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have a nervous system; a muscular system, including muscle cells under the cuticle; and a developed intestinal tract, including an oral cavity and an elongated gut that ends in an anal pore. Adults may range in size from minute to >1 meter in length (with Dracunculus medinen sis, for example, at the long end of this spectrum).

Humans acquire infections with nematode parasites by various routes, depending on the parasitic species. Ingestion of eggs passed in human feces is a major global health problem with many of the intes tinal helminths (e.g., Ascaris lumbricoides). In other species, infecting larvae penetrate skin exposed to fecally contaminated soil (e.g., S. ster coralis, hookworms) or traverse the skin after the bite of infected insect vectors (e.g., filariae). Some nematode infections are acquired by con sumption of specific animal-derived foods (e.g., trichinellosis from raw or undercooked pork or wild carnivorous mammals). As noted above, only two nematodes, S. stercoralis and C. philippinensis, can internally reinfect humans; thus, for all other nematodes, any increases in worm burden must be due to continued exogenous reinfections. ■ ■CESTODES Tapeworms are the cestode parasites of humans. Adult tapeworms are elongated, segmented, hermaphroditic flatworms that reside in the intestinal lumen or, in their larval forms, may live in extraintestinal tissues. Tapeworms include a head (scolex) and a number of attached segments (proglottids). The worms attach to the intestinal tract via their scolices, which may possess suckers, hooks, or grooves. The scolex is the site of formation of new proglottids. Tapeworms do not have a functional gut tract; rather, each tapeworm segment passively and actively obtains nutrients through its specialized surface tegu ment. Mature proglottids possess both male and female sex organs, but insemination usually occurs between adjacent proglottids. Fertilized proglottids release eggs that are passed in the feces. When ingested by an intermediate host, an egg releases an oncosphere that penetrates the gut and develops further in tissues as a cysticercus. Humans acquire infection by ingesting animal tissues that contain cysticerci, and the resultant tapeworms develop and reside in the proximal small bowel (e.g., Taenia solium, T. saginata). Alternatively, if humans ingest eggs of these cestodes that have been passed in human or animal feces, onco spheres develop and can

cause space-occupying extraintestinal cystic lesions in tissues; examples include cysticercosis due to T. solium and hydatid disease due to species of Echinococcus. PART 5 Infectious Diseases ■ ■TREMATODES Trematodes of medical importance include blood flukes, intestinal flukes, and tissue flukes. Adult flukes are often leaf-shaped flatworms. Oral and/ or ventral suckers help adult flukes maintain their positions in situ. Flukes have an oral cavity but no distal anal pore. Nutrients are obtained both through their integument and by ingestion into the blind intestinal tract. Flukes are hermaphroditic except for blood flukes (schistosomes), which are sexually dimorphic. Eggs are passed in human feces (Fasciola, Fascio lopsis, Clonorchis, Schistosoma japonicum, S. mansoni), urine (Schistosoma haematobium), or sputum and feces (Paragonimus). Expelled eggs release miracidia—usually in water—that infect specific snail species. Within snails, parasites multiply and cercariae are released. Depending on the species, cercariae can penetrate the skin (schistosomes) or can develop into metacercariae that can be ingested with plants (e.g., watercress for Fasciola) or with fish (Clonorchis) or crabs (Paragonimus). ■ ■CONCLUSION Many of the so-called neglected tropical diseases are due to helmin thic infections. The health impacts of many helminthic infections are varied and are based on the frequent need for repeated exposures to increase the worm burdens in infected humans. In global regions where exposures to specific helminths occur even in childhood (e.g., fecally derived intestinal nematodes, mosquito-transmitted filariae, or waterborne snail-transmitted schistosomes), the morbidities in infected individuals can include nutritional, developmental, cognitive, and functional impairments. Ongoing global mass-treatment programs are currently aimed at diminishing the local intensity of infection and prevalences of specific helminths and their consequent impacts on the health of local populations.

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Trichinellosis and

Other Tissue Nematode

Infections Nematodes are elongated, symmetric roundworms. Parasitic nema todes of medical significance may be broadly classified as either pre dominantly intestinal or tissue nematodes. The intestinal nematodes are covered in Chap. 239; filarial tissue-dwelling nematodes and Dracunculus medinensis infection are both covered in Chap. 240. This chapter covers the tissue nematodes that cause trichinellosis, visceral and ocular larva migrans, cutaneous larva migrans, cerebral angio strongyliasis, and gnathostomiasis. All of these zoonotic infections result from incidental exposure to infectious nematodes. The clinical symptoms of these infections are due largely to invasive larval stages that (except in the case of Trichinella) do not reach maturity in humans. ■ ■TRICHINELLOSIS Trichinellosis develops after the ingestion of meat containing cysts of Trichinella (e.g., pork or other meat from a carnivore/omnivore). Although most infections are mild and asymptomatic, heavy infections can cause severe enteritis, periorbital edema, myositis, and (infrequently) death. Life Cycle and Epidemiology At least nine species of Trichinella and 13 genotypes are recognized as causes of infection in humans. Two species are distributed worldwide: T. spiralis, which is found in a great variety of carnivorous and omnivorous animals, and T. pseudospiralis, which is found in mammals and birds. T. nativa is present in Arctic and subarctic regions and infects bears, foxes, and walruses; T. nelsoni is found in equatorial eastern Africa, where it is common among felid predators and scavengers such as hyenas and bush pigs; and T. britovi is found in Europe, western Africa, and western Asia among carnivores but not among

domestic swine. T. murrelli is present in wild animals in North American and Japan. T. papuae is found in Papua New Guinea, Thailand, Taiwan, and Cambodia in domestic and feral pigs and in salt water crocodiles and turtles. T. zimbabwensis is present in crocodiles in Tanzania. T. patagoniensis is found in cougars in South America. After human consumption of trichinous meat, encysted larvae are liberated by digestive acid and proteases (Fig. 238-1). The larvae invade the small-bowel mucosa and mature into adult worms. After ~1 week, female worms release newborn larvae that migrate via the circulation to striated muscle. The larvae of all species except T. pseu dospiralis, T. papuae, and T. zimbabwensis then encyst by inducing a radical transformation in the muscle cell architecture. Host immune responses may help to expel intestinal adult worms but have few del eterious effects on muscle-dwelling larvae. Human trichinellosis classically has been caused by the ingestion of infected pork products and thus can occur in almost any location where the meat of domestic or wild swine is eaten. Increasingly, human trichi nellosis has also been acquired from the meat of other animals, including dogs (in parts of Asia and Africa), horses (in Italy and France), and bears and walruses (in northern regions). Although cattle and horses (being herbivores) are not natural hosts of Trichinella, beef and horse meat have been implicated in outbreaks when contaminated or adulterated with trichinous pork or perhaps following ingestion of fodder contaminated with meat parts from naturally infected animals. Approximately 10–20 cases of trichinellosis are reported annually in the United States, down from 400–500 cases per year in the 1940s, largely reflecting the impact of control programs targeting education, pig-raising, and the processing and freezing of pork. Consumption of wild game (especially bear and walrus meat) now accounts for the majority of cases reported each year in North America. Most mild cases probably remain undiagnosed. Pathogenesis and Clinical Features Clinical symptoms of trich inellosis arise from the successive phases of parasite enteric invasion,

Larvae are released in the stomach and mature into adults over 1–2 wks in the small bowel, causing: Irritation and mild abdominal cramping or even diarrhea Encysted larvae ingested in undercooked pork, boar, horse, or bear *T. papuae, T. zimbabwensis, and T. pseudospiralis do not encyst. FIGURE 238-1 Life cycle of Trichinella spiralis (cosmopolitan); nelsoni (equatorial Africa); britovi (Europe, western Africa, western Asia); nativa (Arctic); murrelli (North America); papuae (Papua New Guinea); zimbabwensis (Tanzania); and pseudospiralis (cosmopolitan). CNS, central nervous system. (Reproduced with permission from RL Guerrant et al [eds]: Tropical Infectious Diseases: Principles, Pathogens and Practice, 2nd ed. Elsevier, 2006.) larval migration, and muscle encystment (Fig. 238-1). Most light infec tions (those with <10 larvae per gram of muscle) are asymptomatic, whereas heavy infections (which can involve >50 larvae per gram of muscle) can be life-threatening. An initial enteric phase due to release of ingested muscle larvae may elicit diarrhea, abdominal pain, consti pation, and nausea during the first weeks after infection. Symptoms due to larval migration and muscle invasion begin to appear in the second week after infection. The migrating Trichinella larvae provoke a marked local and systemic hypersensitivity reaction, with fever and eosinophilia. Periorbital and facial edema is common, as are hemorrhages in the subconjunctivae, retina, and nail beds (“splinter” hemorrhages). A maculopapular rash, headache, cough, dyspnea, or dys phagia sometimes develops. Myocarditis with tachyarrhythmias or heart failure—and, less commonly, encephalitis or pneumonitis—may develop and accounts for most deaths of patients with trichinellosis. Upon onset of larval encystment in muscle 2–3 weeks after infec tion, symptoms of myositis with myalgias, muscle edema, and weak ness develop, usually overlapping with the inflammatory reactions to migrating larvae. The most commonly involved muscle groups include the extraocular muscles; the biceps; and the muscles of the jaw,

neck, lower back, and diaphragm. Peaking ~3 weeks after infection, symptoms subside only gradually during a prolonged convalescence. Uncommon infections with T. pseudospiralis, whose larvae do not encapsulate in muscles, elicit a prolonged polymyositis-like illness. Laboratory Findings and Diagnosis Blood eosinophilia devel ops in >90% of patients with symptomatic trichinellosis and may peak at a level of >50% 2–4 weeks after infection. Serum levels of muscle enzymes, including creatine phosphokinase, are elevated in most symptomatic patients. Patients should be questioned thoroughly about their consumption of pork or wild animal meat and about illness in other individuals who ate the same meat. A presumptive clinical diagnosis can be based on fevers, eosinophilia, periorbital edema, and myalgias after a suspect meal. A rise in the titer of parasite-specific antibody, which usually does not occur until after the third week of infection, confirms the diagnosis. Alternatively, a definitive diagnosis requires surgical biopsy of at least 1 g of involved muscle; the yields

Larvae migrate, penetrate striated muscle, reside in “nurse-cells,” and encyst,* causing: Muscle pain, fever, periorbital edema, eosinophilia, occasional CNS or cardiac damage Similar cycle (as humans) in swine or other carnivores (rats, bears, foxes, dogs, or horses) CHAPTER 238 are highest near tendon insertions in symptomatic muscles. The fresh muscle tissue should be compressed between glass slides and examined microscopically (Fig. 238-2) because larvae may be missed by exami nation of routine histopathologic sections alone. Trichinellosis and Other Tissue Nematode Infections
TREATMENT Trichinellosis Most lightly infected patients recover uneventfully with bed rest, anti pyretics, and analgesics. Glucocorticoids like prednisone (Table 238-1) are beneficial for severe myositis and myocarditis. Mebendazole and albendazole are active against enteric stages of the parasite, but their efficacy against encysting/encysted larvae has not been conclusively demonstrated. FIGURE 238-2 Trichinella larva encysted in a characteristic hyalinized capsule in striated muscle tissue. (CDC/Wadsworth Center, New York State Department of Health.)

TABLE 238-1 Therapy for Tissue Nematode Infections INFECTION SEVERITY TREATMENT Trichinellosis Mild Supportive Moderate Albendazole (400 mg bid × 10–14 days) or Mebendazole (200–400 mg tid × 3 days, then 500 mg tid × 10 days) Severe Add glucocorticoids (e.g., prednisone, 1 mg/kg qd × 5 days) Visceral larva migrans Mild to moderate Supportive Severe Glucocorticoids (as above) Ocular Not fully defined; albendazole (800 mg bid for adults, 400 mg bid for children) with glucocorticoids × 5–20 days has been effective Cutaneous larva migrans Ivermectin (single dose, 200 µg/kg) or Albendazole (200 mg bid × 3 days) Angiostrongyliasis Mild to moderate Supportive Not fully defined: albendazole (15 mg/kg per day in two divided doses × 14 days) with glucocorticoids (as above) Severe Not fully defined: albendazole (15 mg/kg per day in two divided doses × 14 days) with glucocorticoids (as above) Gnathostomiasis Ivermectin (200 µg/kg per day × 2 days)a or Albendazole (400 mg bid × 21 days)a aThese agents may have efficacy for cutaneous gnathostomiasis; their use in neurologic and ocular gnathostomiasis is less certain and may be detrimental. Steroids are often given during neurologic and ocular gnathostomiasis. PART 5 Infectious Diseases Prevention Larvae are usually killed by cooking pork until it is no longer pink or by freezing it at −15°C for 3 weeks. However, Arctic T. nativa larvae in walrus or bear meat are relatively resistant and may remain viable despite freezing. ■ ■VISCERAL AND OCULAR LARVA MIGRANS Visceral larva migrans is a syndrome caused by nematodes that are normally parasitic for nonhuman host species. In humans, these nematode larvae do not develop into adult worms but instead migrate through host tissues and elicit eosinophilic inflammation. The

most common form of visceral larva migrans is toxocariasis due to larvae of the canine ascarid Toxocara canis; the syndrome is due less commonly to the feline ascarid T. cati and even less commonly to the pig ascarid Ascaris suum. Rare cases with eosinophilic meningoencephalitis have been caused by the raccoon ascarid Baylisascaris procyonis. Life Cycle and Epidemiology The canine roundworm T. canis is distributed among dogs worldwide. Ingestion of infective eggs by dogs is followed by liberation of Toxocara larvae, which penetrate the gut wall and migrate intravascularly into canine tissues, where most remain in a developmentally arrested state. During pregnancy, some larvae resume migration in bitches and infect puppies prena tally (through transplacental transmission) or after birth (through suckling). Thus, in lactating bitches and puppies, larvae return to the intestinal tract and develop into adult worms, which produce eggs that are released in the feces. Eggs must undergo embryonation over several weeks to become infectious. Humans acquire toxocariasis mainly by ingesting soil/sand—such as found in public parks and playgrounds— contaminated by dog or cat feces that contains infective T. canis eggs. Pathogenesis and Clinical Features Clinical disease most com monly afflicts preschool children. After humans ingest Toxocara eggs, the larvae hatch and penetrate the intestinal mucosa, from which they are carried by the circulation to a wide variety of organs and tissues. The larvae invade the liver, lungs, central nervous system (CNS), and other sites, provoking intense local eosinophilic granulomatous responses. The degree of clinical illness depends on larval number

and tissue distribution, reinfection, and host immune responses. Most light infections are asymptomatic and may be evidenced only by blood eosinophilia. Characteristic symptoms of visceral larva migrans include fever, malaise, anorexia and weight loss, cough, wheezing, and rashes. Hepatosplenomegaly is common. These features may be accom panied by extraordinary peripheral eosinophilia at levels that may approach 90%. Uncommonly, seizures or behavioral disorders develop. Rare deaths are due to severe neurologic, pneumonic, or myocardial involvement. The ocular form of the larva migrans syndrome occurs when Toxocara larvae invade the eye. An eosinophilic granulomatous mass, most commonly in the posterior pole of the retina, develops around the entrapped larva. The retinal lesion can mimic retinoblastoma in appearance, and mistaken diagnosis of the latter condition can lead to unnecessary enucleation. The spectrum of eye involvement also includes endophthalmitis, uveitis, and chorioretinitis. Unilateral visual disturbances, strabismus, and eye pain are the most common presenting symptoms. In contrast to visceral larva migrans, ocular toxocariasis usually develops in older children or young adults with no history of pica; these patients seldom have eosinophilia or visceral manifestations. Diagnosis In addition to eosinophilia, leukocytosis and hypergam maglobulinemia may be evident in visceral larva migrans; these attri butes are often absent in ocular larva migrans. Transient pulmonary infiltrates are apparent on chest x-rays of about one-half of patients with symptoms of pneumonitis. The clinical diagnosis can be con firmed by an enzyme-linked immunosorbent assay for toxocaral anti bodies, although this assay may be negative in ocular larva migrans. Stool examination for parasite eggs is worthless in toxocariasis, since the larvae do not develop into egg-producing adults in humans. TREATMENT Visceral and Ocular Larva Migrans The vast majority of Toxocara infections are self-limited and resolve without specific therapy. In patients with severe myocardial, CNS, or pulmonary involvement, glucocorticoids may be employed to reduce inflammatory complications. Available anthelmintic drugs, including mebendazole and albendazole, have not been shown conclusively to alter the course of larva migrans, although most individuals with moderate to severe visceral larva migrans receive albendazole with concomitant steroids (Table 238-1). Treatment of ocular disease is not fully defined, but the administration of steroids (to

decrease ocular inflammation) and albendazole has been effec tive. Control measures include prohibiting dog excreta in public parks and playgrounds, deworming dogs, and preventing pica in children. ■ ■CUTANEOUS LARVA MIGRANS Cutaneous larva migrans (“creeping eruption”) is a serpiginous skin eruption caused by burrowing larvae of animal hookworms, usually the dog and cat hookworm Ancylostoma braziliense. The larvae hatch from eggs passed in dog and cat feces and mature in the soil. Humans become infected after skin contact with soil in areas frequented by dogs and cats. Cutaneous larva migrans is prevalent among children and travelers in regions with warm humid climates. After larvae penetrate the skin, erythematous lesions form along the tortuous tracks of their migration along the dermal-epidermal junction; the larvae advance several centimeters in a day. The intensely pruritic lesions may occur anywhere on the body and can be numerous if the patient has lain on the ground. Vesicles and bullae may form. The animal hookworm larvae do not mature in humans and, without treatment, will die after an interval ranging from weeks to a couple of months, with resolution of skin lesions. The diagnosis is made on clinical grounds. Skin biopsies only rarely detect diagnos tic larvae. Symptoms can be alleviated by ivermectin or albendazole (Table 238-1).

2 weeks 3rd-stage larvae (consumed in snail or slime) penetrate gut, go to CNS (then lung in rat) Larvae consumed by land snail/slug (Achatina fulica) FIGURE 238-3 Life cycle of Angiostrongylus cantonensis (rat lung worm) found in Southeast Asia and the Pacific Basin as well as on Caribbean islands, in countries of Central and South America, and in the southern United States. CNS, central nervous system. (Reproduced with permission from RL Guerrant et al [eds]: Tropical Infectious Diseases: Principles, Pathogens and Practice, 2nd ed. Elsevier, 2006.) ■ ■ANGIOSTRONGYLIASIS Angiostrongylus cantonensis, the rat lungworm, is the most common cause of human eosinophilic meningitis (Fig. 238-3). Life Cycle and Epidemiology This infection occurs principally in Southeast Asia and the Pacific Basin (including Hawaii) but has spread to other areas of the world, including the Caribbean islands, countries in Central and South America, and the southern United States. A. cantonensis larvae produced by adult worms in the rat lung migrate to the gastrointestinal tract and are expelled with the feces. They develop into infective larvae in land snails and slugs. Humans acquire the infection by ingesting raw infected mollusks; vegetables con taminated by mollusk slime; or crabs, freshwater shrimp, and certain marine fish that have themselves eaten infected mollusks. The larvae then migrate to the brain. Pathogenesis and Clinical Features The parasites eventually die in the CNS, but not before initiating pathologic consequences that, in heavy infections, can result in permanent neurologic sequelae or death. Migrating larvae cause marked local eosinophilic inflammation and hemorrhage, with subsequent necrosis and granuloma formation around dying worms. Clinical symptoms develop 2–35 days after the ingestion of larvae, typically at about 2 weeks. Patients usually present with an insidious or abrupt excruciating frontal, occipital, or bitempo ral headache. Neck stiffness, nausea and vomiting, and paresthesias are also common. Fever, cranial and extraocular nerve palsies, seizures, paralysis, and lethargy are uncommon. Laboratory Findings Examination of cerebrospinal fluid (CSF) is mandatory in suspected cases and usually reveals an elevated opening pressure, a white blood cell count of 150–2000/μL, and an eosinophilic pleocytosis of >20%. The protein concentration is usually elevated and the glucose level normal. The larvae of A. cantonensis are only rarely seen in CSF. Peripheral-blood eosinophilia may be mild. The diagnosis is generally based on the clinical presentation of eosinophilic menin gitis together with a compatible epidemiologic history. Serologic tests are sometimes available in endemic regions, although they have not been standardized and may be negative at onset of clinical symptoms. A polymerase chain reaction assay that detects A.

cantonensis DNA in CSF or other tissues is available, including through the U.S. Centers for Disease Control and Prevention (CDC) and the Hawaii Department of Public Health in the United States.

Eosinophilic meningitis Adult in pulmonary artery produces fertile eggs; larvae hatch, penetrate arterioles, migrate up bronchi, and are coughed up, swallowed, and passed in feces viable in fresh water CHAPTER 238 TREATMENT Angiostrongyliasis Many individuals with eosinophilic meningitis from angiostron gyliasis improve with supportive therapy alone. Steroids are often given to decrease inflammation. Specific chemotherapy is of uncertain benefit and should never be given without steroids to prevent exacerbation of inflammatory brain lesions; when given, albendazole is usually employed. (Table 238-1). Repeated lumbar punctures with removal of CSF can assist with symptom relief. In most patients, cerebral angiostrongyliasis has a self-limited course, and recovery is complete. The infection may be prevented by adequately cooking snails, crabs, and prawns and inspecting vegetables for mollusk infestation. Other parasitic or fungal causes of eosinophilic meningitis in endemic areas may include gnathos tomiasis (see below), paragonimiasis (Chap. 241), schistosomiasis (Chap. 241), neurocysticercosis (Chap. 242), and coccidioidomy cosis (Chap. 219). Trichinellosis and Other Tissue Nematode Infections
A separate species of Angiostrongylus, A. costaricensis, can inhabit the mesenteric arteries of humans, causing an eosinophilic inflam matory mass in abdominal viscera. A. costaricensis is a zoonosis (rat–mollusk/slug life cycle) found in the New World, especially Latin America, and occurs following ingestion of slugs or raw produce contaminated by slug slime. Diagnosis is most commonly made at the time of surgical excision, which is often curative. ■ ■GNATHOSTOMIASIS Infection of human tissues with larvae of Gnathostoma spinigerum can cause eosinophilic meningoencephalitis, migratory cutaneous swell ings, or invasive masses of the eye and visceral organs. Life Cycle and Epidemiology Human gnathostomiasis occurs in many countries and is notably endemic in Southeast Asia and parts of China and Japan. In nature, the mature adult worms parasitize the gastrointestinal tract of dogs and cats. First-stage larvae hatch from eggs passed into water and are ingested by Cyclops species (water fleas). Infective third-stage larvae develop in the flesh of many animal species (including fish, frogs, eels, snakes, chickens, and ducks) that have eaten either infected Cyclops or another infected second intermediate host. Humans typically acquire the infection by eating raw or undercooked

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