Helminths, Bedbugs, Scabies, and Lice Infections

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KEY POINTS

- Immigration and travel continue to increase the parasitic diseases seen in emergency departments.
- Consider parasitic infections in patients presenting with abdominal pain, diarrhea, unexplained fever, rash, or eosinophilia.
- History of illness, travel, occupation, recreation, and behavior is paramount in diagnosing parasitic disease.

HELMINTHS

EPIDEMIOLOGY

Helmint infections are estimated to affect 1 billion people in developing countries, namely, sub-Saharan Africa, Latin America, and Asia. Most commonly seen in resource-poor areas with poor sanitation, helminthiases are increasingly recognized in refugees, immigrants, rural populations, and international travelers.

PERSPECTIVE

Parasite morbidity includes anemia, growth stunting, undernutrition, disfigurement, reduced work capacity, and increased susceptibility to other infections. Complications can include elephantiasis, blindness, gastroenterologic and urinary obstruction, and bladder cancer. Despite the widespread distribution of helminthic disease, research funding comprises less than 1% of global dollars spent.

CLASSIFICATION

Two major phyla are responsible for human disease: Nemathelminthes (roundworms) and Platyhelminthes (flatworms). Nematodes are categorized by their migration route. Enterobius and Trichuris are found intraintestinally, whereas filarial worms live in tissue and lymphatics. Platyhelminthes is further divided into two classes: cestodes, or tapeworms; and trematodes, or flukes.

Nemathelminthes (Roundworms)

Nematodes range in length from 1 mm to up to 50 cm. Intestinal nematodes include Ascaris, Necator, and Ancylostoma, which develop in soil, whereas Strongyloides and Enterobius can be directly transmitted from person to person. With the exception of Strongyloides, most helminthes do not undergo replication within the host. Nematodes causing human disease include Dracunculus and filarial worms such as Wucheria, Brugia, and Onchocerca.

INTESTINAL ROUNDWORMS: ASCARIS

EPIDEMIOLOGY

Ascaris lumbricoides infects approximately 25% of the world’s population. It is prevalent in warm countries and areas of poor sanitation and is endemic to the southern United States. Ova can remain infective for several years. They are sensitive to temperatures higher than 65°C or lower than 20°C, direct sunlight, and organic solvents. Ascaris lumbricoides reaches up to 40 cm and is characterized by a constricted area at the junction of the first and middle thirds. The golden brown ovoid eggs measure 60 by 40 mcm.

TRANSMISSION AND LIFE CYCLE

Ascaris lumbricoides is transmitted by ingesting mature eggs from contaminated soil or in conjunction with human feces used as fertilizer. Infection may cross into nonendemic areas through vegetable transport and tainted water. Children frequently contract the disease by playing in dirt. Fertilized eggs become infective in soil 3 to 4 weeks after excretion. When the eggs are swallowed, the larvae hatch in the duodenum, penetrate the intestinal wall, and migrate through the portal venous system to the liver. The larvae migrate through the right side of the heart, the lungs, and into the tracheobronchial tree. Over 2 weeks, the larvae are...
carried up the trachea to the larynx, where they move into the esophagus and are swallowed a second time, to reach the small intestine, where they mature. Female *Ascaris* worms lay eggs 2 to 3 months after ingestion and produce up to 240,000 eggs per day. The life span of *Ascaris* is 6 months to 1 year. *Ascaris* coexists with *Trichuris trichiura* in the United States, predominantly in the Appalachian Mountains.

**PRESENTING SIGNS AND SYMPTOMS**

Many patients are asymptomatic or have mild symptoms, and the infection ends after expulsion of the adult worms. Ectopic migration is possible, and it causes severe clinical manifestations. The pulmonary larval migration occurs 9 to 12 days after egg ingestion and may manifest with cough, bloody or mucoid sputum, pleurisy, and fever.

Eosinophilia develops during this pulmonary migration and may manifest as eosinophilic pneumonia, known as Loeffler syndrome. Chest radiographs may demonstrate small, rounded infiltrates that are transient and usually resolve after several weeks. Loeffler syndrome is seen mainly in ascariasis, but it also may occur in other parasitic infections such as hookworm infestation and strongyloidiasis. Allergic reaction can occur with reinfection. Two to 3 months after ingestion, the parasite matures in the small intestine and can cause abdominal pain, nausea, vomiting, anorexia, diarrhea, constipation, volvulus, or intussusception. Biliary obstruction and intestinal inflammation can manifest as an acute surgical abdomen, mimicking acute appendicitis or cholecystitis. Liver abscesses and pancreatitis are possible when the female worm migrates up the common bile duct. Protein malnutrition and intestinal obstruction are more likely to occur in heavily infected children rather than in adults.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

The diagnosis can be made from the passage of worms in the stool or by finding eggs in the feces. A single stool specimen sent for ova and parasite examination is usually sufficient. As the worm transverses the esophagus, it may be coughed out. In eosinophilic pneumonia, larvae may be isolated from sputum or gastric aspirate.

Gastrointestinal radiographic examination may visualize adult worms on contrast studies, ultrasound scans of the pancreatic-biliary system, plain abdominal films, and endoscopic retrograde cholangiopancreatography (ERCP). ERCP can be therapeutic in extracting worms. Serologic antibody tests include complement fixation, precipitin, agar gel diffusion, immunoelectrophoresis, and the radioallergosorbent test.

**INTESTINAL ROUNDWORMS: NECATOR AND ANCYLOSTOMA (HOOKWORMS)**

**EPIDEMIOLOGY**

Hookworm infestations of *Ancylostoma duodenale* prevail in southern Europe, northern Asia, and North Africa, whereas *Necator americanus* is the main species affecting the Western Hemisphere and equatorial Africa. Worldwide, 1 billion persons are believed to be infected with hookworms. The frequency of infection is a general indication of the local level of hygiene and sanitation. Disease burden from hookworms results in iron-deficiency anemia and hypoproteinemia.

**TRANSMISSION AND LIFE CYCLE**

Hookworm infection is contracted by penetration of the skin or oral mucosa by filariform larvae. Persons who walk barefoot on contaminated soil or who eat contaminated vegetables become infected. Transplacental infection and transmammary infection are also possible. Eggs are passed in the stools to the soil, where they hatch into rhabditiform larvae that develop into filariform larvae. The larvae penetrate the skin or mucosa directly and reach the alveoli through the circulation. The larvae then ascend the airway, enter the esophagus, are swallowed, and reach the small intestine, where they mature.

Adult worms use hooks to attach to the intestinal mucosa, and they suck blood directly from the host. The time frame from skin invasion to egg appearance is 6 to 8 weeks. *Ancylostoma* adults live on average for 6 to 8 years, and the life span of *Necator* ranges from 2 to 5 years, but both species of worms can survive for more than a decade.

**PRESENTING SIGNS AND SYMPTOMS**

Presentation includes rash, cough, low-grade fever, abdominal pain, diarrhea, generalized weakness, weight loss, heme-positive stools, and eosinophilia. Early larval invasion of the skin causes pruritus and erythematous maculopapular or vesicular dermatitis, known as “ground itch.” Sensitized hosts may have serpiginous tracts as larvae migrate through the subcutaneous tissue, similar to cutaneous larva migrans.

Transient pneumonitis can occur as larvae travel through the lungs. Epigastric pain, inflammatory diarrhea, and eosinophilia may develop with early intestinal invasion. Loss of plasma protein results from malabsorption and increased intestinal permeability and may cause anasarca.

Chronic hookworm infection leads to iron deficiency. In malnourished patients, infection can cause severe anemia and growth delay. Infection in pregnancy may lead to low fetal birth weights and birth defects. Pica and geophagy are observed in infected children.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Multiple stool specimens for ova and parasite studies or concentration techniques may be necessary to confirm the diagnosis. The parasite burden may be estimated using the Beaver stool or Kato 50 adult slide smear method. The polymerase chain reaction (PCR) technique can identify a hookworm from a single egg.

Hookworm infection may be confused with pneumonia, anemia, and malnutrition from other causes. Hypochromic microcytic anemia, coupled with eosinophilia or hypoproteinemia, indicates a heavy infection.
TREATMENT

Oral iron supplementation is usually sufficient to correct mild anemia, although blood transfusion may be needed to correct more severe anemia. Vaccine development is currently under way, based on canine and sheep models.\(^3\)

INTESTINAL ROUNDWORMS: STRONGYLOIDES

EPIDEMIOLGY

*Strongyloides stercoralis* is found in the tropics, subtropics, and in temperate areas, and it affects an estimated 55 million to 100 million persons.\(^4\) *Strongyloides* is endemic to Southeast Asia, Latin America, the West Indies, Bangladesh, Pakistan, Africa, Spain, and the Appalachian region of the United States. *S. stercoralis* is an unusual helminth in its ability to replicate within the human host, thus resulting in continuous autoinfection. The infection is often difficult to eradicate, especially in immunocompromised hosts (Box 177.1). The larvae can disseminate and can cause systematic disease and mortality.

TRANSMISSION AND LIFE CYCLE

In addition to replication within the human host, *Strongyloides* can survive in a free-living cycle in soil. The rhabditiform larvae can directly transform into infective filariform larvae or a free-living soil form. The filariform larvae penetrate skin or mucosa, travel to the alveoli, and ascend the airway. The larvae are then swallowed and reach the small intestine. The host has no adult male worms, and the 2-mm female worms reproduce by parthenogenesis, hatching eggs within the intestinal mucosa. Rhabditiform larvae migrate to the intestinal lumen and either pass into the feces or directly transform into filariform larvae and cause autologous reinfection.

Presenting Signs and Symptoms

Patients may be asymptomatic or have mild abdominal discomfort, cough, dyspnea, wheezing, and peripheral eosinophilia. Ten percent of patients present with wheezing as their primary complaint.\(^5\) If adult worms invade the duodenojejunal mucosa, the symptoms can mimic those of peptic ulcer disease, and small bowel obstruction may occur.

Fluctuating eosinophilia is a common finding. Strongyloidiasis may also manifest with urticaria, usually recurrent along the buttocks and wrists. These urticarial serpiginous eruptions are called larva currens, and they are pathognomonic findings.

DIAGNOSIS AND MEDICAL DECISION MAKING

Stool examinations need to be repeated 3 to 5 times. An immunosorbent assay for aspiration or biopsy of duodenjejunal contents is also available. Serum immunoglobulin G (IgG) antibodies can be detected with enzyme-linked immunosorbent assay (ELISA). In disseminated strongyloidiasis, sputum, bronchoalveolar lavage samples, and surgical drainage fluids should be examined for larvae. Eosinophilia greater than 5% or a finding of more than 400 eosinophils/microliter is consistent with *Strongyloides* infection.\(^5\)

Strongyloidiasis can mimic gastroenteritis, gastritis, colitis, irritable bowel syndrome, asthma, and pneumonia. It may also cause meningitis or sepsis.

TREATMENT

Strongyloidiasis must be treated to avoid hyperinfection. In patients coinfected with human T-cell lymphotropic virus-1 (HTLV-1), treatment failures are common. A regimen of ivermectin plus thiabendazole or ivermectin plus albendazole is recommended for patients with disseminated disease.

INTESTINAL ROUNDWORMS: ENTEROBIUS (PINWORMS)

EPIDEMIOLOGY

Enterobiasis is worldwide in distribution and is commonly found in young children. Pinworms are most common in developed countries with temperate or colder climates. The adult worms are 6 to 12 mm in length and 0.3 to 0.5 mm in diameter. The eggs are 50 by 25 mcm and are asymmetrically flattened on one side.

TRANSMISSION AND LIFE CYCLE

Eggs are ingested orally, hatch in the stomach, and pass through to the intestine, where they invade the glandular...
crypts. The adult worms live in the cecum and appendix and survive for approximately 2 months. Whether pinworms cause appendicitis is not known. The female pinworm migrates out through the rectum and onto the perianal skin to deposit her eggs. Rarely, the worms invade the abdominal cavity and cause threadworm granulomas of the liver, ovary, kidney, spleen, and lung. Transmission of pinworms occurs by direct anus-to-mouth spread from contact with an infected person. It can also be caused by airborne eggs that are shaken free from contaminated clothing or bed linens. Autoinfection is common because patients scratch the pruritic anal area and then bite their nails or put their fingers in their mouth. The whole cycle takes 2 to 4 weeks.

**PRESENTING SIGNS AND SYMPTOMS**

Pruritus ani is the main symptom and varies from mild itching to acute pain, generally worse at night. Pinworm disease is essentially an allergic reaction to the release of eggs and other secreted materials from the gravid female. Associated scratching and excoriation can cause secondary bacterial infection. Vulvitis can occur when pinworms enter the vulva and cause a mucoid discharge with pruritus vulvae. Insomnia, restlessness, loss of appetite, weight loss, irritability, and enuresis may be associated with pinworm infection.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

The eggs are rarely seen in the feces, but they are observed when the adult worms migrate to the anus or vulvar area, particularly at night. The “Scotch tape test” is done by pressing a piece of clear sticky tape against the perianal region and then mounting the tape on a slide. The eggs are identified with light microscopy. Patients usually have no eosinophilia or associated anemia.

**TREATMENT**

The entire family is treated simultaneously, to avoid reinfection. All bedding and contaminated clothing should be washed. Fingernails should be kept short. Frequent hand washing and bathing may reduce reinfection. Eradication of the parasite may necessitate repeated courses of treatment.

**TISSUE NEMATODES: TRICHINELLA**

**EPIDEMIOLOGY**

Trichinosis has a worldwide distribution. Eight species of *Trichinella* infect humans. *Trichinella spiralis* and *Trichinella pseudospiralis* are found worldwide. *Trichinella nativa* is found in the Arctic; *Trichinella nelsoni* occurs in eastern Africa; *Trichinella britovi* is found in Europe, Asia, and western Africa; and *Trichinella murrelli* occurs in North America. *Trichinella papuae* and *Trichinella zimbabwensis* have also been identified. Human infection results from ingestion of poorly cooked infected meat, particularly pork. Cattle, horse, dog, and wild game meat are also potential sources.

**TRANSMISSION AND LIFE CYCLE**

The encysted larvae are released from their capsule by digestive enzymes in the intestine. The larvae then penetrate the intestinal mucosa and mature into adults. After mating, the male worms die, and the female worms discharge larvae to the tissues. After travelling through the circulation, the larvae encyst in muscle and may calcify. Cysts can be seen in the diaphragm, masseter and intercostal muscles, musculature of the larynx, tongue, eye, and heart, and the brain.

The main methods of infection prevention are thorough cooking and regular meat inspection. *Trichinella* larvae in meat may be killed by heating to a temperature of 77° C or freezing to 15° C for 3 weeks. Both encysted and free *Trichinella* larvae remain viable for years.

**PRESENTING SIGNS AND SYMPTOMS**

The presenting symptoms depend on the level of infection and the location of the larvae. Two phases are recognized: intestinal and muscular. Enteric invasion may cause diarrhea, fever, nausea, vomiting, and abdominal pain during the first week of infection and is often confused with food poisoning. During the second week, the larvae migrate into the muscles and cause hypersensitivity reactions with fever, eosinophilia, rash, headache, cough, and myalgias.

Cardinal clinical findings of trichinellosis include cachexia, edema, splinter hemorrhages, dehydration, ongoing fever, and pruritus. Myocarditis, encephalitis, nephritis, congestive heart failure, pneumonitis, hemiplegia, severe pain, psychiatric disturbances, and epilepsy may develop and lead to death.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Eosinophilia is a hallmark of trichinellosis. No relationship exists between the level of eosinophilia and the clinical course of disease. Other laboratory manifestations of trichinosis include leukocytosis and elevated creatine phosphokinase, lactate dehydrogenase, and myokinase levels. The diagnosis is made by identification of larvae in blood or tissue. Serologic assays are available, including ELISA, indirect immunofluorescence, and latex agglutination. The circulating antibody can be detected by serologic tests between 2 and 4 weeks after infection. Immunofluorescence is positive 2 to 3 weeks after infection. If serologic test results are equivocal, larvae may be found in muscle by trichinoscopy. This procedure is performed during the muscular phase approximately 7 days from onset of symptoms with samples taken from the deltoid, biceps, gastrocnemius, or pectoralis major muscle.

Trichinosis resembles many conditions: typhoid, encephalitis, myositis, and tetanus. Trichinosis may also be confused with collagen disorders such as periarteritis nodosa and rheumatoid arthritis.
High-frequency ultrasound with Doppler imaging of the breast and scrotum may reveal dilated lymphatics containing moving adult worms, known as the filarial dance sign. The broad cross-reactivity of filarial antigens with other helminths often confounds the serologic test results. In addition, endemic populations may become sensitized through mosquito exposure without actual infection.

**TREATMENT**

Wolbachia bacterial endosymbionts are present in most filarial worms, with the exception of *L. loa*. When patients are treated with diethylcarbamazine, a hypersensitivity reaction to the intracellular Wolbachia may occur and may progress to encephalopathy. Diethylcarbamazine should not be used as treatment, except for *Loa loa*, because the hypersensitivity reaction can lead to hypotension or angioedema. Chemotherapy and vaccine development directed against Wolbachia are under investigation.

**TISSUE NEMATODES: FILARIA**

**EPIDEMIOLOGY**

Filarial nematodes are estimated to infect 170 million persons. Four main species cause most serious infections: *Wuchereria bancrofti*, *Brugia malayi*, *Onchocerca volvulus*, and *Loa loa*. *W. bancrofti* is found in Africa, South America, Asia, the Pacific islands, and the Caribbean. *Brugia* species are found mainly in Southeast Asia, Asia, and Indonesia.

**TRANSMISSION AND LIFE CYCLE**

Mosquitoes transmit infective *Wuchereria* and *Brugia* larvae during a blood meal. *Onchocerca* and *Loa loa* larvae are carried by the black and tabanid flies, respectively. The larvae mature into adults that remain alive in the lymphatics or subcutaneous tissues for years. Microfilariae are produced by the adult worms, which are ingested by mosquitoes and develop into infective larvae over a period of weeks.

**PRESENTING SIGNS AND SYMPTOMS**

Most infected persons remain asymptomatic, and few progress to acute or chronic disease. Common presentations include hydrocele, adenolymphangitis, microscopic hematuria and proteinuria, and lymphangiectasia. Acute adenolymphangitis is a form of retrograde lymphangitis accompanied with high fevers, lymphadenitis, and often thrombophlebitis. *W. bancrofti* infection involves the genital lymphatics, and it often manifests with epididymitis and scrotal pain. Travelers to endemic regions may develop acute edema, urticaria, and local lymphadenitis, especially of the femoral, axillary, inguinal, or epitrochlear lymph nodes.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Parasite detection is key for definitive diagnosis. Blood and hydrocele fluid may demonstrate microfilariae, although blood draws should be timed based on the periodicity of the endemic species suspected. ELISA and rapid-format immunochromatographic card tests are available for *W. bancrofti* antigens. In addition, PCR assays for *W. bancrofti* and *Brugia* DNA are available.
diethylcarbamazine (10% in lotion) can be applied to the skin as a localized Mazotti test; it is considered safer than an oral test. If there are Onchocerca volvulus present in the ocular chamber, diethylcarbamazine treatment can lead to ocular damage.

**Platyhelminthes (Flatworms)**

The cestodes, or tapeworms, are segmented. Humans serve as intermediate hosts for *Taenia solium*, *Echinococcus*, *Hymenolepis*, *Sparganosis*, *Coenurosis*, and *Diphylidiasis* species. *Taenia saginata* and *Diphyllobothrium* also can infect humans, although these species cannot complete their life cycle within human hosts. Tapeworm eggs are transmitted by the fecal-oral route and are endemic in areas with poor sanitation and livestock.

**CESTODE: TAENIA SOLIUM (PORK TAPEWORM)**

**EPIDEMIOLOGY**

*Taenia* has a worldwide distribution and is highly endemic in Latin America, Africa, Eastern Europe, Central and South Asia, and the Middle East. Approximately 45 *Taenia* species and subspecies are currently recognized. *T. solium* infection of the CNS, or neurocysticercosis, is a leading cause of acquired epilepsy and may result in more than 50,000 deaths annually.

The most distinctive feature of *Taenia* infection is that humans can serve as both the definitive host and the intermediary host. When humans are infected by the larval stage, the infection is known as cysticercosis. Infection with the adult tapeworm is associated with taeniasis. For *T. saginata* and *T. solium*, humans serve as the final obligatory host. Infection is uncommon in infants and vegetarians.

**TRANSMISSION AND LIFE CYCLE**

Humans acquire the infection by ingestion of undercooked pork containing cysticerci (larval cysts). The cysts contain protoscolices. After ingestion, the protoscolices are released and attach to the intestinal wall. Each protoscolex can become an adult tapeworm. The head of the worm generally resides in the jejunum. Adult worms contain 800 to 900 proglottids. Maturation occurs over 2 to 4 months. The mature proglottids are hermaphroditic, become gravid, and contain 1000 to 2000 eggs. These eggs are passed in the stool. Pigs acquire the infection from soil contaminated with human feces, and then larvae are activated, penetrate tissues, and encyst within 2 to 3 months.

**PRESENTING SIGNS AND SYMPTOMS**

Most patients are infected by a single worm. The symptoms are usually mild, or the patient may be asymptomatic. Because tapeworms can survive for years in an otherwise healthy host, symptomatic patients may have a protracted clinical course. Complaints are nonspecific and include indigestion, anorexia, diarrhea, constipation, and vague abdominal pain.

Severe signs and symptoms may include intestinal obstruction, appendicitis, and perforation. The worms occasionally migrate to the biliary system, respiratory tract, uterine cavity, or nasopharynx. If cysts are in the CNS, the patient may present with seizures, meningitis, stroke, or signs of increased intracranial pressure. Approximately 30% of patients with neurocysticercosis have residual calcification, and less than 20% have seizures. Parasite invasion of the ventricles can result in obstructive hydrocephalus. Extraparenchymal disease leads to a worse prognosis, secondary to invasive cyst growth and increased cerebrospinal fluid (CSF) debris and inflammation. Classic calcified lesions from prior infections are often seen on computed tomography (CT) or magnetic resonance imaging (MRI).

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Infection is confirmed by finding the eggs or proglottids in the feces. The mobile proglottids often move with discharge, and some patients may feel their passage. Because the eggs are often eliminated intermittently, several stool specimens may need to be obtained. ELISA, DNA probes, and co-proantigen assays are available. The enzyme-linked immunoelectrotransfer blot (EITB) assay is quite specific and sensitive.

Neurocysticercosis is usually diagnosed by the characteristic CNS cystic lesions, which are nodular calcifications 5 to 20 mm in diameter, with or without ring enhancement on CT or MRI. Although calcified lesions are identified more easily on CT, MRI is more sensitive for ring enhancement and cystic lesions. The CSF may show pleocytosis, elevated protein, and occasionally low glucose concentrations.

**TREATMENT**

Calcified cysts do not warrant antiparasitic therapy. Patients with seizures may be treated with antiepileptics, and occasionally endoscopic surgery or ventriculoperitoneal shunting is necessary to relieve obstructive hydrocephalus. Corticosteroids may help mitigate cerebrovascular complications and may be used in the treatment of cysticercotic encephalitis.

**CESTODE: ECHINOCOCCUS**

**EPIDEMIOLOGY**

Echinococcosis is caused by the larval invasion of *Echinococcus granulosus*, *Echinococcus multilocularis*, and *Echinococcus vogeli*. These cestodes are ubiquitous, with...
particularly heavy endemicity in Central Asia, the Mediterranean, the Middle East, South America, and eastern Africa. *E. multilocularis* is confined to the Northern Hemisphere. *E. granulosus* causes cystic echinococcosis, with 3 million cases, and *E. multilocularis* is responsible for the alveolar variant, with half a million cases. Canines are the definitive hosts and pass echinococcal eggs in feces. Intermediate mammalian hosts then ingest the eggs, and eventually cysts develop. The cycle is continued as canines eat infected meat.

**TRANSMISSION AND LIFE CYCLE**

*E. granulosus* has three proglottids, one of which is gravid and releases eggs. After ingestion of the parasite, embryos penetrate the intestinal mucosa and enter the portal circulation. Fluid-filled hydatid cysts are formed in organs, commonly the lungs and liver. Within these hydatid cysts, brood capsules that house new larvae develop.

**PRESENTING SIGNS AND SYMPTOMS**

The cysts grow over several years, and patients are usually asymptomatic until large cyst size or encroachment causes abdominal pain, palpable masses, biliary colic, or, if in the lungs, dyspnea, cough, pleurisy, or hemoptysis. Cyst rupture may provoke an anaphylactic or allergic reaction with eosinophilia, fever, pruritus, and rash. Less commonly, the cysts can invade bone, CNS, cardiac tissue, and other intraabdominal structures.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Radiographs, CT, ultrasound, and MRI can all reveal cysts. Additional testing of aspirated fluids may reveal protoscolices. In addition, immunoblotting techniques are available for serologic antigen testing.

**TREATMENT**

Treatment depends on cyst size, location, and symptoms. Ultrasound staging is recommended. Percutaneous aspiration, infusion of scolicidal agents, and reaspiration (PAIR) is less invasive and is preferred over surgical removal. If cysts are superficial, honeycombed, connected to the biliary systems, or intrapulmonary, PAIR is not recommended. For complicated cysts, surgical intervention is warranted.

**CESTODE: DIPHYLLOBOTHRIUM (FISH TAPEWORM)**

*Diphyllobothrium* species are estimated to infect more than 20 million people. Although an overall decline in North America, Europe, and Asia has been reported, outbreaks have occurred in Russia, Japan, South Korea, Italy, France, Switzerland, and South America. *Diphyllobothrium* also enjoys a range of hosts, from humans to foxes, bears, sea birds, and other fish-eating mammals.

**TRANSMISSION AND LIFE CYCLE**

The fish tapeworm, or *Diphyllobothrium latum*, holds the dubious honor for longest tapeworm known to humankind, with adult worms measuring up to 10 to 25 m. The adult tapeworms consist of 3000 to 4000 proglottids and produce up to 1 million eggs a day; they may survive for decades in the human host. Egg-laden feces enter lakes, rivers, and deltas, and the eggs hatch into embryos. Crustaceans, usually *Cyclops* or *Diaptomus* species, ingest the free-swimming embryos, known as coracidia. The coracidia penetrate the crustacean intestinal wall and develop into a procercoid. The crustacean host is eaten by a freshwater or marine fish, and additional development takes place. The procercoid larva migrates into fish tissue and organs and sometimes encysts. Multiple predatory fish, such as perch, pike, burbot, walleye, snook, Alaska blackfish, salmon, whitefish, trout, and Japanese anchovy have been implicated as hosts. After the host ingests a meal of undercooked or raw fish, *Diphyllobothrium* matures in the human gastrointestinal tract within a few weeks.

**PRESENTING SIGNS AND SYMPTOMS**

Most *Diphyllobothrium* infections are asymptomatic or are characterized by mild gastrointestinal upset. Acute abdominal pain, cholecystitis, cholangitis, and intestinal obstruction are rare. Approximately 2% of infected persons demonstrate megaloblastic anemia secondary to tapeworm absorption of vitamin B<sub>12</sub>. In older patients and in patients with chronic infections, neurologic manifestations of vitamin B<sub>12</sub> deficiency may arise.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

Stool examination reveals eggs and segment chains. After ingestion by the human host, adult worms usually begin producing eggs within 15 to 45 days, so results of early stool studies may be negative.

**TREATMENT**

Intraduodenal Gastrografin has been used to remove large cestodes. Heating fish to 54° C for 5 minutes or freezing to −18° C for 24 hours kills *Diphyllobothrium*. Placing fish in a 12% brine solution kills the eggs.
TREMATODA (FLUKES)

EPIDEMIOLOGY

Trematodes represent a diverse group of flatworms, or flukes, that cause human infection through invasion of blood, intestines, biliary system, and lungs. *Schistosoma* is perhaps the best known of the flukes. *Clonorchis* and *Opisthorchis* species cause biliary inflammation and obstruction and predispose patients to cholangiocarcinoma. *Paragonimus* and *Fasciolopsis* species cause granulomatous disease in the pulmonary and intestinal systems, respectively.

TRANSMISSION AND LIFE CYCLE

The life cycle of most trematodes involves a mammalian definitive host and intermediate snail hosts. Sexual reproduction occurs in the mammalian host, with subsequent asexual reproduction in snail hosts. Ova are excreted in feces or sputum, and they become miracidia that then undergo asexual reproduction in often multiple intermediate hosts. Free-living cercariae larvae and encysted metacercariae infect the definitive host. Transmission to humans is through ingestion or direct dermal penetration.

TREMATODA: SCHISTOSOMA

EPIDEMIOLOGY

Human schistosomiasis, also known as bilharziasis, remains a serious health threat in Africa, Southeast Asia, South America, and the Middle East. Schistosomiasis is a complex of acute and chronic parasitic infections caused by digenetic blood nematodes. Infections with *Schistosoma haematobium* (bladder fluke), *Schistosoma mansoni*, *Schistosoma japonicum*, *Schistosoma mekongi*, and *Schistosoma intercalatum* (intestinal flukes) cause illness in humans. Schistosomes have been documented to infect humans for thousands of years and are associated with agricultural civilizations of the great river valleys. Hematuria, most likely caused by *S. haematobium*, occurred in ancient Egypt and Mesopotamia.

TRANSMISSION AND LIFE CYCLE

The adult fluke lays eggs that leave the definitive host through the feces or urine, depending on the species and the host location. A freshwater species of snail is required as an intermediate host for each species of fluke. In general, the miracidium (larval stage) emerges from the egg in freshwater, enters the snail host, and then undergoes multiplication. The cercaria (final larval state) leaves the snail. These larvae penetrate the tissues of humans through contact in infested freshwater. The cercaria then loses its tail, becomes a schistosomulum, and migrates to blood vessels to become an adult. Adults migrate into intestinal veins and vesical veins (*S. haematobium*) and lay eggs in the vasculature. The ova transmigrate through the lumen and are voided in feces or urine. Ova that reach freshwater complete the life cycle in intermediate hosts.

SIGNS AND SYMPTOMS

Patients may be asymptomatic initially, or they may show mild pruritic maculopapular skin lesions within hours to days after exposure to cercariae. Four to 8 weeks after invasion, some patients may present with fever, eosinophilia, lymphadenopathy, and hepatosplenomegaly. Acute schistosomiasis or Katayama fever resembles serum sickness, and it appears to be related to antigen excess during sexual maturation of the parasite. It is associated with a mortality rate of up to 25%.

Chronic schistosomiasis persists for months to years after primary exposure is more common. A granulomatous reaction to *Schistosoma* eggs, coupled with host cell-mediated inflammatory response lead to organomegaly and obstruction. Patients may present with abdominal pain, hematemesis, ascites, hematuria, dysuria, vulvar or perianal lesions, dyspnea on exertion, fatigue, cough, chest pain, or seizures, depending on the site of infection and involvement of the body system.

Eggs induce an immune response as they travel to the liver, intestine, bladder, and rarely, to the brain or spinal cord. Granuloma formation in the bowel wall with *S. mansoni* or *S. japonicum* may cause bloody diarrhea, cramping, and colonic polyposis. *Schistosoma* can also cause pulmonary and CNS disease, with sequelae including pulmonary hypertension, cor pulmonale, epilepsy, and transverse myelitis.

Egg retention and granuloma formation in the urinary tract with *S. haematobium* can lead to hematuria, urinary tract infections, glomerulonephritis, obstructive uropathy, dysuria, and bladder polyps and ulcerations. *S. haematobium* is considered a carcinogen in squamous cell bladder cancer.

DIAGNOSIS AND MEDICAL DECISION MAKING

Geographic history and clinical presentation are key considerations in diagnosing schistosomiasis. The diagnosis can also be aided by recovery of eggs in feces or urine, but Kato thick smear microscopy must be ordered specifically. Hatching assays on fresh stool specimens help to distinguish active from treated infection. Dead eggs may be shed for up to a year. Peripheral eosinophilia supports the diagnosis. Gross and microscopic hematuria is common in patients infected by *S. haematobium*.

Imaging techniques such as ultrasonography, echocardiography, and radiography can help pinpoint organ system involvement. ELISA testing is available and confirms past exposure, although it cannot discriminate between acute and chronic infection. The EITB test also yields good results. In some cases, colonic biopsy is necessary to visualize parasite eggs in the bowel wall.

Acute schistosomiasis should be suspected in patients recently returning from endemic areas who present with fever, headache, malaise, arthralgias or myalgias, bloody diarrhea, and right upper quadrant abdominal pain, especially if they have a history of freshwater swimming or bathing.
**EPIDEMIOLOGY**

Bedbugs, or cimicids, are insects that have plagued humanity since ancient Egypt. These ectoparasites are found in temperate and tropical regions worldwide, and global infestations are increasing. Much media attention has been paid to bedbugs, and millions of dollars have been spent in the hospitality and private sectors. Travelers, backpackers, immigrants, guest workers, the homeless, and persons sharing close quarters, such as military barracks and dormitories, are potential hosts and vectors.

**TRANSMISSION AND LIFE CYCLE**

Cimicids are wingless, obligate hematophages. Both sexes require blood meals. *Cimex lectularius* and *Cimex hemipterus* are the two species that prefer human hosts. Adults are oval, flat, approximately 5 mm in length, and reddish brown. Adults can survive for up to 12 months, perhaps even 2 years, without feeding. Nymphs tend to be lighter in color. Bedbugs usually conceal themselves during the day, often in bedding, carpet, wallpaper, or any crevice, usually within 1 to 2 m of a host.

**PRESENTING SIGNS AND SYMPTOMS**

Most persons bitten by bedbugs are asymptomatic, perhaps bearing miniscule puncture wounds to exposed extremities, face, and neck. Those who seek medical attention commonly present with 2- to 5-mm pruritic maculopapular, erythematos lesions at bite sites. If not excoriated or superinfected, these lesions resolve spontaneously within a week. Occasionally, some patients experience local popular rashes or diffuse urticaria. Bullous rashes, folliculitis, cellulitis, and eczematoid dermatitis may develop after a few days.

Anaphylaxis and asthma have also been reported, and they may be related to increased exposure with subsequent feedings. Saliva of bedbugs contains a protein, nitrophorin, that may be responsible for cutaneous reactions. Beyond the physical irritation, bedbugs are responsible for anxiety, stigma, and insomnia.

**TREATMENT**

Early treatment with cidal drugs may exacerbate Katayama fever, and concomitant steroid therapy is recommended. Patients with long-term infection may require procedures such as bladder stents, endoscopic treatment, or surgery.

Helminths cause a large burden of disease, yet their coevolution with humans and adaptation to our immune defenses may be beneficial (Box 177.2). Numerous epidemiologic and experimental studies have noted the low prevalence of allergy and autoimmune disease among populations with chronic helminth infections. Helminth infections generally cause a skewed type 1 helper T-cell (Th2) response. The Th1 arm of the immune system is often suppressed or down-regulated. Populations with chronic helminthiases may not clear microbial infections adequately or respond optimally to vaccination. *Schistosoma* and *Onchocerca* infections appear to decrease tetanus and tuberculosis vaccine efficacy, and *Ascaris* infection similarly dampens the immune response to *Mycoplasma* pneumonia vaccination.16

**Box 177.2 Helminth Immunomodulation**

“The hygiene hypothesis suggests that microbes and worms are important for shaping and tuning the development and function of our immune system.”

As developed countries experience better sanitation and reduced exposure to infectious disease agents, the immune system may be left “uneduicated” and may develop “incorrect” or uncontrolled inflammation responses: rhinitis, atopic dermatitis, asthma, inflammatory bowel disease, multiple sclerosis, and type 1 diabetes mellitus. Helminth infections as therapies have been observed to decrease colitis in both murine and human models. A few studies have shown benefit from *Trichuris* infection in patients with inflammatory bowel disease; similar results have been reported in patients with multiple sclerosis. Much more research is needed to characterize human-helminth immunomodulatory dynamics. Genetics and environment play important roles.

**PATIENT TEACHING TIPS**

Inspect bedding carefully. Signs of bedbugs can be seen on infested mattresses and in bed frames. Affected areas should be thoroughly cleaned. Bedding, infested furniture, and carpets should be placed in airtight plastic bags, steam cleaned, or thrown away. Plastic mattress covers can be placed to prevent nocturnal emergence of the insects.
Scabies

EPIDEMIOLOGY

Scabies is a common parasitic infection caused by the mite Sarcoptes scabiei var. hominis, an arthropod of the order Acarina. Worldwide prevalence is estimated at 300 million. Scabies is endemic in sub-Saharan Africa, South and Central America, India, the South Pacific, and among Aboriginal communities in Australia. Additionally, infestations are sporadic in industrial countries. Overcrowding, poor hygiene and poor nutrition, poverty, war, and dementia are predisposing factors. Despite the stigma of scabies as an infection of the poor, scabies affects all ethnic and socioeconomic groups.

TRANSMISSION AND LIFE CYCLE

These obligate parasites complete their entire life cycle on humans. Scabies mites measure 0.2 to 0.5 mm in length, naked to the human eye. Only the female mite burrows into the skin, where the mite tunnels at a rate of 2 mm/day. The parasite can survive approximately 36 hours away from the human host. Each mite lays approximately 10 to 25 eggs and then dies in the stratum granulosum. Larvae hatch in 3 to 4 days, molt, then leave the burrow for the surface, copulate, and continue the cycle. Maturation is complete in 15 days.

The number of mites infesting a person usually ranges from 5 to 15, although thousands may be present in patients with crusted scabies. Symptoms of infestation usually manifest 3 to 6 weeks after the mite is acquired, but they may appear as soon as 1 day after the mite is acquired in cases of reinfestation, as a result of a hypersensitivity reaction. The primary mode of transmission is direct skin-to-skin contact. Investigators estimate that a 15- to 20-minute encounter is sufficient to transmit the mite. Transmission through shared clothing or other objects is rare, but it may occur with crusted scabies. Mites crawl at a rate of 2.5 cm/minute on human skin. The greater the parasite load, the greater is the chance of transmission. Sexual transmission also occurs. Immunocompromised hosts, including those who are HIV positive, HTLV-1 positive, undergoing immunosuppressant therapies, and malnourished, are at higher risk for developing crusted (Norwegian) scabies.

SIGNS AND SYMPTOMS

Intense, intractable, pruritic dermatitis with erythematous papulovesicular lesions characterize scabies infections. The itch is usually worse at night or after a hot shower. Skin burrows, gray serpiginous lines 1 to 10 mm in length, are pathognomonic findings. In adults, burrows and nodules are usually found in interdigital web spaces, axillary folds, extremities, buttocks, nipples, and genitals. Very young children and immunocompromised persons may have facial and neck lesions. Lesions to palms and soles, pinkish brown nodules, and acral pustules are unique to infested infants.

Crusted, or Norwegian, scabies is usually confined to immunocompromised, older, cognitively impaired, and institutionalized patients. Presumably, the lack of scratching may allow superinfection to occur. Crusted scabies represents a hyperinfection with thousands to millions of mites present. The lesions appear hyperkeratotic, similar to psoriatic papules, and can cover large areas of the scalp, face, neck, and extremities. Skin crusts may be loose or adherent, flaky or thick. Nail involvement is common with crusted scabies, as are eczematization and impetigo. Large flakes of epidermis slough off, carrying mites and furthering transmission. In crusted scabies, high levels of IgE and IgG and peripheral eosinophilia are present.

Nodular scabies manifests with pruritic, violaceous nodules localized to the groin, axilla, and male genitalia. These nodules may represent a variant hypersensitivity reaction because mites are not found within them. Rarely, bullous lesions may occur with scabies, perhaps because of superinfection with Staphylococcus. Pyoderma, or bacterial skin infections, may be secondary to scabies infections, especially in the tropics.

DIAGNOSIS AND MEDICAL DECISION MAKING

The diagnosis is primarily clinical. Patients complain of generalized and intense pruritus, usually sparing the face and head. Symptoms are often out of proportion to examination findings. The definitive diagnosis is made by identification of mites, eggs, or mite fecaliths on microscopic evaluation of burrow scrapings. Obtaining multiple superficial skin samples from characteristic lesions is helpful. This is done by gently scraping laterally across the skin with a scalpel. If the number of mites is low, as is commonly the case in classic scabies, mites may not be identified. Skin biopsy may also be performed.

Enhanced microscopy techniques, such as epiluminescence microscopy and noncomputed dermoscopy, may also identify scabies products in burrows. ELISA and PCR assays can also aid in detection of scabies antibodies and help in determining the efficacy of treatment.

Scabies may mimic several diseases, including bullous pemphigoid, urticaria, chronic lymphocytic leukemia, B-cell lymphoma, necrotizing vasculitis, lupus erythematosus, secondary syphilis, atopic dermatitis, and urticaria pigmentosa.

TREATMENT

Empiric treatment is not recommended in the absence of a history of prolonged skin-to-skin exposure, typical eruption, or both. The infested patient and close physical contacts should be treated simultaneously, regardless of whether symptoms are present. Crusted scabies is very easily transmitted, and patients who have been even minimally exposed should be treated. Crusted scabies requires hospital admission, and keratolytics improve penetrance of topical therapy.
penetration. Usually, at least three consecutive topical treatments are required. Pruritus normally persists after treatment and may worsen as release of mite antigens exacerbates the hypersensitivity reaction. Most recurrent cases can be traced to untreated contacts.

**TRANSMISSION AND PATHOPHYSIOLOGY**

*Pediculus* species carry out their entire life cycle on the human host and survive only briefly in the environment. One female head louse can lay up to 150 eggs during her 1-month lifespan. Eggs (nits) are cemented onto hair shafts of the host and hatch in 1 week; full maturation to adult stages is completed in another week. If nits are found less than 1 cm from the scalp, an active infestation is considered highly likely. Because human hair grows at a rate of 1 cm/month, and nits can remain attached to hair for up to 6 months, the presence of nits a few centimeters from the scalp may not represent an active infestation.

Lice are transferred directly from host to host. Eggs are transferred from louse-infested clothing or personal articles such as shared combs, head-phones, beds, and hats. Head lice can live up to 55 hours without a host.

**PRESENTING SIGNS AND SYMPTOMS**

Signs and symptoms may include pruritus and pruritic papules at the site of infestation, especially in occipital and retroauricular regions. Saliva and fecal excretions of the louse may cause a local hypersensitivity reaction including fever, conjunctivitis, malaise, cervical lymphadenopathy, and a rash, mimicking a viral exanthem. If a secondary bacterial infection occurs, the lesion may resemble mange. Patients often identify the presence of a parasite before they present for treatment.

**DIAGNOSIS AND MEDICAL DECISION MAKING**

The diagnosis is made by identifying lice or nits on the patient. Using a fine-toothed comb and placing any removed lice or nits on a light-colored surface and viewing them with a magnifying device may assist in the identification. The head louse is a gray-white, 3- to 4-mm insect. Currently, no antibody or serologic tests exist. The diagnosis is based on clinical suspicion, with confirmation on finding *Pediculus* adults, nymphs, or nits.

**TREATMENT**

Three main avenues exist for treatment: mechanical removal, topical agents, and oral therapy (Table 177.1). Children can return to school immediately after completion of the first application of a topical insecticide. Wet hair combing to remove *P. humanus capitis* nits and adults is not as effective as the use of topical agents. Infested clothing and bed linen should be washed in hot water, dry cleaned, or discarded.
Table 177.1 Treatment of Helminth Infections

<table>
<thead>
<tr>
<th>ORGANISM</th>
<th>TREATMENT</th>
<th>PRECAUTIONS AND COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Roundworms: Intestinal</strong></td>
<td></td>
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<tr>
<td><em>Ascaris</em></td>
<td>Albendazole 400 mg PO × 1</td>
<td>Maximum dose, 1 g</td>
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<td></td>
<td>Mebendazole 500 mg PO × 1</td>
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<td></td>
<td>Mebendazole 100 mg PO bid × 3 days</td>
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<tr>
<td></td>
<td>Pyrantel pamoate 11 mg/kg PO × 1</td>
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<td></td>
<td>Ivermectin 150-200 mcg/kg PO × 1</td>
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<td></td>
<td></td>
<td>Lactating/pregnant women</td>
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<td></td>
<td></td>
<td>Weight &gt; 15 kg</td>
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<tr>
<td></td>
<td>Albendazole 400 mg PO × 1</td>
<td></td>
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<tr>
<td><em>Necator, Ancylostoma</em></td>
<td>Mebendazole 500 mg PO × 1</td>
<td>Maximum dose, 1 g</td>
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<td></td>
<td>± 100 mg PO bid × 3 days</td>
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<tr>
<td></td>
<td>Pyrantel pamoate 11 mg/kg PO × 3 days</td>
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<td></td>
<td>Supplemental iron PO</td>
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<td></td>
<td></td>
<td>Lactating/pregnant women</td>
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<td></td>
<td></td>
<td>Weight &gt; 15 kg</td>
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<tr>
<td><em>Strongyloides: simple</em></td>
<td>Ivermectin 200 mcg/kg PO × 2 days; ± repeat in 1-2 wk</td>
<td>Lactating/pregnant women</td>
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<tr>
<td></td>
<td>Albendazole 400 mg PO bid × 3-7 days</td>
<td>Weight &gt; 15 kg</td>
</tr>
<tr>
<td><em>Strongyloides: disseminated</em></td>
<td>Ivermectin 200 mcg/kg PO once daily; continue +2 wk after symptom resolution</td>
<td>Lactating/pregnant women</td>
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<tr>
<td></td>
<td>+ Albendazole or thiabendazole</td>
<td>Weight &gt; 15 kg</td>
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<tr>
<td></td>
<td>Albendazole 400 mg PO bid; continue +2 wk after symptom resolution</td>
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<tr>
<td></td>
<td>Thiabendazole 25 mg/kg PO bid; continue +2 wk after symptom resolution</td>
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<tr>
<td><em>Enterobius</em></td>
<td>Albendazole 400 mg PO × 1</td>
<td>Maximum dose, 1 g</td>
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<td>Mebendazole 100 mg PO × 1</td>
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<tr>
<td></td>
<td>Pyrantel pamoate 11 mg/kg PO × 1</td>
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<td></td>
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<td>TOC for pregnant women</td>
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<tr>
<td><em>Trichuris</em></td>
<td>Albendazole 400 mg PO × 3 days</td>
<td>Lactating/pregnant women</td>
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<td></td>
<td>Mebendazole 200 mg PO × 3 days</td>
<td>Weight &gt; 15 kg</td>
</tr>
<tr>
<td></td>
<td>Ivermectin 200 mcg/kg PO × 3 days</td>
<td></td>
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<tr>
<td><strong>Roundworms: Tissue</strong></td>
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</tr>
<tr>
<td><em>Trichinella: mild</em></td>
<td>Supportive therapy</td>
<td></td>
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<tr>
<td><em>Trichinella: hypersensitivity</em></td>
<td>Corticosteroid taper</td>
<td></td>
</tr>
<tr>
<td><em>Trichinella: enteric only</em></td>
<td>Albendazole 15 mg/kg/day PO × 10-15 days, may repeat in 5 days</td>
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<td></td>
<td>Mebendazole 5 mg/kg/day PO × 10-15 days, may repeat in 5 days</td>
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<tr>
<td></td>
<td>Corticosteroids if severe disease</td>
<td></td>
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<tr>
<td><em>Cutaneous larva migrans</em></td>
<td>Albendazole 400-800 mg/day PO × 3-5 days</td>
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<td></td>
<td>Ivermectin 200 mcg/kg PO × 2 days</td>
<td></td>
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<tr>
<td><em>Lymphatic filariasis</em></td>
<td>Diethylcarbamazine 6 mg/kg/day PO × 12 days</td>
<td>Hypersensitivity reaction</td>
</tr>
<tr>
<td><em>Wucheria bancrofti</em></td>
<td>Albendazole 400 mg PO bid × 21-30 days</td>
<td></td>
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<tr>
<td></td>
<td>Doxycycline 200 mg PO once daily × 8 wk</td>
<td></td>
</tr>
<tr>
<td><em>Brugia malayi</em></td>
<td>Albendazole 400 mg PO × 1 + diethylcarbamazine 6 mg/kg PO × 1</td>
<td>Microfilaricidal only</td>
</tr>
<tr>
<td></td>
<td>or Ivermectin 200-400 mcg/kg PO × 1</td>
<td>Lactating/pregnant women</td>
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<tr>
<td></td>
<td></td>
<td>Weight &gt; 15 kg</td>
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<tr>
<td><em>Loa loa</em></td>
<td>Diethylcarbamazine 8-10 mg/kg/day PO × 21 days</td>
<td>Microfilaricidal only</td>
</tr>
<tr>
<td></td>
<td>Diethylcarbamazine 300 mg/week for prophylaxis</td>
<td></td>
</tr>
<tr>
<td><em>Mansonella ozzardi</em></td>
<td>Ivermectin 200 mcg/kg PO × 1</td>
<td>Lactating/pregnant women</td>
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<tr>
<td></td>
<td></td>
<td>Weight &gt; 15 kg</td>
</tr>
<tr>
<td><em>Mansonella perstans</em></td>
<td>Mebendazole 100 mg PO bid × 30 days</td>
<td>Often not effective</td>
</tr>
<tr>
<td></td>
<td>Albendazole 400 mg PO bid × 10 days</td>
<td></td>
</tr>
<tr>
<td><em>Mansonella streptocerca</em></td>
<td>Diethylcarbamazine 6 mg/kg/day PO × 14-21 days</td>
<td>Lactating/pregnant women</td>
</tr>
<tr>
<td></td>
<td>Ivermectin 150 mcg/kg PO × 1</td>
<td>Weight &gt; 15 kg</td>
</tr>
<tr>
<td>ORGANISM</td>
<td>TREATMENT</td>
<td>PRECAUTIONS AND COMMENTS</td>
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<td>---------------------------</td>
<td>---------------------------------------------------------------------------</td>
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<tr>
<td><strong>Onchocerca</strong></td>
<td>Ivermectin 150-200 mcg/kg PO × 1; can repeat in 3-6 mo</td>
<td>Microfilaricidal only&lt;br&gt;Mazzotti reaction&lt;br&gt;Pulmonary eosinophilia</td>
</tr>
<tr>
<td></td>
<td>Doxycycline 100-200 mg PO once daily × 6 wk</td>
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<tr>
<td></td>
<td>Mebendazole 1 g PO bid × 28 days</td>
<td></td>
</tr>
<tr>
<td><strong>Flatworms: Cestoidea</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Taenia solium</strong>: intestinal</td>
<td>Praziquantel 10-20 mg/kg PO × 1 + Cimetidine</td>
<td>Hepatic impairment, does not inactivate eggs released from dead adult worms</td>
</tr>
<tr>
<td></td>
<td>Niclosamide 2 g PO</td>
<td></td>
</tr>
<tr>
<td><strong>Taenia solium</strong>: neurocysticercosis</td>
<td>Praziquantel 50-60 mg/kg/day PO × 15-30 days + steroids&lt;br&gt;albendazole 15 mg/kg/day PO × 8-30 days&lt;br&gt;if &gt; 60 kg, then 400 mg PO bid × 8-30 days&lt;br&gt; + Anticonvulsants and corticosteroids:&lt;br&gt;Dexamethasone 16-24 mg/day PO&lt;br&gt;Followed by prednisone 1 mg/kg/day PO&lt;br&gt;Taper over 2-3 wk&lt;br&gt;± Endoscopic surgery/ventriculoperitoneal shunt</td>
<td>Hepatic impairment&lt;br&gt;Maximum, 800 mg/day</td>
</tr>
<tr>
<td><strong>Echinococcus</strong></td>
<td>Nonsurgical cysts: albendazole 10-15 mg/kg/day or mebendazole 40-50 mg/kg/day for 3-6 months&lt;br&gt;Surgical adjuncts: albendazole 15 mg/kg/day PO, start minimum of 4 days before procedure and continue for 8 weeks after procedure&lt;br&gt;Intraoperative: praziquantel 40 mg/kg/day PO&lt;br&gt;PAIR 90% ethanol or 20% hypertonic saline&lt;br&gt;Praziquantel 50 mg/kg/day PO × 2 wk</td>
<td>Neutropenia and liver toxicity with prolonged albendazole and mebendazole use</td>
</tr>
<tr>
<td><strong>Diphyllobothrium latum</strong></td>
<td>Praziquantel 25 mg/kg PO × 1</td>
<td></td>
</tr>
<tr>
<td><strong>Diphyllobothrium other spp.</strong></td>
<td>Praziquantel 10 mg/kg PO × 1 &lt;br&gt;Niclosamide 2 g PO × 1 adults &lt;br&gt;1 g PO × 1 pediatrics &gt; 6 yr old</td>
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</tr>
<tr>
<td><strong>Flukes: Trematoda</strong></td>
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<tr>
<td><strong>Schistosoma</strong></td>
<td>Praziquantel 40-60 mg/kg/day PO × 1 day</td>
<td>Age &gt; 4 yr&lt;br&gt;Hepatic impairment</td>
</tr>
<tr>
<td><strong>Other Flukes</strong></td>
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<tr>
<td><strong>Clonorchis</strong></td>
<td>Praziquantel 75 mg/kg/day PO × 1-2 days</td>
<td>Hepatic impairment</td>
</tr>
<tr>
<td><strong>Fasciola</strong></td>
<td>Albendazole 10 mg/kg/day PO × 7 days</td>
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<tr>
<td><strong>Opisthorchis</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Paragonimus</strong></td>
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<tr>
<td><strong>Bedbugs: Cimex spp.</strong></td>
<td>Antipuritics ± corticosteroids</td>
<td></td>
</tr>
<tr>
<td><strong>Scabies: Sarcopes spp.</strong></td>
<td>Permethrin 5% topical, 8-14 hr; wash off ± repeat 1 wk</td>
<td>Age &gt; 2 mo&lt;br&gt;First-line treatment in United States&lt;br&gt;Resistance reported</td>
</tr>
<tr>
<td></td>
<td>Lindane 1% topical, 6 hr then wash; can repeat in 1 wk</td>
<td>Lactating/pregnant women&lt;br&gt;Increased seizure risk&lt;br&gt;Aplastic anemia risk&lt;br&gt;Weight &gt; 50 kg&lt;br&gt;Age &gt; 6 mo&lt;br&gt;Resistance reported</td>
</tr>
<tr>
<td></td>
<td>Crotamiton 10% topical bid × 5 days&lt;br&gt;or × 1 for 48 hr total</td>
<td>Lactating/pregnant women</td>
</tr>
<tr>
<td></td>
<td>Benzyl benzoate 10-25% 2-3 × in 1 day</td>
<td>Lactating/pregnant women&lt;br&gt;Age &gt; 2 yr&lt;br&gt;Not available in United States&lt;br&gt;Effective in permethrin-resistant scabies</td>
</tr>
</tbody>
</table>

Continued
### Table 177.1  Treatment of Helminth Infections—cont’d

<table>
<thead>
<tr>
<th>ORGANISM</th>
<th>TREATMENT</th>
<th>PRECAUTIONS AND COMMENTS</th>
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</thead>
</table>
| Sulfur 2-10% petroleum, 2-3 days | | Dermatitis  
Stains clothing  
Not available in United States |
| Sulfur 10% may be more effective than permethrin for crusted scabies | | |
| Malathion 0.5% 8-12 hr | | Lactating/pregnant women  
Age > 6 mo  
Not available in United States |
| Ivermectin 0.8% topical | | Lactating/pregnant women  
Weight > 15 kg |
| Ivermectin 200 mcg/kg PO x 1; repeat in 2 wk or Ivermectin 250-350 mcg/kg PO x 1 | | |

#### Lice: *Pediculus humanus capitis*

<table>
<thead>
<tr>
<th>TREATMENT</th>
<th>PRECAUTIONS AND COMMENTS</th>
</tr>
</thead>
</table>
| Permethrin 1% topical 8-14 hr; wash off ± repeat 1 wk | Resistance reported  
Lactating/pregnant women  
Increased seizure risk  
Aplastic anemia risk  
Weight > 50 kg  
Age > 6 mo  
Resistance reported  
Age > 6 mo  
Flammable  
Ovicidal  
Resistance reported |
| May use 5% topical if >2 mo old | |
| Lindane 1% topical, 6 hr then wash; can repeat in 1 wk | |
| Malathion 0.5% topical 12 hr | |
| Carbaryl 0.5% topical | |
| Benzyl alcohol 5%, topical 10 min; repeat in 10 days | |
| Ivermectin 200-400 mcg/kg PO x 1; repeat in 7-10 days | |
| Ivermectin 200 mcg/kg PO days 1, 2, 10 | |
| Spinosad 0.9% cream 10 min; wash | |
| Dimethicone 4% lotion | |
| Essential oils: lavender, coconut, citronella, anise, ylang-ylang | |
| Occlusive dressings: petroleum jelly  
Mayonnaise, olive oil  
LouseBuster 30 min | |
| bid, Twice daily; PAIR, percutaneous aspiration, infusion of scolicidal agents, and respiration; PO, orally; tid, three times daily. | |

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### Suggested Readings

Frankowski BL, Bocchini JA Jr, Committee on School Health the Committee on Infectious Diseases. Head lice. Pediatrics 2010;126:392-403.


### References

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES