulcers (oriental sore), non-ulcerating nodules that resemble leprosy, and chronic mutilating ulcers. Cutaneous leishmaniasis is seen in the USSR, India, the Middle East, the Mediterranean basin, Africa, and Central and South America.

S and O. Cutaneous swellings appear about 2-8 weeks after bites of sandflies. The swellings may ulcerate and discharge pus, or they may remain dry. Dry and moist sores are caused by distinct leishmanias, with the dry forms having longer incubation periods.

A. Cutaneous leishmaniasis. Differential diagnosis: Syphilis, other forms of skin disease.

P. Metronidazole in the dosage required to treat amebiasis has proven effective.

c. Mucocutaneous (naso-oral) leishmaniasis. Naso-oral lesions caused by leishmaniasis are seen in South America. There it is referred to as espundia. The anterior cartilage of the nose is involved and sometimes leads to a complete erosion of the bone with disfigurement. Amphotericin B 0.25-1 mg./kg. every other day for up to 8 weeks is required to kill the organism.

2-7. SCHISTOSOMIASIS (bilharziasis). A blood fluke (trematode) infection with adult male and female worms living in veins of the host. Symptoms are related to the location of the parasite in the human host. Schistosoma mansoni and Schistosoma japonicum give rise to intestinal symptoms. Schistosoma haematobium gives rise to urinary tract symptoms.

S. Transient red itching skin rash with fever, malaise. The patient may have diarrhea, abdominal pain, loss of appetite, loss of weight. Urinary frequency, urethral and bladder pain.

O. Diarrhea and abdominal pain are common in the early stages of the disease. Diagnosis depends on finding the eggs in stool specimens. As many as 8-10 stool specimens are needed to detect the eggs.

A. Schistosomiasis should be considered in all unresponsive gastrointestinal disorders in endemic areas. Differential diagnosis: Early schistosomiasis may be confused with amebiasis or bacterial dysentery.

P. Treatment should be given only if live ova are identified. In the USA, the first drug of choice for S. haematobium and S. mansoni infections is niridazole. Outside the USA, in countries where it is available, the drug of choice is oxamniquine for S. mansoni and metrifonate for S. haematobium. Niridazole should be administered in high doses, under close medical supervision. Oral doses are 25 mg./kg. (maximum 1.5 grams) daily in 2 divided doses for 7-10 days. The side effects of the drugs include nausea, vomiting, headache, and brownish discoloration of the urine.

2-8. FASCIOLOPSIASIS. Fasciolopsis buski is a large intestinal fluke found in China, Taiwan, Southeast Asia, and India. The intermediate host is a snail. Humans are infected by eating uncooked water plants that have the parasite encysted in them. After an incubation period of several months in humans, manifestations of gastrointestinal irritation appear in all but light infections. In severe infections:

S. and O. Cramping epigastric and hypogastric pains, diarrhea, intermittent constipation, anorexia, and nausea. Edema, particularly of the face and ascites (accumulation of fluid in the abdominal cavity) may occur later. Death may result from the parasite or secondary infection.

Lab findings: Leukocytosis with moderate eosinophilia. Diagnosis is made by finding the eggs or occasionally flukes in the stools.

A. Fasciolopsiasis. Differential diagnosis: Other intestinal flukes.

P. Crystalline hexylresorcinol is the drug of choice. Adults 1

gm orally on an empty stomach in the morning. Repeat in 3-4 days. Children 0.1 gm/year of age to age 10. Same as with adult. After 2 hours give sodium sulfate or sodium citrate as a purgation to flush the intestinal tract. Two treatments are usually sufficient. Alternate drug piperazine citrate in recommended course of therapy.

2-9. LIVER FLUKES.

a. Fascioliasis. Sheep liver fluke found primarily in Latin America and the Mediterranean area. Man is infested by ingesting the metacercariae on watercrest or other aquatic vegetables.

b. Clonorchiasis. Endemic in areas of Japan, Korea, China, Formosa, and Indochina. Imported cases are seen in USA. Man is infested by eating raw or undercooked freshwater fish.

S. and O. Light infestations may be asymptomatic. Heavy infestations may present as malaise, fever, liver tenderness, and jaundice. These symptoms are transient. Progressive liver enlargement, right upper quadrant pain, and vague abdominal symptoms such as diarrhea, weakness, weight loss, tachycardia, and a variety of other symptoms may occur.

Lab findings: Leukocytosis with eosinophilia sometimes from 10-40%. Diagnosis is made by finding the eggs in the stool.

A. Fascioliasis or clonorchiasis.

P. Bithional 40 mg./kg. P.O. on alternate days over 20-30 days. Alternate drug: Emetine HCl, 1 mg./kg. IM up to 65 mg. daily for 7 days. Recovery is slow even if all the flukes are killed.

2-10. PARAGONIMIASIS. A lung fluke found throughout the Far East, West Africa, South Asia, central and northern South America. Man is infected by eating infected snails, crabs, and crayfish. Ingested immature flukes migrate through the small intestines usually to the lungs, although they can lodge in other tissues of the body or even migrate to the brain or spinal cord, but these usually fail to mature. The flukes that reach the lungs encapsulate, reach maturity, and lay eggs. These capsules swell and usually rupture into a bronchiole.

S. and O. The infection is usually asymptomatic until the flukes mature and begin laying eggs. The onset is insidious with low-grade fever and a cough that is dry at first, then turning to a viscous sputum that is rusty or blood-flecked. Pleuritic chest pain is common. The condition is chronic and progressive with dyspnea, signs of bronchitis and bronchiectasis, weakness, malaise, and weight loss. In heavy infestations, parasites in the abdomen may cause abdominal pain, diarrhea, or dysentery. Parasites in the brain or spinal cord, depending on their location, may cause seizures, palsies, or meningoencephalitis.

Lab findings: Slight leukocytosis with eosinophilia. Eggs can be readily found in the sputum if it is spun down and a smear is made from the bottom of the tube. Eggs can also be found in stool specimens.

A. Paragonimiasis.

P. Drug of choice is bithionol 40 mg./kg. of body weight given on alternate days for 10-15 doses (20-30 days).

2-11. TAPEWORM INFECTIONS. A number of tapeworms can infect humans, but only six are commonly found. Distribution is worldwide. Infestations usually occurs by eating infected and undercooked or raw beef, pork, fresh water fish, and crustaceans. Tapeworms vary in size from 1 cm. or less to 300 cm. or more.

S. and O. Adult tapeworms in human intestines usually cause no symptoms. Heavy infestations may present as weight loss, vague abdominal complaints, diarrhea, anorexia, abdominal pain, and nervous disturbances, particularly in children.

The larva of some tapeworms migrate throughout the body. In muscle or connective tissue they cause no problems, but in the brain they may cause a wide variety of manifestations: epileptic seizures, mental deterioration, personality disturbances, and internal hydrocephalus.

Lab findings: Segments of the tapeworm may be found in stool, clothing, or bedding. The ova often can be found using the scotch tape method (as used to diagnose pinworms). The eggs (ova) are found occasionally in the stool.

A. Tapeworm.

P. Drug of choice: Niclosamide. Give 2 gm orally in the morning before eating for 5 days. If niclosamide is not available, use quinacrine HCl (mepacrine). Place patient on liquid diet 24 hours prior to treatment (no milk). The evening before treatment, give saline or soapsuds enema. On morning of treatment, withhold breakfast and confine patient to bed. Give an antiemetic (Compazine) and wait 1 hour. For children 18-34 kg. give 0.5 gm; for adults or children over 45 kg. give 0.8 gm. Dose may be divided but must be given within 30 minutes. Wait 2 hours after the 30 minutes, then give saline or soapsuds purge.

2-12. TRICHINOSIS. Worldwide distribution, but it is a greater problem in the temperate areas than in the tropics. Infection occurs from eating raw or undercooked pork, but bear and walrus meat has also been implicated. Symptoms may appear in a few hours, but usual incubation period is 5-15 days.

S. and O. Symptoms vary considerably depending on the number of larva and the tissue invaded. Initial symptoms occur when mature female roundworms burrow into the small intestinal mucosa and may persist until the adults die at about 5 weeks. Diarrhea, abdominal cramps, malaise, nausea, vomiting, and occasionally constipation. The larva migrate through the bloodstream to most tissues of the body beginning at the end of the first week. This brings fever, low-grade to marked; muscle pain, especially on movement; muscle tenderness; edema; spasms; periorbital and facial edema; sweating; headaches; photophobia; weakness or exhaustion; pain on swallowing; dyspnea; coughing; hoarseness; conjunctival, retinal and nail hemorrhages; and rashes. Inflammatory reactions may produce meningitis, encephalitis, myocarditis, pneumonitis, nephritis, and peripheral and cranial nerve disorders. Death can occur in 4-6 weeks.

Lab findings: Eosinophilia 20-75% in the third or fourth week, slowly declining to normal. Adult worms are rarely found in the feces. Larva may occasionally be found in the blood in the second week. Definitive diagnosis is possible by biopsy of skeletal muscle in the third or fourth week.

A. Trichinosis.

P. Symptomatic treatment is normally all that is required. If it is known a patient has eaten infected meat within the last few days (not over 1 week), give thiabendazole 25 mg./kg. (maximum of 1.5 gm) b.i.d. after meals for 2-4 days. Severe infections, when the larva invade muscle tissue, require hospitalization and high doses of corticosteroids for 24-48 hours followed by lower doses for several days or weeks to control symptoms.

2-13. TRICHURIASIS (whipworms). Small slender worms, 30-50 mm. in length, found worldwide, particularly in the subtropics and tropics.

S. and O. Light to moderate infections rarely cause symptoms. Severe infections (10,000 or more ova per gram of feces) may present with a variety of symptoms that include abdominal pain, tenesmus (spasmodic contraction of anal sphincter with pain and persistent, involuntary, ineffectual straining effort to empty the bowel), diarrhea, distention, flatulence, nausea, vomiting, and weight loss. Blood loss may be significant and rectal prolapse may occur.

Lab findings: Characteristic barrel-shaped eggs in the stool. Eosinophilia of 5-20% in all but light infections and hypochromic anemia may be present in heavy infections.

A. Trichuriasis.

P. Mebendazole, 100 mg. b.i.d. before or after meals x 3 days. Tablets should be chewed before swallowing. No alcohol 24 hours before and after treatment. Alternate treatment soapsuds enema followed by hexylresorcinol enema (20-30 ml./kg. up to 1,200 ml.). Enema should be retained for 30 minutes before expulsion.

2-14. ASCARIASIS. The most common intestinal worm. Worldwide distribution. Infection is caused by ingestion of mature eggs in fecally contaminated food and drink. Eggs hatch and the larva penetrate the walls of the small intestines and migrate to the lungs. Adult worms are 20-40 cm. long.

S. and O. Fever, cough, hemoptysis (spitting or coughing up blood), rales, and other evidence of lung involvement. Rarely, the larva may go astray lodging in the brain, kidney, eye, spinal cord, or skin.

Heavy infections may also cause vague abdominal complaints and colic. With heavy infestations, especially if the worms are stimulated by certain oral medications or anesthetics, wandering may occur. Worms may be coughed up, vomited, or passed out through the nose. They may also cause mechanical blockage and inflammation by forcing themselves into the common bile duct, the pancreatic duct, the appendix, diverticula, and other sites.

Lab findings: Eggs in the stool; larva may occasionally be found in the sputum. CBC reveals eosinophilia.

A. Ascariasis lumbricoides. Differential diagnosis: Allergic disorders, other causes of pneumonitis, appendicitis, diverticulitis, etc.

P. Piperazine. Each ml. of syrup contains 100 mg. of piperazine hexahydrate, tablets contain 250-500 mg.

up to 14 kg. give 1 gm	once a day x 2 days
14-22 kg. give 2 gm	Heavy infections may
22-45 kg. give 3 gm	require 3 to 4 days
over 45 kg. give 3.5 gm	of treatment.

Alternate drugs are Pyrantel pamoate, mebendazole, levamisole, and bephenium hydroxynaphthoate.

2-15. STRONGYLOIDIASIS. Common in tropical and subtropical areas worldwide. Essentially an infection of humans but dogs may become infected. Larvae that are passed in the feces can remain alive for several weeks in certain soil conditions. They infect man by penetrating the skin and entering the bloodstream, and are carried to the lungs. They leave the bloodstream and ascend the bronchial tree. The larvae are then swallowed and are carried to the small intestines where they mature and lay eggs.

S. and O. Many cases are asymptomatic. Sensitized patients may develop linear, erythematous, or urticarial wheals that may be intensely pruritic or even hemorrhagic following entry of the larvae into the skin. During the migratory phase, vague symptoms develop including malaise, anorexia, fever, asthma, recurrent cough, and urticaria. Frequent gastrointestinal symptoms follow; diarrhea (may alternate with periods of normal bowel movement or constipation), nausea, vomiting, and diffuse colicky pain. In children there may be abdominal distention and persistent diarrhea accompanied by malabsorption syndrome plus weight loss and debilitation.

Lab findings: Eosinophilia normal to 50%, W.B.C. up to 20,000, and larvae or adult worms in the stools (allow the stool to stand 24-48 hours before examining).

A. Strongyloidiasis. Differential diagnosis: Epigastric pain may mimic peptic ulcer syndrome but with less relationship to meals. Can cause pneumonia. Skin invasion can resemble hookworm.

P. Drug of choice: Thiabendazole 25 mg./kg. (maximum 1.5 gm) b.i.d. x 2-3 days orally after meals.

Alternate drugs: Mebendazole, pyrantel pamoate, or levamisole.

2-16. ENTEROBIASIS (pinworms). Humans are the only host of this parasite. It occurs worldwide. Humans become infected by contaminated food, drink, or hands.

S. and O. Many patients are asymptomatic. Symptoms include pruritis of the perianal area, insomnia, restlessness, involuntary urination, and irritability, particularly in children. Mild gastrointestinal symptoms are also possible such as abdominal pain, nausea, vomiting, diarrhea, and anorexia.

Lab findings: W.B.C. normal except for modest eosinophilia (4-12%). To find eggs, apply scotch tape to the perianal skin and spread the tape over a slide for examination. This should be done on three consecutive days before the patient bathes or defecates. Adult worms should be looked for in the stool.

A. Pinworms.

P. Symptomatic patients should be treated and concurrent treatment of all household members should be considered. All bedding should be washed and personal hygiene should be stressed, e.g., careful washing of hands with soap and water after defecation and before meals, trim fingernails, avoid scratching rectal area, and keep hands away from the mouth. Eggs in a moist environment remain infective for 2-3 weeks, so it is best to repeat the medication every 2 weeks for 3 doses. Drug of choice is pyrantel pamoate 10 mg./kg. (maximum of 1 gm) in a single dose before or after meals. Repeat in 2 weeks. Alternates: Pyrvinium pamoate, mebendazole and piperazine citrate. Piperazine is last choice because the course of treatment requires 1 week.

2-17. HOOKWORM. Widespread in the tropics and subtropics. Infection of humans is through the skin in the same path as strongyloidiasis with the exception that hookworm eggs do not hatch in humans; they are passed in the stool.

S. and O. The first signs of hookworm infection is a pruritic erythematous dermatitis, either maculopapular or vesicular (ground itch) where the larvae invade the skin (allergic reactions to the invasion can occur and may be severe). Pulmonary signs are cough and bloody sputum. Two weeks or more after the skin invasion, abdominal symptoms including abdominal discomfort, flatulence, and diarrhea develop.

Lab findings: Eosinophilia present in the first few months of infection. Stool usually contains blood. (Guaiac test.) Anemia may be present depending on the number of worms. Eggs can be found in the stool; 4-5 ova per low power microscope field relates to about 5,000 eggs per gram of unconcentrated stool.

A. Hookworm.

P. Light infections in asymptomatic patients do not require treatment (up to 2,000 ova per gram of stool). Drug choice: Pyrantel pamoate 10 mg./kg./d. x 3 days orally in single dose, before or after meals.

Alternate drugs: Mebendazole 100 mg. b.i.d. x 3 days (do not use in pregnancy), bephenium hydrorynaphthoate 5 gm b.i.d. x 3 days on an empty stomach and withhold food for 2 hours; repeat in 1 week (for children less than 22 kg., cut dose in half).

2-18. FILARIASIS. Caused by one of two filarial nematodes that are transmitted by the bite of certain mosquitos. Widely distributed in the tropics and subtropics of both hemispheres and on Pacific islands. Over months the adult worms mature in or near the lymphatics or lymph nodes.

S. and O. Early manifestations are inflammatory with episodes of fever with or without inflammation of lymphatics and nodes, occurring at irregular intervals. Funiculitis (inflammation of the spermatic cord) and orchitis are common. Persistent lymph node enlargement may occur and abscesses may form at these sites. Later stages are obstructive and may not appear for months or years. Obstructive manifestations include hydrocele (accumulation of serous fluids in a saclike cavity), scrotal lymphedema, lymphatic varices, and elephantiasis. Elephantiasis may involve legs, genitalia, and less often arms and breasts.

Lab findings: Eosinophilia (10-30% or higher) in the early

stages. The count falls as the obstructive phase develops. Motile (mobile) larvae (microfilariae) are rare in the blood in the first 2-3 years, abundant after that and rare again in the advanced obstructive stage. Microfilariae should be microscopically looked for using wet thick smears of fresh anticoagulated blood.

A. Filariasis.

P. General measures: Bed rest during febrile and local inflammatory episodes. Antibiotic therapy to treat secondary infections. Suspension bandages for orchitis, epididymitis, and scrotal lymphedema. Treat mild limb edema with bed rest, elastic bandage wrap, and elevation of the affected part.

Surgical measures: Surgical removal of elephantoid scrotum, vulva, or breast should be considered. It is relatively easy and the results are usually satisfactory. Surgery for elephantiasis of a limb should be avoided. The surgery is difficult and results are poor.

Drug of choice: Diethylcarbamazine 2 mg./kg. orally after meals t.i.d. x 21-28 days. Headache, malaise, nausea, and vomiting may occur from the medication. Concurrent administration of an antihistamine and antiemetic may reduce the likelihood and intensity of allergic reactions.

Relapses may occur 3-12 months after treatment requiring several courses of treatment over 1-2 years.

Section II - Mycotic (Fungal)

2-19. COCCIDIOIDOMYCOSIS. Infection results from inhalation of arthrospores of Coccidioides immitis, a mold that grows in soil in arid regions of Southwest United States, Mexico, Central and South America. About 60 percent of infections are subclinical and unrecognized; incubation period 10-30 days.

S. Forty percent of patients develop mild to severe and prostrating symptoms that resemble those due to viral, bacterial, or other mycotic infections. Onset is usually that of a respiratory infection with fever and occasional chills, pleural pain (usually severe), muscular ache, backache, and headache (may be severe). Nasopharyngitis may be followed by bronchitis accompanied by a dry or slightly productive cough. Weakness and anorexia may become marked, leading to prostration. Symptoms of progressive coccidioidomycosis depend upon the site of dissemination. Any or all organs may be infected.

O. A morbilliform (measlelike) rash may appear 1-2 days after onset of symptoms. Arthralgia accompanied by periarticular swellings, often of the knees and ankles, is common. Erythema nodosum (painful red nodules on legs) may appear 2-20 days after onset of symptoms. Erythema multiforme (macular eruption with dark red papules or tubercules with no itching, burning, or rheumatic pain appearing in separate rings, concentric rings, disk-shaped patches, distributed elevations, and figured arrangements) may appear on the upper extremities, head, and thorax. Lab findings: May be moderate leukocytosis and eosinophilia. Sedimentation rate is elevated, returning to normal as infection subsides. There is a skin test available for coccidioidomycosis.

A. Coccidioidomycosis. Differential diagnosis: Viral,

bacterial, or other mycotic infections presenting flulike syndrome.

P. Bed rest and general symptomatic treatment until there is a complete regression of fever and a normal sedimentation rate. Amphotericin B has proven effective in some patients with disseminated disease, but because of its toxic properties, adult dose should not exceed 0.5-1 mg./kg. Therapy should begin with 1 mg./d. increasing by 5 mg. increments to 25-35 mg./d. or to 40-60 mg./d. in the acutely ill.

2-20. HISTOPLASMOSIS. Caused by Histoplasma capsulatum, a mold found in the soil in central and eastern United States, eastern Canada, Mexico, Central and South America, Africa, and Southeast Asia. Infection is presumably by inhalation of spores. May be carried by the blood to other parts of the body.

S. and O. Most cases are asymptomatic or mild and unrecognized. Symptomatic infections may present mild influenzalike characteristics lasting 1-4 days. In moderately severe cases, the patients have fever, cough, and mild chest pain lasting 5-15 days. Physical examination is usually negative.

Severe infections are divided into three groups: (1) Acute histoplasmosis frequently occurs in epidemics. Symptoms are marked prostration, fever, and occasional chest pain, but no particular symptoms relative to the lungs. X ray may show severe disseminated pneumonitis. Infection may last from 1 week to 6 months; it is rarely fatal. (2) Acute progressive histoplasmosis is usually fatal within 6 weeks or less. Fever, dyspnea, cough, weight loss, and prostration are usual sypmtoms. Diarrhea is usual in children. Mucous membrane ulcers of the oropharynx may be present. All the organs of the body are involved and liver and spleen nearly always enlarged. (3) Chronic progressive histoplasmosis is usually found in older patients with chronic obstructive lung disease. It closely resembles chronic tuberculosis; occasionally the patient will have both diseases. It appears to be primarily confined to the lungs, but all organs are involved in the terminal stage.

Lab findings: Sedimentation rate is elevated in moderate to severely ill patients. Leukopenia with normal differential count or neutropenia. Most patients with progressive disease show a progressive hypochromic anemia.

A. Histoplasmosis. Differential diagnosis: Mild cases--influenza; moderate--a typical pneumonia; severe cases--tuberculosis.

P. No specific therapy. Bed rest and symptomatic treatment for the primary form. Normal activity should not be resumed until fever has subsided. Amphotericin B has helped some patients (see coccidioidomycosis for treatment plan). Some milder forms of acute primary or early chronic disease respond to sulfadiazine therapy.

2-21. NORTH AMERICAN BLASTOMYCOSIS. A chronic systemic fungus infection caused by Blastomyces dermatitidis. Occurs more often in men. Found in central and eastern United States and Canada. A few cases have been found

in Mexico and Africa.

S. and O. Mild or asymptomatic cases are rarely found. Little is known of the mildest pulmonary phase of this disease. Cough, moderate fever, dyspnea, and chest pain are evident in symptomatic cases. These may disappear or progress with bloody and purulent sputum production, pleurisy, fever, chills, loss of weight, and prostration. Raised verrucous (tumor of the epidermis) cutaneous lesions that have an abrupt downward sloping border are usually present in disseminated blastomycosis. The surface is covered with miliary (small lesions resembling millet seeds) pustules. The border extends slowly leaving a central atrophic scar. Only cutaneous lesions are found in some patients. Lesions are most frequently seen on the skin, in bones, and in the genitourinary system, but any or all organs or tissues in the body can be attacked.

Lab findings: Usually leukocytosis, hypochromic anemia, and elevated sedimentation rate. Organism can be found in lesions. It is a thick-walled cell that may have a single bud.

A. North American blastomycosis. Differential diagnosis: Epididymitis, prostatitis, other diseases attacking bone or skin.

P. No specific treatment but amphotericin B (see coccidioidomycosis for treatment schedule). Surgical excision of cutaneous lesions may be successful. Careful follow-up for early evidence of relapse should be made for several years so therapy may be resumed if needed.

2-22. PARACOCCIDIOIDOMYCOSIS (South American Blastomycosis). Found only in South or Central America or Mexico. Caused by Paracoccidioides brasiliensis.

S. and O. Ulceration of nasopharynx usually the first symptom. Papules ulcerate and enlarge both peripherally and deeper into the subcutaneous tissue. Eventually may result in destruction of the epiglottis, vocal cords, and uvula with extension to the lips and face. Eating and drinking are extremely painful. Skin lesions of variable appearance may occur on the face. They may have a necrotic central crater with a hard hyperkeratotic border. Lymph node enlargement may be the presenting symptom or may follow mucocutaneous lesions. Lymph nodes eventually ulcerate and rupture through the skin. Some patients may present with gastrointestinal disturbances, including enlargement of liver and spleen, but symptoms are vague. Extensive ulceration of the upper gastrointestinal tract prevents sufficient intake and absorption of food causing malnutrition. Death may result from respiratory failure or malnutrition.

Lab findings: Elevated sedimentation rate, leukocytosis with neutrophilia showing a shift to the left, and sometimes eosinophilia and monocytosis. The fungus is a spherical cell that may have many buds arising from it.

A. Paracoccidioidomycosis.

P. Amphotericin B (see coccidioidomycosis for treatment plan) has had considerable success in hospitalized patients. Sulfadiazine (2-4 gm) daily or "Triple Sulfa" (1 gm) daily has been used for control and occasional cures have been reported following months or years of treatment. Relapses may occur when the drug is stopped. Drug toxicity with prolonged

high dosage is common. Rest and supportive care help in promoting a favorable response.

2-23. See Chapter 1, Section I, Integumentary System for sporotrichosis, dermatophyte infections (ringworm, athlete's foot, dandruff, etc.), and chromomycosis.

2-24. CANDIDIASIS (moniliasis, thrush). A yeast found normally in the mouth, vagina, and feces of most people. Overgrowth does not occur unless the "balance" of the oral flora is disturbed by debilitating or acute illness or in those being treated with antibiotics. Overgrowth is also favored by diabetes, iron deficiency anemia, and immunosuppressed status.

S. and O. Creamy-white curdlike patches anywhere in the mouth. Adjacent mucosa is usually erythematous, and scraping the lesion often uncovers a raw, bleeding surface. Commonly, a candidal lesion may appear as a slightly granular or irregularly eroded erythematous patch. Pain is usually present but fever and lymphadenopathy are uncommon. Concomitant candidiasis of the gastrointestinal tract (including the pharynx and esophagus) may occur. Vaginal overgrowth (see Chapter 7, Gynecology).

Systemic candidal infections are of two types: Endocarditis that almost always affects previously damaged heart valves, usually follows heart surgery or inoculation by contaminated needles or catheters. Splenomegaly and petechiae are usual, and emboli are common. Upper gastrointestinal tract candidiasis is the usual source in the other type of systemic infection. Dissemination follows antibiotic or cytotoxic chemotherapy for serious debilitating disease. The kidneys, spleen, lungs, liver, and heart are most commonly involved. Fungiuria is usual in renal disease.

Lab findings: Candida albicans is seen as a gram-positive budding cell and a pseudomycelium and is the most common cause of systemic disease.

A. Candidiasis. Differential diagnosis: Other systemic diseases depending on which area of the body is affected and other fungal skin infections.

P. Amphotericin B IV (as for coccidioidomycosis) is necessary for serious systemic infection. When combined with rifampin or flucytosine (Ancobon) 150 mg./kg./d. orally, lower doses of amphotericin B can be used and still prevent emergence of resistant organism.

Oral, gastrointestinal, and cutaneous lesions should be treated with amphotericin B, nystatin, or miconazole mouthwash, tablets, or lotions. Gentian violet, 1% in 10-20% alcohol, is also effective for oral, cutaneous, and vaginal lesions. Antibiotic therapy should be discontinued if possible. All patients with candidiasis should be checked for diabetes.

2-25. CRYPTOCOCCOSIS. An encapsulated budding yeast that is found worldwide in soil and on dried pigeon dung. Infection is acquired by inhalation.

S. and O. In the lungs, the infection may remain localized, heal, or disseminate. Upon dissemination, lesions may form in any part of the body; the most common part involved is the C.N.S. and is the usual

cause of death. Generalized meningoencephalitis occurs more frequently than localized granuloma in the brain or spinal cord. Solitary localized lesions may develop in the skin and, rarely, in bones or other organs. Pulmonary cryptococcosis presents no specific signs or symptoms. Many patients are asymptomatic, others may present with low-grade fever, pleural pain, and cough possibly with sputum production. C.N.S. involvement usually presents a history of recent URI or pulmonary infection. Usually the first and most prominent symptom is increasingly painful headaches. Vertigo, nausea, anorexia, ocular disorders, and mental deterioration develop. Neck rigidity is present, and Kernig's and Brudzinski's signs are positive. Patellar and achilles reflexes are often diminished or absent. Acneiform lesions enlarge slowly and ulcerate, often coalescing with other lesions to cover a large area.

Lab findings: Mild anemia, leukocytosis, and increased sedimentation rate.

A. Cryptococcosis. Differential diagnosis: Other systemic fungal infections with C.N.S. involvement.

P. Combination of amphotericin B (see coccidioidomycosis for dosage) and flucytosine (Ancobon), 150 mg./kg./d. in 6 hourly doses, may be curative in a 6-week regimen.

Section III - Bacterial

2-26. General. Bacteria are the most common disease causing organisms. They cause a wide variety of infections that can be located anywhere on or in the body.

2-27. STREPTOCOCCAL INFECTIONS.

a. Beta-hemolytic group A streptococci are the most common cause of exudative pharyngitis, and they also cause skin infections (impetigo). Respiratory infections are transmitted by droplets; skin infections by contact. Either may be followed by suppurative and nonsuppurative (rheumatic fever, glomerulonephritis) complications. Beta-hemolytic group B streptococci are often carried in the female genital tract and thus may infect the newborn. They are a common cause of neonatal sepsis and meningitis and may be associated with respiratory distress syndrome.

b. Streptococcal sore throat (strep throat).

S. Sudden onset of fever, sore throat, severe pain on swallowing, malaise, and nausea. Children may vomit or convulse. If scarlet fever rash occurs, the skin is diffusely erythematous, with superimposed fine red papules. The rash is most intense in the groin and axillas, blanches on pressure, and may become petechial. It fades in 2-5 days, leaving a fine desquamation.

O. Tender, enlarged cervical lymph nodes; the pharynx, soft palate, and tongue are red and edematous; and there may be a purulent exudate. In scarlet fever, the face is flushed with circumoral pallor, and the tongue is coated with protrusions of enlarged red papillae (strawberry tongue). CBC showing leukocytosis with an increase in polymorphonuclear neutrophils. Smears of the exudate from the throat show streptococci. Complications of streptococcal sore throat include sinusitis, otitis media, mastoiditis, peritonsillar abscess, suppuration of cervical lymph nodes,

reheumatic fever, and glomerulonephritis.

A. Streptococcal sore throat. Differential diagnosis: Streptococcal sore throat resembles (and cannot be reliably distinguished clinically from) the pharyngitis caused by adeno-viruses, herpes viruses, and occasionally other viruses. It also is commonly confused with infectious mononucleosis, diphtheria, candidiasis, and necrotizing ulcerative gingivostomatitis.

P. Antibiotic therapy is often given without proof of streptococcal origin if fever and leukocytosis accompany a sore throat with tender cervical lymph nodes.

(1) Benzathine penicillin G 1.2 million units IM as a single dose or procaine penicillin G 300,000 units IM daily x 10 days.

(2) Penicillin V 400,000 units q.8h. x 10 days.

(3) Patients hypersensitive to penicillin may be treated with erythromycin 500 mg. q.i.d. x 10 days.

(4) General measures include aspirin and gargling with warm saline solution to relieve sore throat. Bed rest and forced fluids until the patient is afebrile.

c. Rheumatic fever. Triggered by group A beta-hemolytic streptococcus producing a first attack of rheumatic fever in 0.3 percent of untreated or inadequately treated children. If a child has rheumatic fever once, his chances of reinfection within the next 5 years are 50 percent. Usually, the clinical manifestations of an attack of rheumatic fever tend to repeat themselves in subsequent attacks. The peak period of risk for children is 5-15 years of age.

S. and O. It takes two major or one major and two minor manifestations to justify a presumptive diagnosis of rheumatic fever. Major manifestations are:

(1) Active carditis (any one of the following).

(a) Significant new murmurs that are clearly mitral or aortic insufficiency.

(b) Pericarditis (pericardial friction rub or evidence of pericardial effusion).

(c) Evidence of congestive heart failure.

(2) Polyarthritis. Two or more joints must be involved either simultaneously or in a migratory fashion.

(3) Subcutaneous nodules. Nontender and freely movable under the skin, a few millimeters to 2 cm. in diameter, most commonly found over joints, scalp, and spinal column, and usually seen only in severe cases.

(4) Erythema marginatum. Usually occurs only in severe cases and is often mistaken for other types of skin lesions. It is a macular erythematous rash with a circinate border appearing primarily on

the trunk and extremities; the face is usually not involved.

(5) Sydenham's chorea. Progressively more severe emotional instability, involuntary movements, and muscular weakness often followed by muscular incoordination and slurring of speech. Involvement is not uncommonly limited to one side. Individual attacks are self-limiting, but may last up to 3 months.

Minor manifestations of rheumatic fever are:

(1) Fever: Usually low grade but occasionally 103-104 degrees F.

(2) Polyarthralgia: Pain in two or more joints without heat, swelling, and tenderness.

(3) History: Prior history of acute rheumatic fever or recent scarlet fever.

(4) Accelerated sedimentation rate.

(5) Positive throat culture or smear for group A streptococcus. Associated findings may include abdominal, back, and precordial pain; erythema multiforme, malaise, vomiting, nontraumatic epistaxis (nose bleed), weight loss, and anemia.

In the absence of carditis, rheumatic fever lasts on the average 89 - 27 days. With carditis, rheumatic fever lasts on the average 124 - 68 days.

A. Rheumatic fever. Differential diagnosis: Other causes of carditis, arthritis, and skin lesions. Other debilitating diseases, e.g., mononucleosis.

P. Therapy is divided into short-term and long-term treatment.

(1) Short-term therapy ranges from saving the life of a patient with severe carditis to relieving joint discomfort.

(a) Streptococcal infection must be eradicated. Benzathine penicillin G, in a single IM injection 0.6-1.2 million units, depending on patient weight, or 125-250 mg. of pencillin orally q.i.d. x 10 days. Alternate is erythromycin 250 mg. orally q.i.d. x 10 days.

(b) Aspirin (in the absence of severe carditis with congestive heart failure) 100 mg./kg./d. orally divided into 4 doses. Maximum dose regardless of weight is 5,000 mg./d. (four 5 gr. aspirin tablets q.i.d.). After 1 week reduce dosage to 50 mg./kg./d. in 4 doses and continue for at least 1 month.

(c) Congestive heart failure therapy (see Chapter 1, Section IV, The Circulatory System).

(d) Corticosteroids should be used for all patients with congestive heart failure and/or carditis. Dosage: prednisone 2 mg./kg./d. x 2 weeks orally, then 1 mg./kg./d. x 1 week, begin aspirin 50 mg./kg./d. on the third week and continue for 8 weeks.

(e) Strict bed rest is not required for patients with arthritis and mild carditis. Bed-to-chair with bathroom privileges and meals at the table for patients without severe carditis is all that is required. Strict bed rest should be maintained for patients with severe carditis at least until corticosteroid therapy is completed. Both should have gradual indoor ambulation followed by modified outdoor activity after symptoms have disappeared. This should last at least 2 months and the child should not return to school while there is clear evidence of rheumatic activity.

(f) Symptomatic treatment as necessary.

(2) Long-term therapy is aimed toward those patients who had carditis and/or congestive heart failure during the clinical course of rheumatic fever. At the present, antibacterial therapy is a lifetime undertaking to prevent recurrence. Benzathine penicillin G 1.2 million units IM once a month for life, or sulfadiazine 500 mg. in a single dose daily for patients under 60 lbs and 1 gm orally daily in a single dose for patients over 60 lbs, or erythromycin 250 mg. b.i.d. orally for patients allergic to penicillin and sulfonamides.

2-28. DIPHTHERIA. See Chapter 6, Pediatrics.

2-29. MENINGITIS.

a. General considerations. Meningitis is caused by numerous organisms. Even fungal and viral infections can cause meningitis. The most common causes of bacterial meningitis are meningococcal, pneumococcal, streptococcal, staphylococcal, Haemophilus influenzae, and tubercular infections. All but tuberculous meningitis are similar in sign and symptoms and treatment.

b. Meningococcal meningitis. About 15-40 percent of the population are nasopharyngeal carriers of meningococci, but few develop the disease. Infection is transmitted by droplets.

S. High fever, chills, and headache; back, abdominal, and extremity pain; and nausea and vomiting are present. In severe cases, rapidly developing confusion, delirium, and coma occur. Twitch or frank convulsions may also be present.

O. Petechial rash of skin and mucous membranes is found in most cases. Petechiae may vary from pinhead size to large ecchymoses or even areas of skin gangrene that may later slough if the patient survives. These petechiae usually fade in 3-4 days. Neck and back stiffness with positive Kernig (sitting or lying with the thigh flexed upon the abdomen, the leg cannot be completely extended) and Brudzinski sign. (In meningitis, flexion of the neck usually results in flexion of the hip and knee. Also when passive flexion of the lower limb on one side is made, a similar movement will be seen in the opposite limb.) Shock due to the effects of endotoxin may be present and is a bad prognostic sign.

CBC shows usually marked leukocytosis early in the course of the disease. Urine may contain protein, casts, and red cells. Lumbar puncture reveals a cloudy to frankly purulent cerebrospinal fluid, with elevated pressure, increased protein, and decreased glucose content. The fluid usually contains numerous white cells and gram-negative intracellular diplococci. The absence of organisms in a gram-stained smear does not rule

out the diagnosis.

A. Meningococcal meningitis. Differential diagnosis: Other meningitides.

P. Antibacterial therapy by IV route must be started immediately. Aqueous penicillin G 24 million units/24 hours for adults and 400,000 units per kg./24 hours for children is the drug of choice. One-fourth of the dose is given rapidly IV and the rest by continuous drip. If the patient is allergic to penicillin, chloramphenicol 100 mg./kg. daily is the preferred alternate. Treatment should continue for 7-10 days by IV route. If the possibility of Haemophilus influenzae meningitis has not been ruled out, give both sodium ampicillin 300 mg./kg. daily IV (1/4 of the dose initially and the remainder in divided doses every 4 hours) and chloramphenicol (same as before) (separately, not in mixed doses). General measures include Ringer's lactate IV drip for maintenance and to prevent hypovolemic shock. Monitor vital signs closely. If patient survives the first day, the prognosis is excellent.

2-30. TYPHOID FEVER. Caused by the gram-negative rod Salmonella typhi. Infection is transmitted by consumption of contaminated food or drink. The sources of most infections are chronic carriers with persistent gallbladder or urinary tract infections. The incubation period is 5-14 days.

S. Onset is usually insidious but may be abrupt, especially in children, with chills and a sharp rise in temperature. Usually the patient develops increasing malaise, headache, cough, general body aching, sore throat, and nosebleeds. Frequently there is abdominal pain, constipation or diarrhea, and vomiting. During this period, the fever ascends in a stepladder fashion; the maximum temperature each day is slightly higher than the previous day. Temperature is generally higher in the evening than the morning. After 7-10 days the fever stabilizes and the patient becomes very sick. "Pea soup" diarrhea or severe constipation or marked abdominal distention is common. In severe cases, the patient lies motionless and unresponsive, with eyes half shut and appearing wasted and exhausted (the "typhoid state"), but can usually be aroused to carry out simple commands. If the patient survives this portion and no complications occur, he gradually improves. Fever declines in a stepladder fashion to normal in 7-10 days and with it the other symptoms gradually disappear. Relapses may occur as late as 1-2 weeks after temperature returns to normal, but they are usually milder than the original infection.

O. Early physical findings are slight. Later, splenomegaly, abdominal distension and tenderness, relative bradycardia, dicrotic (double wave) pulse, and occasionally systolic murmur and gallop rhythm appear. During the second week of the disease, a rash (rose spots) appears principally on the trunk (pink papules 2-3 mm. in diameter that fade on pressure) and disappears over a period of 3-4 days. Leukopenia and moderate anemia are the rule. The organism may be found in the stool after the first week or possibly may be found in the urine. Blood, stool, or urine cultures are usually positive after the first week.

A. Typhoid fever. Differential diagnosis: Tuberculosis, viral pneumonia, psittacosis, infective endocarditis, brucellosis, or Q fever.

P. Active immunization should be provided for household contacts of typhoid carrier, travelers to endemic areas, and during epidemic outbreaks. Food and water should be protected and waste should be

adequately disposed of. Specific measures include ampicillin 100 mg./kg. daily IV or 4-250 mg. capsules every 4 hours orally, or chloramphenicol 1 gm q.6h. orally or IV until fever disappears, then 500 mg. q.6h. for 2 weeks. IV fluids may be necessary to supplement oral intake and maintain urine output; 100 mg. hydrocortisone q.8h. may help severely toxic patients. Strict stool and urine isolation techniques must be observed. Treatment of carriers is usually ineffective, but a trial of ampicillin first then chloramphenicol should be tried. Cholecystectomy may be effective.

2-31. CHOLERA. An acute diarrheal disease caused by vibrio cholerae or related vibrios. The infection is caused by ingestion of food or drink contaminated by feces from cases or carriers. Cholera is fatal in 50 percent of all untreated patients. The incubation period is 1-5 days, but only a small minority of those exposed become ill.

S. Typical cases have an explosive onset of frequent, watery stools that soon lose all fecal appearance and odor. The stool is grayish, turbid, and liquid, containing degenerated epithelium cells and mucus, but rarely gross pus or blood. The patient can lose up to 1 liter per hour. Vomiting may also occur early.

O. The patient rapidly becomes dehydrated and acidotic, with sunken eyes, hypotension, subnormal temperature, rapid and shallow breathing, muscle cramps, oliguria, shock, and coma. Hematocrit will rise sharply due to loss of plasma resulting in a concentration of red cells. The vibrios can easily be cultured from the stool and might possibly be found using Gram's stain of stool specimens.

A. Cholera. Differential diagnosis: Other causes of severe diarrhea, particularly those due to shigellae, viruses, E. coli enterotoxins and protozoa in endemic areas.

P. Water and electrolyte loss must be restored promptly and continuously, and acidosis must be corrected. Diarrheal loss and hemoconcentration must be measured continuously. In moderately ill patients, it may be possible to provide replacement by oral fluids given in the same volume as that lost. (See Chapter 18, IV Therapy.) Those unable to take fluid by mouth require IV fluid replacement. Tetracycline 500 mg. q.6h. x 3-5 days should also be given. Effective decontamination of excreta is essential, but strict isolation of patients is unnecessary and quarantine is undesirable.

Prevention: Cholera vaccine gives only limited protection and is of no value in controling outbreaks. In endemic areas, all water, other drinks, food, and utensils must be boiled or avoided.

2-32. BACILLARY DYSENTERY. See Chapter 1, Section V, Digestive System.

2-33. GAS GANGRENE. Produced by entry of one of several clostridia into devitalized tissues. These gram-positive rods grow and produce toxins under anaerobic conditions.

S. Onset usually sudden with rapidly increasing pain in the affected area. The wound becomes swollen and the surrounding skin is pale. This is followed by a discharge of a brown to blood-tinged, serous, foul-smelling fluid from the wound. As the disease advances, the surrounding tissue changes from pale to dusky and finally becomes deeply

discolored, with coalescent, red, fluid-filled vesicles. In the last stages of the disease, severe prostration, stupor, delirium, and coma occur.

O. The increasing pain is accompanied by a fall in blood pressure. Temperature may be elevated, but not proportionate to the severity of infection. Gas may be palpable in the tissues. In clostridial sepsis, hemolysis and jaundice are common, often complicated by renal failure. Gram's stain of the exudate should show the organism and is a valuable clue, but the clinical picture must be present to make the diagnosis.

A. Gas gangrene. Differential diagnosis: Other infections that cause gas formation, e.g., enterobacter, Escherichia, and mixed anaerobic infections including Bacteroides and Peptostreptococcus.

P. Antibiotic therapy in the form of penicillin, chloramphenicol, or chlortetracycline should be started promptly in heroic doses. Massive debridement of all involved tissue. Frequently gas in the subcutaneous tissue or fascial planes extends beyond the area of muscle involvement. In such cases the overlying skin should be incised widely and the necrotic fascia excised. Careful and complete debridement of all wounds and good wound care will eliminate almost all chance for gangrene to develop.

2-34. TETANUS. An acute central nervous system intoxication caused by toxins produced by the slender, spore-forming, gram-positive anaerobic bacillus Clostridium tetani that are found mainly in the soil and in the feces of animals and humans and that enter the body by wound contamination. In the newborn, infection often enters through the umbilical stump. Incubation period is 5-15 days.

S. Occasionally, the first symptom is pain and tingling at the wound site followed by spasticity of the nearby muscle groups; this may be all that happens. Usually the presenting symptoms are stiffness of the jaw, neck stiffness, difficulty in swallowing, and irritability. Hyperreflexia develops later, with spasms of the jaw muscles (trismus) or facial muscles and rigidity and spasm of muscles of the abdomen, back, and neck.

O. Painful tonic convulsions caused by minor stimuli (any loud noise, etc.) are common. The patient is awake and alert during the entire course of the illness. During convulsions, the glottis and the respiratory muscles go into spasm so that the patient is unable to breath, and cyanosis and asphyxia may ensue. Temperature is only slightly elevated. Although there is usually a leukocytosis, the diagnosis of tetanus is made clinically.

A. Tetanus. Differential diagnosis: Other types of acute C.N.S. infections and strychnine poisoning should also be considered.

P. Active immunization with tetanus toxoid should be universal. Adequate debridement of wounds and a booster tetanus immunization is the most important preventive measure. Specific treatment: Give tetanus immune globulin (human) 5,000 units IM. If not available, test for sensitivity to horse serum and give 100,000 units tetanus antitoxin IV. Place patient at bed rest and minimize stimulation. Sedation and anticonvulsant therapy is essential. Penicillin is of value but should not

be substituted for antitoxin. IV fluids as necessary. Tracheostomy and/or assisted respiration may be required. Mortality rate is about 40 percent higher in children and very old people.

2-35. BOTULISM. See Chapter 1, Section V, Digestive System.

2-36. *ANTHRAX.* A disease of sheep, cattle, horses, goats, and swine caused by Bacillus anthracis, a gram-positive spore-forming aerobe transmitted to humans by entry through broken skin mucous membranes or by inhalation. Uncommon, but most apt to occur in farmers, veterinarians, and tannery and wool workers.

S. Cutaneous anthrax. An erythematous papule appears on the exposed area of skin and becomes vesicular with a purple to black center. The area around the lesion is swollen or edematous and surrounded by vesicles. The center finally forms a necrotic eschar and sloughs. Malaise, headache, nausea, and vomiting may be present.

Pulmonary anthrax (woolsorter's disease): Fever, malaise, headache, labored or difficult breathing (dyspnea), and cough.

O. Cutaneous anthrax. Regional adenopathy and variable fever may be present. After eschar sloughs, sepsis may occur at times manifested by shock, cyanosis, sweating, and collapse. Hemorrhagic meningitis may occur. Anthrax sepsis may occur without a skin lesion.

Pulmonary anthrax: Congestion of the nose, throat, and larynx; and auscultatory or X ray signs of pneumonia.

Lab findings: White count may be elevated or low. Smears of skin lesions show gram-positive encapsulated rods.

A. Anthrax. Differential diagnosis: Rarely gram-positive spore-forming aerobic bacilli other than B. anthracis can produce similar disease.

P. Penicillin G 10 million units IV daily; or in mild localized cases tetracycline 500 mg. q.6h. x 10 days.

2-37. TULAREMIA. An infection of wild rodents, particularly rabbits and muskrats, transmitted to humans by contact with animal tissue (e.g., trapping and skinning rabbits, etc.), by the bite of certain ticks and biting flies, by eating infected undercooked meat, or by drinking contaminated water. Incubation period is 2-10 days.

S. Fever, headache, and nausea begin suddenly, and a papule develops at the site of inoculation and soon ulcerates. Lesion may be on the skin of an extremity or in the eye. If ingested, it may manifest as gastroenteritis, stupor, and delirium. There may be rashes, generalized aches, and prostration.

O. Regional lymph nodes become enlarged and tender and may suppurate (to form pus). In any type of involvement, the spleen may be enlarged. Asymptomatic infection is not rare. W.B.C. may be slightly elevated or normal. Cultures of blood, lesion, or lymph node aspirate require special culture media. There is a delayed type skin test (read in 48 hrs) that can be used.

A. Tularemia. Differential diagnosis: Rickettsial and meningococcal infections, cat scratch fever, infectious mono, and various pneumonias and fungal diseases.

P. Streptomycin 500 mg. q.6-8h. IM, together with tetracycline 500 mg. q.6h. until 5 days after patient is afebrile. Adequate fluid intake is essential and O_2 therapy may be necessary. Drainage of fluctuant lymph nodes may be needed and is safe after proper antibiotic therapy for several days.

2-38. PLAGUE. An infection of wild rodents with Pasteurella pestis, a small gram-negative rod. Transmitted from rodent to rodent and to humans by the bites of fleas. If a plague victim develops pneumonia, the infection can be transmitted by droplets and an epidemic may start. The incubation period is 2-10 days.

S. Usually sudden onset with high fever, malaise, intense headache, and generalized muscular ache. The patient appears profoundly ill and very anxious. Delirium may ensue. With systemic spread, the patient may rapidly become severely septic and comatose with purpuric spots (black plague) appearing on the skin.

O. Tachycardia is usually noted with onset of symptoms. If pneumonia develops, tachypnea, productive cough, blood-tinged sputum, and cyanosis also occur. Meningeal signs may develop; a pustule or ulcer at the site of inoculation and signs of lymphangitis may occur. Axillary, inguinal, or cervical lymph nodes become enlarged and tender and may eventually suppurate and drain. Primary plague pneumonia from droplets coughed by another patient with plague pneumonia is a fulminant pneumonitis with bloody, frothy sputum and sepsis. It is usually fatal unless treatment is started within a few hours of onset.

Lab findings: W.B.C. 12-20,000; the plague bacillus may be found in smears from aspirates of buboes using Gram's stain.

A. Plague. Differential diagnosis: Lymphadenitis accompanying staph or strep infections of an extremity, lymphogranuloma venereum, syphilis, or tularemia. Systemic manifestations resemble those of enteric or rickettsial fevers, malaria, or flu.

P. Therapy must be started promptly when plague is suspected. Streptomycin 1 gm. IM q.6h. x 2 days then 500 mg. q.6-8h. tetracycline 500 mg. q.6h. is given at the same time. IV fluids, pressor drugs, oxygen, and tracheostomy are used as required.

2-39. LEPROSY (Hansen's disease). A chronic infectious disease caused by the acid-fast rod Mycobacterium leprae. Mode of transmission is unknown; probably involves prolonged exposure in childhood; adults rarely become infected (e.g., by tattooing). Endemic in tropical and subtropical Asia, Africa, Central and South America, the Pacific regions and southern USA.

S. & O. Onset is insidious, lesions involve skin, superficial nerves, nose, pharynx, larynx, eyes, and testicles. May occur as pale anesthetic macular lesions 1-10 cm. in diameter, discrete erythematous infiltrated nodules 1-5 cm. in diameter, or diffuse skin infiltration. Neurologic disturbances are manifested by nerve infiltration and thickening, with resultant anesthesia, neuritis, paresthesia, trophic ulcers, bone reabsorption, and shortening of the digits. In untreated

cases, the disfigurement may be extreme. Leprosy is clinically and by laboratory tests divided into two types: lepromatous and tuberculoid. In the lepromatous type, the course is progressive and malignant with abundant acid-fast bacilli in the skin lesion and a negative lepromin skin test. The tuberculoid type is benign and nonprogressive with severe asymmetrical nerve involvement of sudden onset with few bacilli in the lesions and a positive lepromin skin test. Eye involvement (keratitis and iridocyclitis), nasal ulcers, nose bleeds, anemia, and lymphadenopathy may occur.

A. Leprosy. Differential diagnosis: Skin lesions resemble those of lupus erythematosus, sarcoidosis, syphilis, erythema nodosum, erythema multiforme, and vitiligo.

P. Untreated lepromatous leprosy is progressive and fatal in 10-20 years. In tuberculoid leprosy, spontaneous recovery usually occurs in 1-3 years; however, it may produce crippling deformities. With treatment, lepromatous leprosy regresses slowly (over a period of 3-8 years). Recovery from tuberculoid leprosy is more rapid. Return of symptoms is always possible and it is safe to assume that the bacilli are never totally eradicated. The treatment of leprosy is very complicated, requiring numerous drugs (dapsone, amithiozone, thalidomide, rifampin, clofazimine and corticosteroids) in increasing doses over a period of years or indefinitely. All of this necessitates evacuation to a hospital or area better equipped to handle these cases.

2-40. TUBERCULOSIS. Caused by acid-fast Mycobacterium tuberculosis and characterized by the formation of tubercles in the lung. Occurs almost exclusively by inhalation of airborne droplets from the cough of a person with tubercle bacilli in the sputum. Ingestion of milk containing tubercle bacilli (unpasteurized) is another mode of transmission. The danger of infection from contaminated surfaces is negligible. The first or primary infection is usually a self-limiting disease in children that escapes detection. A few patients develop progressive primary tuberculosis. Another small percentage develop progressive pulmonary disease. Primary infections occurring in adults may evolve into progressive pulmonary disease without the characteristic changes of primary disease seen in children. Most people who are infected at any age do not develop the disease. Malnutrition, diabetes, measles, chronic corticosteroid treatment, silicosis, and general debility favor progression of infection into progressive pulmonary disease.

S. Symptoms may be absent or mild and nonspecific in the presence of active disease. The most frequent symptoms, when present, are cough, malaise, easy fatigability, weight loss, low-grade afternoon fever, night sweat, and pleuritic pain. Cough, when present, has no specific characteristics. Patients with pulmonary tuberculosis occasionally present with symptoms due to extra pulmonary complications such as laryngeal, renal, intestinal, or C.N.S. involvement.

O. Pulmonary signs may be difficult to elicit even in the presence of active disease. Fine persistent rales over the upper lobes may be found. These are best heard after a slight cough. Advanced disease may lead to retraction of the chest wall, deviation of the trachea, wheezes, rales, and signs of pulmonary consolidation. Pulmonary TB cannot be ruled out by physical examination only. A chest X ray is the minimum diagnostic requirement. Lab findings: Sputum smears are positive when bacteria count is high but should be confirmed with culture. Tine test may be used for

screening, but PPD 0.1 cc. 1.0 is more accurate. These tests are only for screening of patients, not for diagnostic purposes. Patients with positive skin tests should have chest X rays.

A. Pulmonary tuberculosis. Differential diagnosis: TB can mimic almost any pulmonary disease such as bacterial or viral pneumonias, lung abscess, pulmonary mycoses, bronchogenic carcinoma, sarcoidosis, and "atypical" (nontuberculosis) mycobacterial infections. Negative tine or PPD test make diagnosis of TB very unlikely.

P. Prevention: Patients with active TB should be isolated during the first 2 weeks of treatment and taught to cover their mouth and nose with disposable tissue during coughing. Close contacts must have skin test and if positive, chest X rays. If negative they should be retested in 2 months. If contact is positive and chest X ray is negative, they should receive isoniazid treatment for 1 year. Infants and children who are in close contact should be given isoniazid even if skin tests are negative, but their treatment can be discontinued if the skin test is still negative 3 months after exposure is discontinued. Persons who convert from negative to positive within 2 years who have negative X rays should receive isoniazid for 1 year. Positive reactors with negative X rays with high risk factors (e.g., prolonged corticosteroid therapy for other diseases, Hodgkin's disease, leukemia, diabetes, and silicosis) should receive isoniazid for 1 year. Preventive treatment with isoniazid consists of 300 mg. daily (10 mg./kg. daily for children) for 1 year.

Treatment for active TB

Drug	Adult Dose	Comments
Isoniazid (NH)	5-10 mg./kg. daily orally	With the sole exception of preventive treatment, this should be used only in combination with other drugs.
and		
Streptomycin	1 gm IM daily or twice weekly	
and		
ethambutol	15 mg./kg. daily orally	
or		
Aminosalicylic acid(PAS)	4-5 gm orally t.i.d. after meals	Use only when ethambutol is not available
or Isoniazid	same as above	
and		
Rifampin	600 mg. daily orally	

Most authorities advise a minimum of 12 months of treatment after it has been shown X ray lesions are stable, no cavitation is present, and cultures are negative (control is usually achieved in 2-3 months).

Severe cases may require surgery. Because of the complications, special tests, and prolonged treatment, it is best to evacuate these patients if possible.

Section IV - Viral

2-41. GENERAL. Viruses are extremely small organisms that cannot be seen under a normal microscope. Viruses cause a variety of important infectious diseases; among these are the common cold, yellow fever, hepatitis, and the majority of the infections of the upper respiratory tract.

2-42. MEASLES (Rubeola). An acute systemic viral infection transmitted by inhalation of infective droplets. One attack confers permanent immunity. Communicability is greatest during the preemptive stage, but continues as long as the rash remains. Incubation period is 10-14 days.

S. Fever often as high as 104-105 degrees F., coryza (nasal obstruction, sneezing, sore throat), persistent and nonproductive cough, malaise (may be marked), and conjunctivitis with redness, swelling, photophobia, and discharge. Koplik's spots (small red spots with bluish-white centers on the oral mucosa and often on the inner conjunctival folds and vaginal mucous membrane) appear about 2 days before the rash and last 1-4 days. Rash usually appears first on the face and behind the ears 4 days after the onset of symptoms.

O. The pharynx is red and a yellowish exudate may appear on the tonsils. The tongue is coated in the center and the tip and margins are red. Moderate generalized lymphadenopathy is common; splenomegaly occurs occasionally. The initial lesions of the rash are pinhead-sized papules that coalesce to form the brick-red irregular blotchy maculopapular rash and that may further coalesce, in severe cases, to form an almost uniform erythema on some areas of the body. By the second day, the rash begins to coalesce on the face as it appears on the trunk. On the third day, the rash begins to coalesce on the trunk as it appears on the extremities and begins to fade on the face. Thereafter, it fades in the order of its appearance. Hyperpigmentation remains in fair-skinned individuals and severe cases.

A typical measles is a rarely occurring syndrome in children or adults who have received inactive or live measles vaccine and as a result have developed hypersensitivity rather than protective immunity. When infected with mild measles virus, they develop high fever, unusual rashes (papular, hemorrhagic), arthralgias, and pneumonitis, often with severe illness and a substantial mortality rate. Leukopenia is usually present unless there is a secondary bacterial infection. Complications include encephalitis, bronchopneumonia or bronchiolitis, and secondary bacteria infections.

A. Measles. Differential diagnosis: Rubella, chickenpox, smallpox, infectious mononucleosis, enterovirus infections, and drug eruptions.

P. Isolate the patient for the week following onset of rash and keep at bed rest until afebrile. Give aspirin, saline eye sponges, vasoconstrictor nose drops, and sedative cough mixture as necessary; treat complications as needed.

Prevention: Multiple virus vaccines are available (measles,

mumps, rubella) and can be used for prevention in the first 24 hours after exposure.

2-43. RUBELLA (German measles). A systemic viral infection transmitted by inhalation of infective droplets. Only moderately communicable. One attack usually confers permanent immunity. Disease can be transmitted for 1 week before rash appears. Incubation period is 14-21 days.

S. Fever and malaise, usually mild, with tender suboccipital adenitis may precede eruption by 1 week. Symptoms of mild head cold may be present. Joint pains occur in 25 percent of adult cases. Symptoms usually subside in about 7 days. A fine, pink maculopapular rash appears on face, trunk, and extremities in rapid progression, usually lasting one day in each area. Rubella without the rash is as common as with the rash.

O. Posterior cervical and postauricular lymphadenopathy is very common. Redness of the palate and throat, sometimes blotchy, may be noted. Diagnosis can be suspected when there is epidemiologic evidence of rubella in the area. CBC may show leukopenia early and may be followed by an increase in plasma cells.

Complications: In pregnancy, risk to the fetus is high in the first trimester and continues into the second trimester. An infant acquiring rubella in uterus may be normal at birth, but more likely will have a wide variety of manifestations including growth retardation, maculopapular rash, thrombocytopenia (abnormal decrease in number of blood platelets), cataracts, deafness, congenital heart defects, organomegaly (enlargement of organs), and many other manifestations.

A. Rubella. Differential diagnosis: Infectious mononucleosis, echovirus infections, and coxsackievirus infections.

P. Symptomatic treatment: Aspirin, fluids, rest. Rubella is mild and rarely lasts more than 3-4 days. Congenital rubella has high mortality rate and congenital defects require years of medical and surgical management.

Prevention: Live attenuated rubella virus vaccine offers complete protection. Birth control must be practiced by women for at least 3 months after the use of the vaccine.

2-43. HERPES ZOSTER (Shingles). See Chapter 1, Section I, Integumentary System.

2-44. VARICELLA (Chickenpox). See Chapter 6, Pediatrics.

2-45. VARIOLA (Smallpox). An acute, contagious, systemic viral disease. Transmitted by direct contact with infected patient or handling of contaminated articles. Thought to be eradicated worldwide as of 1979 through the efforts of the W.H.O. using smallpox vaccination. Incubation period is 7-17 days, usually 10-12 days to onset of illness, and 2-4 more days to onset of rash.

S. Abrupt onset with chills, headaches (usually frontal), intense lumbar pain, fever (up to 104 degrees F. or higher), nausea, or more frequently vomiting. Fever falls sharply on evening of third or morning of fourth day, often to normal, and eruption appears as temperature falls. Normally, rash starts first on face and soon after, on extremities

and to lesser extent on trunk.

O. Rash is of the same character in any general location, in this respect, differing markedly from rash of chickenpox. Rash is initially macules; about the second day they become papules that become vesicles from the third to fifth day. The vesicles increase in size and by the seventh to eighth day become well developed pustules. Finally scabs form. These scabs fall off in about 3 or 4 weeks. The lesions of smallpox are deep-seated with a thick protective covering and do not rupture easily. The lesion does not collapse when pricked by a needle. Recovery in untreated cases is doubtful.

A. Smallpox. Differential diagnosis: Chickenpox, herpes zoster.

P. Absolute isolation of patient in a screened but well ventilated room until all scabs and crusts have disappeared. Symptomatic treatment is forced fluids, aspirin. Do not use ointments on the skin before the drying up is complete as it increases the likelihood of abscess formation. Close attention must be given to the eyes; if necessary, they may be irrigated several times a day with 2% sodium bicarbonate solution. Weak iodine or weak permanganate baths can be used on the skin for cleansing and as a deodorant.

Successful vaccination against smallpox is an absolute preventive, but this should be repeated during an epidemic or when an individual has been exposed.

2-46. MUMPS (Endemic parotitis). See Chapter 6, Pediatrics.

2-47. POLIOMYELITIS. Three antigenically distinct types are recognized, with no cross immunity between them. Probably acquired by respiratory droplet route or by ingestion. Incubation period is 5-35 days (usually 7-14 days). Infectivity is maximal during the first week. Since the introduction of effective vaccine, poliomyelitis has become rare in the developed areas of the world.

S. and O.

(1) Abortive poliomyelitis: Headache, fever, vomiting, diarrhea, constipation, and sore throat.

(2) Nonparalytic poliomyelitis: Headache; neck, back, and extremity pain; lethargy; and irritability are present. Muscle spasm in extensors of neck and back is always present and usually present in the hamstring muscles. Muscle spasm is variably present in other muscles. Spasm may be seen when patient is at rest or elicited by putting each muscle through the maximum range of motion. Resistance to neck flexion is noted after a varying range of free flexion. Straight leg raising is less than 90 degrees. The muscles may be tender to palpation.

(3) Paralytic poliomyelitis: May occur at any time during the febrile (feverish) period. Symptoms of nonparalytic poliomyelitis plus tremor and muscle weakness. Paresthesia and urinary retention are noted occasionally. Constipation and abdominal distention are common. Paralytic poliomyelitis may be divided into two forms that may coexist. Spinal poliomyelitis (weakness of muscles supplied by spinal nerves) and bulbar poliomyelitis (weakness of muscles supplied by cranial nerves and variable

"encephalitis" symptoms). Other symptoms include diplopia (double vision) (uncommon), facial weakness, dysphasia (speech impairment), nasal voice, weakness of the sternocleidomastoid and trapezius muscles (difficulty in chewing, inability to swallow or expel saliva), and regurgitation of fluids through the nose. The most life threatening aspect is respiratory paralysis. Paralysis may quickly become maximal or progress over several days until temperature becomes normal. Deep tendon reflexes are diminished or lost, often asymmetrically. Lethargy or coma may be due to encephalitis or hypoxia, most often caused by hypoventilation.

Lab findings: W.B.C. may be normal or slightly elevated.

A. Poliomyelitis. Differential diagnosis: Other forms of aseptic meningitis due to other enterovirus (muscle tenderness and spasm, if present, point to polio) is very difficult to distinguish from polio. Acute infectious polyneuritis (Guillain-Barre) and tick bite paralysis may initially resemble poliomyelitis.

P. Symptomatic: Maintain comfortable but changing positions on a firm mattress with footboard, sponge rubber pads or rolls, sandbags, and light splints. Hotpacks for the extremities and analgesic drugs usually control muscle spasm and pain. IV therapy may be needed to prevent dehydration. Indwelling catheter may be required. Intestinal hypoactivity may lead to fecal impaction. Cases of bulbar poliomyelitis involving respiratory muscles require intensive care. Attention must be focused on maintaining a clear airway, handling secretions, preventing respiratory infections, and maintaining adequate ventilation. Assisted ventilation and tracheostomy are often required.

Prevention of deformities is best accomplished by avoiding active exercise during febrile period and substituting passive range of motion exercises and frequent changes of position. As soon as fever subsides, early mobilization and active exercise should be started. Early bracing and splinting for therapeutic purposes are recommended.

Prevention: Oral live virus vaccine (Sabin), the trivalent form is preferable for immunizing children and infants. Adults who are exposed to poliomyelitis or plan to travel in endemic areas should receive the oral vaccine also.

2-48. DENGUE (Breakbone fever, dandy fever). Viral disease transmitted by Aedes mosquito. Occurs only in active mosquito season (warm weather). Incubation period 3-15 days (usually 5-8 days).

S. Sudden onset of high fever, chilliness, severe aching (breakbone) of the head, back, and extremities, accompanied by sore throat, prostration, and depression. Initial febrile phase lasts 3-4 days, usually followed by remission of a few hours to 2 days. A rash appears in 80 percent of cases during remission or during second febrile phase that lasts 1-2 days and is accompanied by similar but milder symptoms.

O. May be conjunctival redness and flushing or blotching of the skin. Rash may be scarlatiniform, morbilliform, macropapular, or petechial, appearing first on dorsum of hands and feet and spreads to the arms, legs, trunk, and neck, but rarely to the face. Rash lasts 2 hours to several days and may be followed by peeling. Petechial rashes and gastrointestinal hemorrhages occur in a high portion of cases in Southeast Asia.

Lab findings: Leukopenia is characteristic.

A. Dengue. Differential diagnosis: Before the rash appears, it is difficult to distinguish from malaria, yellow fever, or influenza.

P. Symptomatic treatment: Treat shock, give salicylates as required, forced fluids, gradual restoration of activity during prolonged convalescence.

Prevention: Mosquito control. An effective vaccine has been developed but has not been produced commercially.

2-49. COLORADO TICK FEVER. An acute viral infection transmitted by tick bites, limited to western USA and most prevalent during tick season (March to August). Incubation period 3-6 days.

S. Abrupt onset of 102-105 degree F. fever, sometimes with chills. Severe myalgia, headache, photophobia, anorexia, nausea, vomiting, and generalized weakness.

O. Abnormal findings are limited to an occasional faint rash. Fever lasts 3 days followed by remission of 2-3 days, and then by full recurrence of symptoms for 3-4 days. Occasionally, there may be 2-3 bouts of fever. Lab findings: Leukopenia (2,000-3,000 W.B.C. with a shift to the left.

A. Colorado tick fever. Differential diagnosis: Influenza, Rocky Mountain spotted fever, and other acute leukopenic fevers.

P. Symptomatic treatment: Aspirin or codeine may be given for pain.

2-50. RABIES. See Chapter 12, Bites.

2-51. YELLOW FEVER. Transmitted by Aedes and jungle mosquitoes. Endemic to Africa and South America. Incubation period is 3-6 days.

S. Mild form: Malaise, headache, fever, retro-orbital pain, nausea, vomiting, and photophobia. Severe form: Same symptoms with sudden onset and then severe pains throughout the body, extreme prostration, bleeding into the skin and from mucous membranes, "coffee ground" vomitus, and jaundice, followed by a period of calm on about the third day when the temperature returns to normal. Then fever returns, bleeding, and later delirium.

O. Mild form: Bradycardia may be present. Severe form: Tachycardia, oliguria, erythematous face, and conjunctival redness during congestive phase. After the period of calm; bradycardia, hypotention, jaundice, and hemorrhages (gastrointestinal tract, bladder, nose, mouth, subcutaneous).

Lab findings: Proteinuria sometimes as high as 5-6 gm/L. and disappears with recovery; hematuria and leukopenia occurs, although it may not be present at the onset.

A. Yellow fever. Differential diagnosis: Mild form is difficult to distinguish from hepatitis, leptospirosis, and other forms of jaundice on clinical evidence alone.

P. Symptomatic treatment: Liquid diet, limiting food to high-carbohydrate, high-protein liquids as tolerated; IV glucose and normal saline as required; analgesics and sedatives as required; and saline enemas for constipation.

Prevention: Mosquito control and live virus vaccine for persons living in or traveling to endemic areas.

Prognosis: Mortality is high in severe form, with death occurring most commonly between the sixth and ninth days. In survivors, temperature returns to normal by seventh or eighth day.

2-52. INFLUENZA. See Chapter 1, Section IX, Eye, Ear, Nose, and Throat.

2-53. VIRAL HEPATITIS.

a. Hepatitis A ("infectious" or short incubation period hepatitis) is a generalized viral infection in which liver involvement dominates the clinical picture. It may occur sporadically or in epidemics. Transmission is usually by fecal-oral route; however, it may be transmitted (rarely) by contaminated needle stick or transfusion. There is no known carrier state with hepatitis A.

b. Hepatitis B ("serum" or long incubation period hepatitis) usually transmitted by inoculation of infected blood or blood products but can be spread by oral or sexual contact. Fecal-oral transmission has also been documented. Approximately 5-10 percent of infected individuals become carriers. The incubation period is 6 weeks to 6 months. The clinical picture is similar in Type A and B hepatitis but in Type B, the onset tends to be more insidious.

S. Clinical picture is extremely variable from asymptomatic infection without jaundice to a fulminating disease and death in a few days.

Prodromal phase: Onset varies from abrupt to insidious with general malaise, myalgia, arthralgia, easy fatigability, upper respiratory symptoms (nasal discharge, pharyngitis), and severe anorexia. Nausea and vomiting are common and diarrhea or constipation may occur. Fever usually present but rarely more than 103.1 degrees F. Return of temperature to normal often coincides with onset of jaundice. Chills or chilliness may mark an acute onset. Abdominal pain usually mild and constant in upper right quadrant or right epigastrium often aggravated by jarring or exertion. A distaste for smoking paralleling anorexia may occur early.

Icteric (jaundice) phase: Usually occurs after 5-10 days but may appear at same time as initial symptoms. There is often an intensification of prodromal symptoms with onset of jaundice. Some patients never develop jaundice.

Convalescent phase: Gradual improvement over a 3-16 week period. Most patients recover fully.

O. Hepatomegaly: Rarely marked - present in over half of cases. Liver tenderness is usually present. Splenomegaly is present in 15 percent of cases, and soft enlarged lymph nodes, especially in cervical or epitrochlear area, may occur. Signs of general toxemia vary from minimal to severe.

Lab findings: W.B.C. is normal to low (abnormal or "atypical" lymphocytes may suggest mononucleosis; mono spot test may be positive). Mild proteinuria is common and bilirubinuria often precedes jaundice.

A. Hepatitis. Differential diagnosis: Infectious mononucleosis, cytomegalic inclusion, leptospirosis, secondary syphilis, Q fever, and drug-induced liver disease. Distinguish prodromal phase from influenza, URI, and prodromal stages of the exanthematous diseases. In obstructive phase, rule out other obstructive lesions such as choledocholithiasis.

P. Symptomatic treatment: Bed rest at patient's option, forced fluids (or IV 10% dextrose if nausea and vomiting are significant problems), avoid morphine sulfate, drugs that have to be broken down by the liver, and hepatotoxic agents. Steroids have no value in hepatitis treatment. Patients should avoid strenuous exercise and alcohol. Strict isolation is not necessary, but handwashing after bowel movements is required. Thorough handwashing after handling contaminated utensils, bedding, or clothing is essential. Disinfection of feces is necessary when waterborne sewage disposal is not available. Give 5 cc. of gamma globulin (GG) to all close contacts of infected patients.

2-54. INFECTIOUS MONONUCLEOSIS. An acute infectious disease due to EB herpes virus. Universal in distribution and may occur at any age but usually occurs between ages of 10-35, either in epidemic form or sporadic cases. Probably transmitted by respiratory droplets. Incubation period is probably 5-15 days.

S. Symptoms are varied in type and severity. Fever, sore throat, and toxic symptoms (malaise, anorexia, and myalgia) occur frequently in early phase of the illness. A macular to maculopapular or occasionally petechial rash occurs in less than 50 percent of cases. Exudative pharyngitis, tonsillitis, or gingivitis may occur. Common manifestations are easy fatigability, nausea, jaundice (from hepatic involvement), headache, neck stiffness, photophobia, neuritis, and occasionally even Guillain-Barre syndrome (see Chapter 1, Section VII, Nervous System) (from C.N.S. involvement), chest pains, dyspnea, and cough (from pulmonary involvement).

O. Discrete, nonsuppurative, slightly painful, moderately enlarged lymph nodes especially those of the posterior cervical chain. Splenomegaly in 50 percent of cases. Hepatomegaly is common; and myocardial involvement with arrhythmias and tachycardia.

Lab Findings: Initially there is a granulocytopenia (decrease in number of neutrophils, basophils, and eosinophils) followed within 1 week by a lymphocytic leukocytosis (increase in lymphocytes and total number of white cells). Many lymphocytes are atypical, i.e., larger than normal adult lymphocytes, stain more darkly, and frequently show vacuolization (look like small air bubbles) of the cytoplasm and nucleus. Mononucleosis spot test will be positive.

A. Mononucleosis. Differential diagnosis: Hepatitis, streptococcal tonsillitis, diphtheria, rubella, toxoplasmosis, and, with C.N.S. involvement, meningitis.

P. Symptomatic treatment: Patient requires support and reassurance because of frequent feeling of lassitude and duration of

symptoms. If diagnosis is well established, a short course of corticosteroids can give symptomatic relief to severely ill patients. In uncomplicated cases, the fever disappears in 10 days and the lymphadenopathy and splenomegaly in 4 weeks. In some cases the illness may linger for 2-3 months, especially the lassitude and easy fatigability.

Section V - Rickettsial and Spirochetal

2-55. RICKETTSIA. Are between viruses and bacteria in size and are usually transmitted by arthropods (lice, fleas, ticks, mites), which serve as vectors.

a. Epidemic louse-borne typhus. Due to infection with Rickettsia prowazekii, a parasite of the body louse that ultimately kills the louse. Transmission occurs when a louse sucks blood from an infected individual; the louse then sucks blood from another individual and defecates at the same time; then the individual in scratching the bite rubs the infected feces into the bite wound. Dry, infectious louse feces may also be inhaled and result in human infection.

An individual who recovers from clinical or subclinical typhus may carry R. prowazekii in his lymphoid tissue for many years and even have a recurrence of typhus without exposure to lice or the infectious agent. During such a recurrence, he can serve as a source of infection for lice.

S. Prodromal malaise, cough, headache, and chest pains after 10-14 day incubation period, followed by an abrupt onset of chills, high fever, and prostration, with influenza-like symptoms, progressing to delirium and stupor. The fever is unremitting for many days, and the headache is intractably severe.

O. Conjunctivitis, flushed face, rales at lung bases, and often splenomegaly, a macular rash (that soon becomes papular) appears first in the axillas and spreads over the trunk and then the extremities. Rarely involves the face, palms, or soles. The rash becomes hemorrhagic and hypotension becomes marked in severely ill patients. There may be renal insufficiency, stupor, and delirium. Improvement begins in 13-16 days after onset with rapid drop of fever in spontaneous recovery.

Lab findings: W.B.C. is variable. Proteinuria and hematuria occur commonly.

A. Epidemic louse-borne typhus. Differential diagnosis: Murine typhus.

P. Tetracycline 250-500 mg. q.i.d. x 10 days or Vibramycin 200 mg. the first day followed by 100 mg. a day x 10 days. Alternate is chloramphenicol. Prevention consists of louse control with insecticides, particularly clothing and bedding, and frequent bathing. Immunization provides good protection against the severe disease but does not prevent infection or mild disease.

b. Endemic flea-borne typhus (murine typhus). Caused by Rickettsia typhi (R. mooseri), a parasite of rats. Transmitted to humans by bite from an infected flea that releases infected feces while sucking blood.

S. and O. Flea typhus resembles recurrent epidemic (Brill's disease) in that it has a gradual onset, fever and rash are of shorter

duration (6-13 days), and the symptoms are less severe. The rash is maculopapular mainly on the chest and fades fairly rapidly. Even without antibiotics it is a mild disease.

A. Murine typhus. Differential diagnosis: Recurrent epidemic typhus.

P. Antibiotic therapy (same as for epidemic louse-borne typhus).

Prevention: Control fleas and rats. Apply insecticides to rat runs, nests, and colonies and then poison or trap the rats.

c. Rocky Mountain spotted fever (Queenland tick typhus in Australia, Boutonneuse fever in Africa). All are caused by related Rickettsia. Rickettsii organisms through the bite of infected hard ticks. Rickettsia are often transmitted from one generation of ticks to the next without passage through an intermediate host.

S. The patient develops anorexia, malaise, nausea, headache, and sore throat 3-10 days after an infectious tick bite, progressing with chills; fever; aches in bones, joints, and muscles; nausea and vomiting; restlessness; insomnia and irritability. Delirium, lethargy, stupor, and coma may appear.

O. Face is flushed and conjunctivas injected. After 2-6 days of fever, a rash appears starting on the wrists and ankles spreading to the arms, legs, and trunk. The rash is initially small, red, and macular; over 2-3 days it becomes larger and petechial. Hepatomegaly, splenomegaly, jaundice, gangrene, myocarditis, or uremia may occur.

Lab findings: Leukocytosis, proteinuria, and hematuria are common.

A. Rocky Mountain spotted fever. Differential diagnosis: Measles, typhoid, or meningococcemia. Many other infections have similar early signs and symptoms.

P. Response to tetracycline or chloramphenicol is prompt if started early.

Prevention: Protective clothing, insect repellent, and buddy system checking for ticks at frequent intervals help.

d. Scrub typhus (Tsutsugamushi disease). Caused by Rickettsia Tsutsugamushi, a parasite of rodents that is transmitted by the bite of mite larva. The mite larva spends most of its life cycle on vegetation, and when an animal or human brushes against the vegetation, the larva drops onto them.

S. Incubation period of 1-3 weeks after bite by mite larva. Malaise, chills, severe headache, and backache. A papule develops at the site of the mite bite that vesicates and forms a flat black eschar.

O. Regional draining lymph nodes are enlarged and tender. There may be generalized adenopathy. Gradually rising fever with a generalized macular rash developing at the end of first week and is most marked on the trunk. During the second week of fever, pneumonitis, encephalitis, myocarditis, and cardiac failure may occur. The patient appears confused,

out of contact with the environment, and dulled in sensitivity.

A. Scrub typhus. Differential diagnosis: Leptospirosis, typhoid, dengue, malaria, and other rickettsial infections.

P. A tetracycline or chloramphenicol.

Prevention: Repeated area application of long-acting miticide and/or insect repellents on clothing or skin.

e. Rickettsialpox. Caused by Rickettsia akari, a parasite of mice, and transmitted by mites. The disease is fairly mild and self-limited.

S. and O. Incubation of 7-12 days with sudden onset of chills, fever, headache, photophobia, and disseminated aches and pains. Primary lesion at bite site is a red papule that vesicates and forms a black eschar. A widespread papular eruption appears 2-4 days after the onset of symptoms, becomes vesicular, and forms crusts that are shed in about 10 days.

A. Rickettsialpox. Differential diagnosis: Chickenpox or smallpox.

P. A tetracycline or chloramphenicol.

Prevention: Apply insecticide to mice runs and nests, then eliminate the mice.

f. Trench fever. A self-limited louse-borne relapsing febrile disease caused by Rickettsia quintana. Humans appear to be only animal reservoir. Occurs in epidemic form in louse-infested troops and civilians during wars and in endemic form in Central America.

S. Abrupt onset of fever lasting 3-5 days, often followed by relapses. Weakness; severe pain behind the eyes and in the back and legs.

O. Lymphadenopathy, splenomegaly, and a transient maculopapular rash may appear.

A. Trench fever. Differential diagnosis: Dengue, leptospirosis, malaria, relapsing fever, and typhus.

P. A tetracycline or chloramphenicol. The illness is self-limiting and recovery regularly occurs without treatment.

g. Q fever. Caused by Coxiella burneti, a parasite of cattle, sheep, and goats. Transmitted to humans by inhalation of contaminated dust or droplets or by ingestion of infected milk. It is excreted by cattle, goats, and sheep through feces, milk, and placenta. Coxiella is relatively resistant to pasteurization in milk. Spread from human to human is rare, but fetal infection can occur.

S. Incubation of 1-3 weeks with developing headache, prostration, muscle pains, and occasionally with a nonproductive cough, abdominal pains, or jaundice.

O. Physical signs of pneumonitis are slight. Hepatitis may be severe and endocarditis occurs rarely. Occasionally signs of

encephalopathy are present. The clinical course may be acute, chronic, or relapsing.

Lab findings: Leukopenia is often present.

A. Q fever. Differential diagnosis: Atypical pneumonia, hepatitis, brucellosis, tuberculosis, psittacosis, and other animal-borne diseases must be considered.

P. Tetracyclines can suppress symptoms and shorten the clinical course, but do not always eradicate the infection. Even in untreated cases, the mortality rate is negligible.

Prevention: Based on detection of infection in livestock, treatment and reduction in contact with the animal and dust contaminated by them, and effective pasteurization of milk.

2-56. SPIROCHETAL.

a. Syphilis. See Chapter 2, Section VI, Venereal.

b. Yaws (Frambesia, pian, bouba, parangi, domaria). An acute and chronic relapsing, contagious, nonvenereal, spirochetal disease caused by Treponema pertenue, which is morphologically indistinguishable from Treponena pallidum. Restricted to the tropical zones; the highest incidence is among native populations whose level of personal hygiene is low. It is predominately a disease of childhood, but transmission from child to mother by contact is frequent.

S. and O. Incubation period of 2-8 weeks. Initial lesion (mother yaw) appears at the site of implantation. It resembles the typical granulomatous secondary lesion, except it is often larger and healing takes longer. It is frequently still present when the secondary eruption appears. There is aching of the limbs, joint pains, and often an irregular fever is present. There may be enlargement of the regional lymph nodes. A few weeks to 4 months later the secondary or generalized stage begins with the appearance of secondary lesions scattered over the surface of the body. These lesions may involve the palms of the hands and/or the soles of the feet. The lesions are usually elevated, apparently granulomatous papules varying from a few to 50 mm. or more in diameter and tend to be round or oval. Initially the surface is composed of greatly proliferated epithelium exuding clear serum that contains concentrations of spirochettes. Later, a yellow crust forms (may be discolored by debris). In young children suffering from anemia or malnutrition, the lesions may appear as erosions with bright pink borders and whitish centers. Successive eruptions often appear before the preceding ones heal. These later lesions tend to be most numerous around the lips, axillae, genitalia, and anus. These recurring eruptions may continue for 2-3 years and lesions about the lips or on the soles of the feet may recur after many years. Healing of the secondary lesions leave only slight scarring that is never permanently atrophic and pigmented.

Nondestructive lesions of the bones are frequent in the secondary stage. They develop rapidly and resolve spontaneously in a few weeks or months, but the periosteal reaction may cause thickening of the bone resulting in deformities.

The tertiary stage of yaws usually does not appear until after a

relatively or completely symptom-free period of several years. Most commonly it begins during the third or fourth decades of life. In this stage, resolution and spontaneous cure may occur, or the disease become latent, with the subsequent appearance of relapsing tertiary lesions. The tertiary lesions are of three types: (1) extensive, spreading, superficial, and relatively clean ulcerations that gradually heal from the center; (2) cutaneous and subcutaneous nodules that break down forming deep, indolent ulcers with irregular bases (these heal from the margin and isolated islands in the base, causing atrophic scars that may be unpigmented in the early stages but later are often deeply pigmented and may cause severe contractures); (3) hyperkeratotic lesions of the soles of the feet and less commonly of the palms of the hands ("Crab Yaws") causing extensive thickening of the skin with fissures and ulcerations (painful and a source of severe disability).

Destructive bone and periosteal lesions most commonly involving the tibia, other long bones, and the hands are frequent. These are usually single or few in number and develop slowly. They may extend through the subcutaneous tissue and skin, producing chronic ulceration that responds slowly to treatment. The lesions are accompanied by local swelling, tenderness, and pain. These lesions can also occur on the skull, clavicles, scapulae, sternum, hard palate (can cause extensive destruction of the structure of the nose), and joints.

Lab findings: Spirochetes can usually be found by Giemsa's stain of exudates from lesions under darkfield examination. (India ink stain of slide also works.) Serum test for syphilis is positive.

A. Yaws. Differential diagnosis: The mucocutaneous lesions of leishmaniasis, the ulcerating lesions of leprosy, tuberculosis, and the late lesions of syphilis.

P. Treatment for the various stages of yaws is the same as for the various stages of syphilis (see Chapter 2, Section VI, Venereal).

c. Endemic syphilis. An infectious, chronic, nonvenereal infection of the intermediate tropical and temperate climates caused by Treponema pallidum (?), morphologically indistinguishable from the spirochetes of syphilis or yaws. Some authorities think that syphilis and endemic syphilis are the same disease. It occurs in localized areas in backward regions where socioeconomic levels are low and advanced education is lacking. When modern civilization reaches endemic areas through the construction of highways or development of an oil field, endemic syphilis disappears and venereal syphilis appears. It is primarily an early childhood disease and is spread by direct contact.

S. and O. Primary lesions consist of eruptions of the skin or mucous membranes, but are seldom recognized. Eruptions in the mouth are usually first, followed by moist papules in the folds of the skin. These lesions often resemble those of secondary syphilis. The late stage may appear within a few years after onset or be delayed for many years. It is characterized by plantar and palmar lesions, patchy pigmentation of the skin, and destructive lesions of the long bones, nose, and throat. Cardiovascular lesions are fairly common but involvement of the eyes, central nervous system, tabes, and paresis is rare.

Lab finding: Spirochetes may be found in wound aspirates using dark-field examination and serum test for syphilis is positive.

A. Endemic syphilis.

P. Same as yaws and syphilis (see Chapter 2, Section VI, Venereal).

d. Pinta (Mal del pinto, carate, azul, tina, lota, empeines). An acute and chronic nonvenereal disease caused by a spirochete (Treponema carateum) that is also morphologically indistinguishable from T. pallidum. Found in Central and South America, Mexico, and Cuba. Most frequent in the young and occurs most frequently in low lying and wooded areas, usually near rivers, where relative humidity is 80 percent or more and temperature is between 79 to 86o F. These people's primitive way of life and wearing of few clothes appear to promote their contacting pinta.

S. and O. Characterized by a superficial nonulcerative primary lesion, a secondary eruption, and late depigmentation and hyperkeratosis of the skin. The hands and wrists are most frequently involved, but feet and ankle involvement is common. Neurologic and cardiovascular involvement is fully as significant in late pinta as in syphilis.

Lab findings: Positive darkfield examination and STS.

A. Pinta. Differential diagnosis: Yaws, syphilis.

P. Same as for syphilis (see Chapter 2, Section VI, Venereal).

e. Relapsing fever (tick fever, famine fever, spirillum fever, febris recurrens, kimputu, garapata disease, and many others). Caused by the Borrelia species of spirochete and transmitted by tick bite or by crushed lice through abraded skin. Louse-borne relapsing fever has disappeared from the US but occurs in parts of South America, Europe, Asia, Africa, and Australia. Tick-borne relapsing fever is found in western US and Canada, Mexico, Central and South America, Europe, Africa, and Asia. Louse-borne relapsing fever is frequently found concomitantly with epidemic louse-borne typhus. Incubation period is from 2-10 days, but may be as long as 3 weeks.

S. Abrupt onset of fever (up to 104-105o F. or higher), chills, vertigo, severe headache, nausea, and vomiting. Transitory erythematous or petechial eruptions are common during the initial fever. Usually most pronounced about the neck and shoulder girdle and later extending to the chest and abdomen. Initial fever usually lasts 3-10 days. After an interval of 1-2 weeks, a relapse occurs, often somewhat milder. There may be 3-10 relapses before recovery.

O. Tachycardia occurs with the onset. Delirium occurs with high fever, and there may be various neurologic and psychic abnormalities. A slight icteric tint of the sclerae is common and marked jaundice may occur in severe cases. Hepatomegaly and splenomegaly may develop.

Lab findings: During episodes of fever, large spirochetes are seen in blood smears stained using Wright's or Giemsa's stain. Mild anemia and thrombocytopenia are common, but W.B.C. is usually normal.

A. Relapsing fever. Differential diagnosis: Malaria, leptospirosis, meningococcemia, yellow fever, typhus, or rat-bite fever.

P. Give 0.5 gm tetracycline or erythromycin in a single dose orally; 600,000 units of procaine penicillin G IM can also be used.

f. Rat-bite fever (sodoku). Uncommon acute infectious disease caused by a spirochete (Spirillum minus) that is transmitted by the bite of a rat.

S. The original rat bite heals rapidly unless secondarily infected. After an incubation period of one to several weeks, the bite site becomes swollen, indurated, painful, assumes a dusky purplish hue, and may ulcerate. Fever, chills, malaise, myalgia, arthralgia, and headache are present. After a few days, the local and systemic symptoms subside only to reappear in 24-48 hours. After the first few relapses, only the fever returns on this 24-48-hour cycle and may persist for weeks.

O. Regional lymphangitis and lymphadenitis are present. Splenomegaly may occur. A sparse, dusky-red maculopapular rash may appear on the trunk and extremities.

Lab findings: Spirochete may be found in aspirated lymph node material or in the ulcer exudate under darkfield examination. Leukocytosis is often present and STS is often falsely positive.

A. Rat-bite fever. Differential diagnosis: Streptococcal rash, tularemia, relapsing fever.

P. Give 300,000 units procaine penicillin IM q.12h. x 7 days.

g. Leptospirosis (Fort Bragg fever, Weil's disease, swineherd's disease). An acute and often severe infection caused by several Leptospira species. Leptospirosis is found worldwide. It is transmitted by ingestion of food or drink contaminated by rodents, cattle, or pigs. The disease can also be acquired by direct contact through minor skin lesions, and probably via the conjunctiva, and also through bathing in contaminated water. Incubation period is 2-20 days.

S. Sudden onset of fever (102-104o F.), chills, abdominal pains, vomiting, nausea, myalgia (especially of the calf muscles), and unrelenting frontal headache. Photophobia, sore throat, cough, and diarrhea are common. Petechial and maculopapular rashes may occur. Usually all signs and symptoms disappear within 3-4 days, but some patients may be ill for weeks. In some cases symptoms disappear for 1-3 days, then the fever and any of the initial symptoms may return.

O. Conjunctiva is markedly reddened. The liver can be palpated in 50 percent of the cases and jaundice is present about the fifth day. Capillary hemorrhages and purpuric skin lesions may appear. Meningeal irritation and associated findings of aseptic meningitis may occur.

Lab findings: W.B.C. may be normal or as high as 50,000 with neutrophilia. Urine may contain bile, protein, casts, and red cells. Spirochete may be found in urine from the tenth day to the sixth week. It can also be found in blood smears using dark-field examination during the first 10 days.

A. Leptospirosis. Differential diagnosis: Hepatitis, yellow fever, relapsing fever.

P. Give 600,000 units procaine penicillin IM q.3h. x 24h. then

q.6h. x 6 days, or 500 mg. tetracycline q.6h. x 7 days.

2-57. Venereal diseases are contagious diseases most commonly acquired through sexual intercourse or other genital contact.

2-58. GONOCOCCAL INFECTIONS (clap, dose). A specific infection of the genitourinary tract caused by Neisseria gonorrhoeae. Extragenital infections (rectal, oral, skin, and eye infection of the newborn) do occur, but not as frequently.

S. In the male, incubation 2-7 days after contact; average is 3 days. A transient mucoid urethral discharge develops that becomes a profuse, thick, greenish, purulent urethral excretion. Painful urination is the outstanding symptom. Both the discharge and the painful urination may be severe, moderate, or even absent. About 10 percent of all cases have no S or S. Rectal infections are most often asymptomatic and the result of direct implantation of infection almost always by homosexual activity. The most common complication of untreated gonorrhea is urethral strictures; others include inguinal lymphadenitis, seminal vesiculitis, epididymitis, or prostatitis.

In the female, 80-90 percent are asymptomatic, but can continue to spread the infection. In the female, dysuria or vaginal discharge is the most frequent S or S, but may be so mild as to be unnoticed. Rectal infection can be caused by contamination from cervical discharge or rectal intercourse. Complications in the female are local spread of gonorrhea causing an inflammation of the vulvovaginal gland and/or fallopian tube. This spread may continue from the fallopian tubes into the peritoneal cavity.

In both male and female, but usually female, the infection may spread through the blood and may present in varied ways depending on the area or organs the infection attacks. The most common are arthritis, skin eruptions, meningitis, endocarditis, or conjunctivitis (via blood or by contamination from genital secretion).

O. Typical intracellular gram-negative diplococci are found in the smear of the urethral discharge or cultured from any site, particularly the urethra, cervix, or rectum. It is possible to gram stain smears from urethra, cervix, or rectum and find the organism, but a negative finding does not rule out gonorrhea. History and S and S can make the diagnosis.

A. Gonorrhea. Differential diagnosis: Nonspecific urethritis (50 percent caused by chlamydiae), trichomonal and candidal vaginitis, and cervicitis. The many agents causing salpingitis, pelvic peritonitis, arthritis, proctitis, and skin lesions must be considered also.

P. Uncomplicated gonorrhea: 1 gm probenecid orally; 4.8 million units aqueous procaine penicillin G IM in 2 or more sites.

Alternative: Give 3.5 gm ampicillin together with 1 gm probenecid orally at one time. NEVER TREAT GONORRHEA WITH BENZATHINE PENICILLIN G. If allergic to penicillin, give 1.5 gm tetracycline orally stat., then 0.5 gm orally q.i.d. x 4 days or spectinomycin 2 gm IM at one time. Watch for penicillin-resistant gonorrhea. Do a followup 7 days after completion of treatment. Treat complications with spectinomycin 2 gm IM. If after followup gonorrhea is still present, think of reinfection and give spectinomycin 2 gm IM again. If spectinomycin resistant, give

cefoxitin 2 gm IM with 1 gm probenecid P.O. Alternates are tetracycline or erythromycin 0.5 gm orally q.i.d. x 10 days.

2-59. SYPHILIS. Causative agent is Treponema pallidum, a spirochete capable of infecting any organ or tissue in the body. Transmission occurs most frequently during sexual contact, but may be extragenital. The clinical course of untreated syphilis is divided into 4 stages: primary (early), secondary, latent (hidden), and tertiary (late) syphilis. The lesions associated with primary and secondary syphilis are self-limiting and resolve with few or no residual. Tertiary syphilis may be very destructive and permanently disabling and may lead to death. In general, if untreated, one-third of the people infected will undergo spontaneous cure, one-third will remain in latent stage for life, and one-third will develop serious late (tertiary) lesions.

Syphilis can be clinically cured in all of the stages, but the killing of the treponemes can cause Jarisch-Herxheimer reaction. This reaction is thought to be caused by the rapid release of antigenic materials from lysed treponemes. There may be a local and general reaction. The local reaction consists of intensification of the lesions (rashes become more pronounced, chancre becomes edematous). Systemically, frequently the temp rises to 101-102° F., occasionally as high as 104° F. Some patients have convulsions or increasing agitation requiring restraints or sedatives. Reaction usually occurs within 12 hours of treatment and usually lasts only a few hours, rarely more than 24 hours. This reaction is usually benign and of itself is not reason to discontinue treatment.

a. Primary syphilis.

S. A 10-90-day incubation period, then a primary chancre develops. This is a painless superficial ulcer with a clean base and firm indurated margins. Chancres are usually singular, but multiple lesions are not rare. Bacterial secondary infection may occur causing pain. Most frequently located on the penis, labia, cervix, or anorectal region. Occasionally found on lip, tongue, or tonsil and rarely on breast or finger. Press the edges of the primary lesion an you will feel a round pealike ball. The lesion will heal by itself, but may cause a scar. The primary chancre may pass unrecognized.

O. Enlarged regional lymph nodes that are rubbery, discrete, and nontender. Smear from lesion stains the spirochete pink using Giemsa's stain and black using silver impregnation method under dark-field illumination. The spirochete is somewhat hard to find and may require numerous smears before it is found. A serologic test for syphilis (STS) is the best test. These tests usually turn positive 1-3 weeks after the appearance of the primary lesion. If the initial STS and dark-field examination are negative, the STS should be repeated once weekly for 4 weeks.

A. Primary syphilis. Differential diagnosis: chancroid, genital herpes, lymphogranuloma venereum, or neoplasm.

P. Benzathine penicillin G 1.2 million units in each buttock for a total of 2.4 million units once. Only if patient is allergic to penicillin should tetracycline or erythromycin be used. Tetracycline 500 mg. orally q.i.d. x 15 days. Erythromycin 500 mg. orally q.i.d. x 20 days.

b. Secondary syphilis.

S. Generally appears a few weeks to 6 months after primary chancre. The most common manifestations are skin and mucosal lesions. The skin lesions are usually bilaterally symmetrical and are nonpruritic, macular, papular, pustular, or folliçular (or any combination of these). Lesions are usually generalized but often involve the palms of the hands and the soles of the feet. The mucosal lesions range from ulcers and papules of the lips, mouth, throat, genitalia, and anus (mucous patches) to a diffuse redness of the pharynx. Mucous membrane and skin lesions are highly infectious during this stage. Meningeal, hepatic, renal, bone and joint invasion with resulting cranial nerve palsies, jaundice, nephrotic syndrome, and periostitis may occur. The lesions of secondary syphilis will heal spontaneously, but may relapse if undiagnosed or inadequately treated. These relapses may include any of the findings of secondary syphilis, but unlike the usually asymptomatic neurologic involvement of secondary syphilis, neurologic relapses may be fulminating, leading to death.

O. STS is positive in almost all cases. Skin and mucous membrane lesions often will show the T. pallidum spirochete on dark-field exam.

A. Secondary syphilis. Differential diagnosis: Infectious exanthems, pityriasis rosea, and drug eruptions. Visceral lesions may suggest nephritis or hepatitis from other causes. Red throat may mimic other forms of pharyngitis.

P. Same treatment as primary syphilis.

c. Latent syphilis (lasts from months to lifetime).

S. No physical signs; total diagnosis is on history.

O. Positive STS.

A. Latent syphilis.

P. Give 2.4 million units benzathine penicillin G IM once a week x 3 weeks.

d. Tertiary (late) syphilis may occur anytime after secondary syphilis, even after years of latency.

S. Essentially a vascular disease that may attack any tissue or organ. Signs and symptoms may mimic almost any disease. Called the "Great Imitator" because of this. A good in-depth history is required, looking for history of primary chancre and secondary syphilis untreated or inadaquately treated.

O. STS usually positive; T. pallidum might possibly be found in skin or mucous lesions.

A. Tertiary syphilis.

P. Same as latent syphilis, but there is no known method for reliable eradication of the treponeme from humans in the late stages of syphilis. There is also no confirmed cases where the treponeme left after treatment are capable of causing progressive disease.

e. Congenital syphilis transmitted through the placenta to the fetus.

S. May have minimal to no signs for 6-8 weeks after birth. Most common findings are on skin and mucous membranes - serous nasal discharge, mucous membrane patches, maculopapular rash, and/or condylomas (broad flat wartlike growths usually seen on genitals or near anus). These lesions are infectious. Lesions heal by themselves and if left untreated child develops defects: interstitial keratitis, Hutchinson's teeth, saddle nose, saber skins, deafness, and/or C.N.S. involvement.

O. Smears taken from lesion and checked under dark field show T. pallidum. STS is not conclusive as it is complicated by transplacental acquisition of maternal antibodies. Baby must be checked every 2-3 weeks for 4 months.

A. Congenital syphilis.

P. Aqueous penicillin G 50,000 units/kg. IM or IV in 2 divided doses daily x 10 days. Antibiotics other than Pen are not recommended.

2-60. CHANCROID. An acute localized usually self-limiting venereal disease with an incubation period of 3-5 days.

S. Initial lesion is vesicopustular with a necrotic base, surrounding erythema, and undermined edges. Multiple lesions started by autoinoculation and inguinal adenitis often develop. The adenitis is usually unilateral and consists of tender matted nodes of moderate size with overlying erythema. The nodal mass softens, becomes fluctuant, and may rupture spontaneously. With lymph node involvement, chills, fever, and malaise may develop; balanitis (inflammation of glans penis) and phimosis (tightening of the foreskin) are frequent complications. These signs usually occur in men; women frequently have no external signs.

O. Smear from lesion gram-stained shows short gram-negative bacillus (Hemophilus ducreyi). There is a skin test for chancroid; once it becomes positive, like tine test, it remains positive for life.

A. Chancroid. Differential diagnosis: Other veneral diseases and pyogenic lesions.

P. Gantrisin 500 mg. q.i.d. x 10-14 days; 0.5 gm tetracycline q.i.d. x 10-14 days; clean ulcer with soap and water b.i.d.; aspirate fluctuant buboes.

2-61. GRANULOMA INGUINALE. A chronic, relapsing granulomatous anogenital infection with an incubation period of from 1-12 weeks.

S. The initial lesion may be a vesicle, papule, or nodule usually on the penis or labia minora. The onset is insidious. This lesion becomes eroded and superficially ulcerated. The ulcer is shallow, sharply demarcated with a beefy-red friable base of granulation tissue with new nodule formation at the edge as the lesion extends. The advancing border has a characteristic rolled edge of granulation tissue. Large ulcerations may advance up onto the lower adbomen and thighs. Scar formation and healing may occur along one border while the other advances. The process may become indolent and stationary.

O. Gram-negative rod-shaped microorganisms found in mononuclear

phagocytes from smears made from tissue scraping or secretions from the ulcers.

A. Granuloma inguinale.

P. Tetracycline 500 mg. q.i.d. x 2 weeks or streptomycin 1 gm q.i.d. x 7 days IM or ampicillin 500 mg. q.i.d. x 2 weeks.

2-62. LYMPHOGRANULOMA VENEREUM. An acute and chronic sexually transmitted disease with a 5-21 day incubation period.

S. The primary lesion that is seldom seen is a transitory small papule, vesicle, or ulcer that vanishes in a week to 10 days. In the male, it is usually found on the penis and in the female, on the vaginal wall or cervix. From there, invasion of the lymphatics occur. In the male, the inguinal nodes are involved with further extension into the deep iliac nodes. At first the nodes are discrete, later becoming enlarged, matted, adherent to the skin and finally fluid filled. The overlying skin becomes discolored and ultimately sinus formation with drainage occurs, which may continue for months. Healing is accompanied by extensive scarring, which may lead to elephantiasis of the genitals and rectal strictures. In the female, inguinal involvement is rare. It usually affects the rectovaginal septum, often with no localizing symptoms, until sinuses open and drain into the rectum, and blood and pus appears in the stool; this may be accompanied by malaise, anorexia, headache, and fever. This may last for many weeks. Later, chronic proctitis occurs and occasionally rectovaginal fistulas and perirectal abscesses. Extensive scarring often leads to rectal strictures and elephantiasis of the genitals.

O. Causative organism is a large virus and requires special tests for antibodies; tests are not totally reliable.

A. Lymphogranuloma venereum. Differential diagnosis: Early lesions; syphilis, genital herpes, and chancroid. Lymph node involvement; tularemia, tuberculosis, plague, neoplasm, or pyogenic infection. Rectal strictures; neoplasm, and ulcerative colitis.

P. Tetracycline 500 mg. q.i.d. x 2-3 weeks, gentamicin 40 mg. IM b.i.d. x 2 weeks, bed rest, warm compresses for buboes, and analgesics p.r.n.; aspirate fluid-filled nodes.

2-63. HERPES GENITALIS. Caused by herpes virus type 2 (herpes simplex). Can be sexually transmitted and is increasing in frequency and seriousness. Infection during pregnancy can cause spontaneous abortion, stillbirth, and neonatal death.

S. A 4-7 day incubation period. Starts with reddened area with itching; progresses into blister that breaks and becomes painful like a burn. All of this is usually recurrent. In severe cases there may be fever, malaise, anorexia, local genital pain, dysuria, leukorrhea (white or yellowish mucous discharge), and even vaginal bleeding.

O. Typical genital lesions are multiple shallow ulcerations, vesicles, and erythematous papules. Painful bilateral inguinal adenopathy is usually present. Scrapings and biopsies may show characteristic "ground glass" appearance of cellular nuclei with numerous small intranuclear vacuoles and small scattered basophilic particles.

A. Herpes genitalis. Differential diagnosis: Other venereal diseases.

P. Symptomatic treatment. There is no known cure but there is a control being tested that appears to be effective but only as long as taken. Amino acid (al-lysine) comes in tablet form; give 1,500 mg. daily in 2 doses. When lesions disappear, 1 tab a day as a maintenance dosage. A paste can be made by crushing a tablet, making into a paste, and applying directly onto the lesion. This usually clears the lesions within 24-48 hours; so far this appears to be very effective but only as a control, not a cure. This is the only venereal disease that does not as yet have a cure.

2-64. Other diseases that are considered venereal in nature include Pediculosis pubis (crabs), scabies, hepatitis B infections, vulvovaginal candidiasis, trichomoniasis, and nongonococcal urethritis. These diseases will be covered in other sections.

2-65. Treatment of venereal diseases by itself is not enough. Control and prevention must be stressed.

a. Prevention includes classes on VD and VD prevention measures plus insuring prophylactic devices are made available.

b. Control involves early detection and treatment of infected personnel and their contacts. Every patient diagnosed as having VD should be interviewed to determine with whom he has had sexual contact during the course of his illness and from whom he might have contracted the disease. If the patient does not want to give out the names and addresses of his contacts, you can establish and use a card system. With this system you have colored 3 x 5 cards, a different color for each type VD You can hand out a number of cards to the patient and tell him to give one card to each person with whom he had sex. Have him tell them to take the card to the medic. In that way you can examine and treat prophylactically each person who brings in a card and give them cards for their sexual contacts. In this way you should be able to eliminate the majority of the VD problem.