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dysrhythmias, hypotension, and pulmonary edema have also been reported. Postmortem examination of brain tissue has shown neuronal necrosis or cell loss and astrocytosis, most prominently in the hippo campus and amygdala. Several months after the primary intoxication, victims may still display chronic residual memory deficits and motor or sensory neuropathy.

TREATMENT Amnesic Shellfish Poisoning Therapy is supportive and based on symptoms. IV fluids and antiemetics may be used for severe nausea, vomiting, and diar rhea. Domoic acid neurotoxicity is primarily seizure mediated; anticonvulsive therapy using GABA agonists (e.g., benzodiazepines, propofol, or barbiturates) should be instituted early. However, some patients without clinically evident seizure activity have developed neurologic sequelae. ■ ■DIARRHETIC SHELLFISH POISONING Diarrhetic shellfish poisoning occurs with consumption of shellfish containing the lipophilic compound okadaic acid. This toxin inhibits serine and threonine protein phosphatases, with consequent protein accumulation and continued secretion of fluid by intestinal cells lead ing to diarrhea. Shellfish acquire these toxins by feeding on dinoflagel lates, particularly of the genera Dinophysis and Prorocentrum. PART 14 Poisoning, Drug Overdose, and Envenomation Symptoms include diarrhea, nausea, vomiting, abdominal pain, and chills. Onset typically occurs between 30 min and 12 h after inges tion of contaminated shellfish. The illness is usually self-limited; most patients recover in 3–4 days and only a few require hospitalization. Treatment is supportive and focused on hydration. Toxins can be detected in food samples by a mouse bioassay, an immunoassay, and fluorometric HPLC. Acknowledgment Kirsten B. Hornbeak, Robert L. Norris, Alex Chen, and Charles Lei contributed to this chapter in the prior edition and material from that chapter has been retained here. We would like to dedicate this chapter to the late Dr. Paul S. Auerbach, who was a contributing author for the previous seven editions of Harrison’s Principles of Internal Medicine. Dr. Auerbach had a tremendous impact on the field of emergency medi cine and founded the subspecialty of wilderness medicine. Dr. Auerbach was a wonderful teacher, mentor,

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Ectoparasite Infestations

and Arthropod Injuries Ectoparasites include arthropods and creatures from other phyla that infest the skin or hair of animals; the host animals provide them with sustenance and shelter. The ectoparasites may remain on the superficial surfaces of the skin or hair, attached by mouthparts or with specialized claws. Other ectoparasites may penetrate the skin’s surface and reside in the epidermis, dermis, or subcutaneous tissues. Ectoparasites may inflict direct mechanical injury, consume blood or nutrients, induce hypersensitivity reactions, inoculate toxins, transmit pathogens, create openings in the skin for secondary bacterial infection, and incite fear or disgust. Human beings are obligate hosts for few ectoparasites but can serve as facultative, dead-end, or paratenic (accidental) hosts for many others. Of the organisms discussed in this chapter, only scabies mites (the hominis variety) and human-infesting lice are obligate parasites of humans. Arthropods that are capable of ectoparasitism or that can otherwise cause injury include insects (such as lice, fleas, bed bugs, wasps, ants, bees, and diverse kinds of flies), arachnids (spiders, scorpions, mites, and ticks), and myriapods (millipedes and centipedes). Several arthro pods can cause uncomfortable reactions when they, their setae (hairlike growths), or their exudates come into contact with skin, mucous membranes, or ocular tissues. Certain nematodes (helminths), such as the hookworms (Chap. 239), are ectoparasitic in that they must enter the skin in some manner, then traverse the skin to reach deeper tissues, or migrate within the skin. Infrequently encountered ectoparasites in other animal phyla include pentastomes (armillifers or tongue worms) and leeches.

Arthropods may cause injury when they attempt to take a blood meal or as they defend themselves by biting, stinging, or exuding ven oms. Papular urticaria and other lesions caused by arthropod bites and stings are so diverse and variable (depending on the host's health status and prior exposure to the arthropod's saliva, venom, or other exu dates) that it is difficult to identify the precise causative organism with visual examination alone without a bona fide specimen and taxonomic expertise. Specimens of the presumably offending arthropod should, whenever possible, be sampled directly (taken from the patient, ideally by medical personnel) or indirectly by using traps or other monitoring devices in the patient's home or workplace. Samples sent to laboratories for identification should be properly fixed, preserved, and packaged. The patient's history of travel and precautionary behaviors; occupa tional, recreational, and environmental exposures; and proximity to animals often helps the clinician and parasitologist resolve the cause.

■ ■SCABIES The human itch mite, Sarcoptes scabiei var. hominis, is an obligate human ectoparasite and a common cause of itchy dermatosis, affecting ~250 million persons worldwide. Both the mites and the skin condition are called scabies. Gravid female mites (~0.3 mm in length) burrow superficially within the stratum corneum, depositing several eggs per day. Gravid adult females emerge to the skin's surface about 8 days later, then (re)invade the skin of the same person or another host. Newly fertilized female mites are transferred between people mainly by direct skin-to-skin contact. Transmission is facilitated by crowding, poor hygiene, and close physical contact with other people. Generally, sca bies mites die within a day or so if not on a suitable host. Transmission by sharing contaminated bedding or clothing occurs less frequently than commonly thought. Outbreaks are known to occur in preschools, hospitals, nursing homes, prisons, other institutional residences, and other congregate settings. The rash and pruritus associated with scabies arise from a sensitiza tion reaction to mites and their secretions/excretions. A person's initial infestation typically remains asymptomatic for up to 6 weeks before intense pruritus starts. Reinfestation, however, promptly induces a hypersensitivity reaction. Burrows become surrounded by inflamma tory infiltrates composed of eosinophils, lymphocytes, and histiocytes. Infested individuals often feel generalized pruritus, not just in the most heavily involved areas. Pruritus typically intensifies at night and after hot showers. Burrows appear as dark wavy lines in the upper epidermis and are 3–15 mm long (Fig. 472-1). Classic burrows are often difficult to find because most patients have perhaps only 10–15 burrows. However, most bur rows are obscured by excoriations or secondary bacterial infections. Scabietic lesions are most common on the volar wrists and along the digital web spaces. In males, the genitalia (penile glans and shaft and the scrotum) are nearly always involved. Small papules and vesicles, often accompanied by eczematous plaques, pustules, or nodules, appear symmetrically at those sites. The axillae and other intertrigi nous areas; around the navel and belt line; and the buttocks and upper thighs are also common sites. Except in infants, the face, scalp, neck, palms, and soles are usually spared. Hyperinfestation with thousands of mites, a condition known as crusted scabies (formerly Norwegian scabies), may result from gluco corticoid use, immunodeficiency (including that due to HIV/AIDS), and neurologic or psychiatric disorders that dampen the itch or impair the scratch response. Crusted scabies often resembles psoriasis: both are characterized by widespread thick yellowish-white keratotic crusts, scaly plaques, and dystrophic nails. Characteristic burrows are not seen in crusted scabies, and patients are often not itchy, even though their infestations are highly contagious and have been responsible for outbreaks of common scabies in hospitals. FIGURE 472-1 Scabies burrows. Scabies mites create short, delicate, linear burrows within the superficial epidermis. Although burrows are pathognomonic for scabies, most have been altered by scratching

or secondary bacterial infection.

Scabies should be in the differential diagnosis for patients with pruritus and symmetric superficial, excoriated, papulovesicular skin lesions in characteristic locations, particularly if they have had direct and prolonged contact with an infested person. The diagnosis can be confirmed by microscopic examination of material scraped from bur rows. Burrows should be sought and unroofed with a sterile needle or scalpel blade, and the scrapings should be examined microscopically for mites, eggs, and fecal pellets. Examination of skin scrapings; biopsy specimens; material obtained by clear cellophane tape or cyanoacrylate adhesive lifted from lesions; dermatoscopic imaging of papulovesicular lesions; and microscopic inspection of clear cellophane tape lifted from lesions also may be diagnostic. In the absence of identifiable mites or eggs, a clinical diagnosis is based on a triad of pruritus, findings on physical examination, and an epidemiologic link. Unrelated skin dis eases are frequently misdiagnosed as scabies, particularly in presumed “outbreak” situations. Sarcoptes mites of other mammals may cause transient irritation, but they do not reside or reproduce in human hosts. In some Australian Aboriginal communities, household dogs may serve as reservoirs for human scabies mites.

TREATMENT Scabies CHAPTER 472 The U.S. Food and Drug Administration (FDA) has approved four scabicides: permethrin, crotamiton, spinosad, and lindane. They are topical products and available by prescription only. Permethrin cream (5%) is less toxic than 1% lindane preparations and is effec tive against lindane-resistant infestations. In adults, scabicides are applied thinly but thoroughly from the jawline to toes after bathing, with careful application to interdigital spaces, the navel, and under the nails. and washed off 6–14 h later with soap and water. These products are relatively ineffective against scabies eggs, so a second round of treatment 1 week later is advisable. Ectoparasite Infestations and Arthropod Injuries Treating crusted scabies is difficult and requires repeated courses of both topical (permethrin) and oral (ivermectin) agents. Pre application of a keratolytic agent, such as 6% salicylic acid, will help debulk the crusts. Permethrin should be applied to the skin’s entire surface, including the scalp, face, and ears. Oral ivermectin is not FDA-approved for treating scabies but is approved to treat the nematodal diseases hookworm and strongyloidiasis. A single oral dose of ivermectin (200 μg/kg) is effective in healthy patients with common (noncrusted) scabies. Patients with crusted scabies require three to seven doses of ivermectin over 8–30 days, along with repeated applications of topical permethrin and possibly a keratolytic compound. Within 1 day of properly administered treatment, a scabies infes tation is considered noncommunicable, thereby permitting a patient to return to work, school, and other public activities. Nevertheless, remind patients that dead mites and their detritus may continue to produce the pruritic hypersensitivity dermatitis for several weeks. Unnecessary retreatment with topical agents, especially permethrin cream, may cause an irritant contact dermatitis. Topical emollients and antipruritic agents, menthol and methyl salicylate products, calamine lotion, and oral antihistamines relieve itching during treatment. Topical glucocorticoids may calm pruritus that lingers after effective treatment. To prevent reinfestations, bedding and clothing should be washed and dried on high heat or heat-pressed, and other environmental surfaces or potential sources of fomites should be cleaned. Household members and other close contacts of confirmed cases, even if asymptomatic, should be treated simulta neously to help prevent back-and-forth reinfestations. Scabies infestations often lead to secondary bacterial infections, usually with Staphylococcus aureus, Streptococcus pyogenes, or both. Consequences of superinfections include impetigo, cellulitis, inva sive bacterial infections, poststreptococcal glomerulonephritis (and subsequent

kidney disease), and possibly acute rheumatic fever (and subsequent cardiac valvular disease).

■ ■CHIGGERS AND OTHER BITING MITES Chiggers are the larvae of trombiculid (harvest) mites that feed mostly on mice and other small vertebrates in grassy or brush-covered sites in tropical, subtropical, and (less frequently) temperate areas during warm months. They reside on low vegetation and attach themselves to passing vertebrate hosts. While feeding, larvae secrete proteolytic saliva that penetrates the epidermis, creating a tube-like invagination, called a stylostome, in the host's skin. The stylostome allows the mite to imbibe tissue fluids. The saliva is highly antigenic and causes small (usually <1 cm in diameter) but exceptionally itchy papular, urticarial, or pustulovesicu lar lesions. In people already sensitized to salivary antigens, the papules develop within hours of attachment. While attached, chiggers appear as minute (~0.5 mm diameter) red dots on the skin. Generally, lesions have a hemorrhagic base and are slightly elevated, resembling purpuric papules seen in cutaneous small-vessel vasculitis.

Chiggers remain on their host animals for several days before fall ing to the ground to complete the nonparasitic stages of their life cycle. However, in humans, the intense pruritus leads to scratching that invariably destroys the chigger before it completes feeding, although the itching and burning often persist for weeks. The rash is common on the ankles and along the belt line and in areas where circumferentially tight clothing obstructs the further wanderings of the mites. Topical repellents on one's skin and insecticide-treated clothing are useful for preventing chigger bites. PART 14 Poisoning, Drug Overdose, and Envenomation Chiggers (primarily Leptotrombidium species) serve as vectors for intracellular rickettsial organisms in the genus Orientia that cause scrub typhus. Scrub typhus caused by Orientia tsutsugamushi was traditionally confined to the eastern half of Asia and the Indomalayan and Australasian regions. Their bites may be asymptomatic. Endemic foci of closely related Orientia species have recently been identified in southern Chile, East Africa, and the Arabian peninsula, where they cause scrub typhus-like illnesses. Additional areas of transmission will likely be found outside the usual endemic regions. Only larval trombi culids are predatory on mammals, and the larvae acquire their Orientia load from transovarial transmission from their mother mites. Many kinds of mites feed on peridomestic birds or rodents and become particularly bothersome when they invade homes and bite people. In North America, the northern fowl mite, chicken mite, tropical rat mite, and house mouse mite normally feed on poultry, other birds, and small mammals. After their natural hosts leave the nest or die, the mites disperse and may invade homes. Although the mites are rarely seen because of their small size, their bites can be painful and pruritic. House mouse mites (Liponyssoides sanguineus) serve as vectors for the agent of rickettsialpox, Rickettsia akari, an uncommon disease characterized by mild fevers, an eschar at the bite site, and a papulovesicular eruption. Rickettsialpox (Chap. 192) has been recognized mainly in large northern temperate urban areas. Once these environmental mites are confirmed as causing pruritic eruptions, they are best eliminated by excluding their animal hosts, removing their nests, and cleaning and treatment of the nesting area with appropriate acaricides. Pyemotes mites are external parasites of insect larvae and commonly occur in insect-infested products such as grain, straw, cheese, hay, and oak leaf galls. Their saliva contains a potent neurotoxin they use to immobilize their prey. The straw itch mite (Pyemotes tritici) occurs worldwide and is an occupational hazard for agriculture workers and others who work with dry plant material. Bites in humans are associ ated with pruritic rashes and may produce a unique dermatologic "comet sign" lesion—a paisley-shaped urticarial plaque (Fig. 472-2). The oak leaf gall mite (Pyemotes herfsi) has been associated with several outbreaks of

dermatosis in the central and eastern United States. These mites feed on oak gall midges and periodic cicada eggs in oak trees, and their populations fluctuate widely each year based on host availability. In the late summer and fall, mites drop or are wind-blown from oak trees and may land on a person. Bites in people are usually reported on exposed surfaces, such as neck, face, arms, and upper torso. Intense itching is reported 10–16 h after the initial bite, which most people do not recall. Diagnosis of mite-induced dermatitis (including those caused by chiggers) relies on confirmation of the mite's identity or elicitation of

a history of exposure to the mite's source. Unlike scabies mites that burrow and live in one's skin, environmental mites do not reside on humans. Therefore, treatment of the patient with acaricides (e.g., per methrin) is discouraged. Oral antihistamines or topical steroids may reduce mite-induced pruritus temporarily. The mites that cause house dust–related allergic conditions neither bite nor infest humans. ■ ■TICK BITES AND TICK PARALYSIS Ticks attach superficially to skin and usually feed painlessly; blood is their only food. Their salivary secretions are biologically active, pre venting the host's blood from coagulating while the tick feeds. Tick saliva can transmit various pathogens and can induce several "sterile reactions" in the host, such as a local inflammatory response, fevers, and tick-bite paralysis. The two main families of ticks are the hard ticks (Ixodidae) and soft ticks (Argasidae). Because no ticks are obli gate parasites on humans, all tick-borne diseases (bacterial, viral, and protozoal) are zoonotic. Generally, soft (argasid) ticks feed quickly, attaching to a host, com pleting a meal in <1 h, and then dropping off the host. Because of their rapid feeding habits, soft ticks are not carried widely by their vertebrate hosts. Soft tick–associated infections usually have fairly focal distribu tions. On humans, red macules may develop at the bite site. Some spe cies in Africa, the western United States, and Mexico produce painful hemorrhagic lesions. Hard (ixodid) ticks are much more common than soft ticks, and they transmit most tick-borne infections that are familiar to physicians, patients, and the public. Hard ticks attach to the host and feed for sev eral days to >1 week, with the exact duration depending upon the tick's species and stage of development. At the site of hard-tick bites, small areas of induration, often purpuric, develop and may be surrounded by an erythematous rim. A necrotic eschar, called a tâche noire ("black spot"), occasionally develops. Long-lasting dermal nodules, called persistent tick-bite granulomas, occasionally appear where the host is bitten. The nodules are roughly 1–3 cm in diameter and may linger for months after the feeding tick has fallen off or been removed. The granulomas can be treated with injected intralesional glucocorticoids, by simple local excision, or simply left to resolve on their own over perhaps a year or more. Tick-induced fever, unassociated with any pathogen, is often accompanied by headache, nausea, and malaise but usually resolves ≤36 h after the tick is removed. Tick bites are also associated with a recently recognized allergic condition known as alpha-gal syndrome (AGS) or red meat allergy. This syndrome occurs in humans when exposure to tick saliva induces production of IgE antibodies that cross-react with a carbohydrate mol ecule, galactose-α-1,3-galactose (alpha-gal), found in muscle tissues of all mammals except humans, other apes, and Old World monkeys. In the United States, AGS is attributed to bites from the lone star tick (Amblyomma americanum). The geographic distribution of human cases closely aligns with that tick's geographic range. In Australia and Europe, AGS is associated with Ixodes spp., and in Asia, it is associated with Haemaphysalis longicornis. It is important for individuals with AGS to avoid additional tick bites as this may prolong or worsen their allergic reactions. Tick paralysis, an acute ascending flaccid paralysis, is believed to be caused by one or more unidentified toxins in tick saliva that block neuromuscular transmission and decrease nerve conduction. This rare complication has been associated with >70 species of ticks. Although it

has been reported worldwide, most cases occur in the Rocky Moun tain region, in northwestern United States and southwestern Canada, and along Australia's eastern seaboard. In North America, dog and wood ticks (Dermacentor species) are most commonly implicated. Weakness begins several days after the tick attaches to the host. The Guillain-Barré-like paralysis starts in the lower legs and ascends sym metrically over several days. The paralysis may culminate in complete paralysis of the extremities and cranial nerves. Deep tendon reflexes are diminished or absent, but sensory examination and findings on lumbar puncture are typically normal. Diagnosis and treatment depend on finding the tick, which is often hidden beneath scalp hair. Removal

FIGURE 472-2 Comet signs in individuals with known or suspected mite-bite reactions, likely due to Pyemotes species. Note central punctum at bite site, surrounded by edematous erythema. Linear or serpiginous "comet tails" emanate from the central site. Pyemotes-induced comet tails generally do not follow typical patterns of ascending lymphatic drainage. of the tick generally leads to improvement within a few hours and com plete recovery after several days, although the patient's condition may continue to deteriorate for a full day. Failure to remove the tick may lead to dysarthria, dysphagia, and ultimately death from aspiration or respiratory paralysis. Removal of hard ticks during the first 24 h of attachment gener ally prevents transmission of the agents of Lyme disease, babesiosis, anaplasmosis, and ehrlichiosis, although tick-borne viruses may be transmitted more quickly. Ticks should be removed by traction with fine-tipped forceps placed firmly around the tick's mouthparts where they enter the skin. Careful handling (to avoid rupture of ticks) and use of gloves may avert accidental contamination with pathogens contained in the tick's body fluids. Attempting to induce the tick to detach by applying heat or occlusive compounds or dressings merely delays tick removal. After removal, the site of attachment should be disinfected. Tick mouthparts sometimes remain in the skin but gener ally are shed spontaneously within days without the need for surgical removal. Current guidelines from the Centers for Disease Control and Prevention suggest that, rather than awaiting the onset of erythema migrans, the results of tick testing, or seroconversion to antigens diagnostic for Lyme disease, physicians may appropriately administer prophylaxis—a one-time oral dose of doxycycline (200 mg) within 72 h of tick removal—to adult patients with bites thought to be from Ixodes scapularis (black-legged or deer ticks) in Lyme disease–endemic areas. Whereas antibiotic prophylaxis may help prevent Lyme disease, it is not recommended as a way to prevent other tick-borne infections.

CHAPTER 472 Ectoparasite Infestations and Arthropod Injuries The Asian longhorned tick (Haemaphysalis longicornis) is a newly invasive species in the United States, first detected in the northeast ern states in 2017. Although it carries several pathogens to domestic animals, wildlife, and humans in its natural range (northeastern Asia), it has not yet been implicated in disease transmission in the United States. ■ ■LOUSE INFESTATION (PEDICULOSIS AND PTHIRIASIS) Three kinds of biting lice are obligate blood-feeding ectoparasites of human beings. These include the human head louse and the human body louse that represent distinct genetic clades of Pediculus humanus, and the pubic ("crab") louse (Pthirus pubis). Nymphs and adults of these lice feed at least once a day, ingesting human blood exclusively, and they partition ecologically on the host. Head lice infest mainly the scalp and scalp hair; body lice infest clothing; and pubic lice infest mainly pubic hair. The saliva of lice produces a pruritic morbilliform or urticarial rash in some sensitized persons. Female head lice and pubic lice cement their eggs (nits) firmly to human hair, whereas female body lice cement their eggs to clothing, particularly to threads along cloth ing seams. After ~10 days of development within the egg, a nymph emerges. Empty eggs may remain

affixed to hair for months thereafter. Body lice are acquired by direct contact with an infested person or their recently used clothing or bedding. These lice venture for just minutes to the skin to feed, but otherwise sequester on clothing. They generally succumb in ≤2 days if separated from their host. In developed

countries, body lice are generally uncommon, found only on indigent persons who have relevant exposure and lack the wherewithal or desire to change or appropriately launder their clothing and bedding.

Body lice are vectors for the agents of louse-borne (epidemic) typhus (Chap. 192), louse-borne relapsing fever (Chap. 190), and trench fever (Chap. 177). Body lice and their associated diseases may proliferate whenever societal upheaval and disasters limit access to clean clothes or laundry facilities. This scenario was exacerbated during the trench warfare of World War I, when troops often wore one uniform for weeks or months at a time. During damp, cold months, entrenched soldiers huddled together to stay warm, enabling body lice and their pathogens to spread easily from person to person. Infestations result in a postinflammatory hyperpigmentation and thickening of the skin known as vagabond's disease. When inspecting a patient for signs of a body lice infestation, one must examine their clothing seams for nits and live lice. Head lice are acquired mainly by direct head-to-head contact rather than via fomites such as shared hats, caps, headgear, bed linens, and grooming implements. Worldwide, the prevalence of head lice varies widely as a function of age, geography, and cultural habits. In North America, the overall prevalence of head lice infestation is generally low, but most communities, irrespective of socioeconomic circum stances, experience episodic high-prevalence focal outbreaks among younger school-aged children. Although these events generally spare adults, they can create considerable distress among families in those communities. PART 14 Poisoning, Drug Overdose, and Envenomation An infested person generally hosts 10 or fewer head lice, but the nits, even when empty or nonviable, remain cemented to one's scalp hairs as they grow. Thus, accumulated nits make it seem that a person has hundreds of head lice, even after successful treatment. Consequently, many schools retain unnecessarily onerous "no nit" policies for school attendance, even though a child has been treated successfully. Bite reactions appear as small, <1 cm diameter edematous pink wheals and are most evident along the posterior hairline. Pruritus, attributed to hypersensitivity to the louse's saliva, leads to scratching, which may lead to secondary impetigo with S. aureus or S. pyogenes. Body lice are well known to transmit diseases, but can head lice (besides superficial secondary bacterial infections)? Several studies using polymerase chain reaction on freshly caught head lice have detected pathogenic bacteria, but clinical and epidemiologic data are generally interpreted to show that head lice are not natural vectors of any disease. On the other hand, experimental models suggest that head lice may have some competence as vectors for Rickettsia prowazekii (louse-borne typhus) and Bartonella quintana (trench fever). Indeed, sporadic reports indicate that cycles of trench fever are maintained in Ethiopia and elsewhere in Africa by head lice in the absence of body lice. Crab or pubic lice are found mostly on pubic hair and are transmit ted mainly by direct sexual contact. They also occur uncommonly on axillary or facial hair, including the eyelashes, as the result of either sexual or close nonsexual contact. Intensely pruritic, bluish mac ules <5 mm in diameter (maculae ceruleae) may develop at bite sites. Blepharitis commonly accompanies infestations of the eyelashes. Eye lash infestations in children should not be construed as incontrovert ible evidence of inappropriate sexual contact. Pediculosis is often suspected upon the detection of presumed nits to hairs or simply on the basis of an itchy scalp.

Objects presumed to be louse eggs are often pseudo-nits composed of debris, flakes of dry skin, and hair-associated fungi. Hatched and dead eggs remain firmly affixed to scalp hair for months. Such relics are frequently misconstrued to be signs of an active louse infestation. Confirmation of a louse infestation, therefore, should rely on the discovery of a live louse. TREATMENT Louse Infestation Body lice usually are eliminated by bathing and by changing to new or properly laundered clothes. Fabric is effectively deloused by heat,

such as in a clothes dryer at ≥55°C (≥131°F) for 30 min or by heatpressing. Emergency mass delousing may be warranted in congre gate settings during periods of civil strife and after natural disasters to reduce the high risk of disease transmission by body lice. Head lice and nits may be removed with a fine-toothed louse or nit comb, but this effort is time-consuming and often fails to eradi cate the lice. Treatment of newly identified, active infestations tra ditionally relies on a 10-min topical application of a FDA-approved 1% permethrin lotion or pyrethrin-based shampoo, which are available over the counter. Also FDA-approved, but requiring a pre scription, are 0.5% malathion lotion, 0.5% ivermectin lotion, and 0.9% spinosad suspension. These products do not kill viable nits, so a second application roughly 10 days later is required to clear newly hatched lice. Lice persisting after this treatment may be due to backand-forth reinfestations within a family, or the lice may be resistant to chemical treatment. Resistance to permethrin, malathion, and lindane is well documented, with less resistance associated with ivermectin and spinosad products. Although children infested by head lice—or those who simply have remnant nits from a previous infestation—are frequently isolated or excluded from school, this practice increasingly is considered to be unjustified, ineffective, and counterproductive. Pubic louse infestations are treated with topical pediculicides, except for eyelid infestations (pthiriasis palpebrum), which gener ally respond to a coating of petrolatum applied for 3–4 days. ■ ■MYIASIS (FLY INFESTATION) Myiasis refers to infestations by fly larvae (maggots) that invade living or necrotic tissues or body cavities and produce different clinical syn dromes, depending on the species of fly. In forested parts of Central and South America, larvae of the human botfly (Dermatobia hominis) produce furuncular (boil-like) dermal and subcutaneous nodules ≤3 cm in diameter. A gravid adult female botfly captures a mosquito or another bloodsucking insect and depos its her eggs on its abdomen. When the carrier insect attacks a human or another mammalian host (often cattle) several days later, the warmth and moisture of the host’s skin stimulate the eggs to hatch. The emerg ing larvae, ~1 mm long, promptly penetrate the skin. After 6–12 weeks of development, mature larvae emerge from the skin, drop to the ground, pupate, and finally metamorphose into adults. The African tumbu fly (Cordylobia anthropophaga, also called mango fly or mputsi fly) deposits its eggs on damp sand, leaf litter, dry ing laundry, or damp clothing contaminated by urine or sweat. Larvae hatch from eggs upon contact with a host’s body and penetrate the skin, producing boil-like lesions from which mature larvae emerge ~9–10 days later. Furuncular myiasis is suggested by uncomfortable lesions with a central breathing pore that emit bubbles when submerged in water. The sensation of movement within the patient’s skin may cause severe (and understandable) emotional distress (Fig. 472-3). Larvae that cause furuncular myiasis may be induced to emerge if the breathing pore is coated with petrolatum or another occlusive substance. Removal may be facilitated by injection of a local anesthetic (or sterile injectable saline) into the subjacent tissue to uplift the larva through the breathing pore. Surgical excision is sometimes necessary because upward-pointing spines of some species hold larvae firmly in place. Other fly larvae cause several forms of nonfuruncular myiasis. Lar vae of the horse botfly (Gasterophilus intestinalis) emerge from eggs that typically are laid on the hairs of a horse’s front legs. Direct contact with a person’s skin

may cause the eggs to hatch and the new larvae to invade human skin. After penetrating human skin, these larvae rarely mature but instead may migrate for weeks in the dermis. The resulting pruritic and serpiginous eruption resembles cutaneous larva migrans caused by canine or feline hookworms (Chap. 238). Larvae of rabbit and rodent botflies (Cuterebra species) occasionally cause cutaneous or tracheopulmonary myiasis. Larvae of the sheep botfly, Oestrus ovis, and other flies responsible for furuncular and wound myiasis also may cause ophthalmomyiasis. Sequelae include nodules in the eyelid, retinal detachment, and destruction of the globe.

FIGURE 472-3 Furuncular myiasis with larva of Cordylobia anthropophaga in the thigh of a teenage girl who recently returned from visiting relatives in East Africa (left). The central pore shows the respiratory elements of the embedded fly larva (right). Certain flies are attracted to blood and pus, laying their eggs on open or draining sores. Newly hatched larvae enter wounds or diseased skin. Larvae of several species of green bottle flies (Lucilia spp.) usually consume only necrotic tissues. Specially raised, sterile “surgical mag gots” are available as FDA-approved medical devices for debridement of chronic wounds, such as deeply infected diabetic ulcers. On the other hand, larvae of screwworm flies (Cochliomyia spp.) and flesh flies (Wohlfahrtia spp.) can invade viable tissues more deeply and cause large suppurating lesions. These can be extraordinarily harmful to domesticated herd animals. Larvae of the sheep botfly, Oestrus ovis, may cause nasal or external ophthalmomyiasis, involving tear ducts, eyelids, and conjunctivae. Larvae that infest decaying tissues may enter body cavities such as the mouth, nose, ears, sinuses, anus, vagina, and lower urinary tract, particularly in unconscious or otherwise debilitated patients. The con sequences range from harmless colonization to destruction of the nose, meningitis, and deafness. Treatment involves removal of maggots, surgical debridement of tissue, and treatment of secondary infections. Maggots are occasionally found in human feces, usually the result of a fly laying eggs on recently passed stools, and not as evidence of an intestinal maggot infestation. ■

■PENTASTOMIASIS Pentastomids (tongue worms), an obscure type of crustacean, inhabit respiratory passages of reptiles and carnivorous mammals. Human infestation by Linguatula serrata is common in the Middle East and results from eating encysted larval stages in raw liver or lymph nodes of sheep and goats, which are true intermediate hosts for the tongue worms. In areas where raw sheep and goat liver are served, pentasto mid larvae migrate to the person’s nasopharynx and produce an acute self-limiting syndrome—known as halzoun in Lebanon and marrara in Sudan—characterized by rapid onset (within <12 h) of pain and itching of the throat and ears, coughing, hoarseness, dysphagia, and dyspnea. Severe edema may cause obstruction that requires tracheostomy. In addition, ocular invasion has been described. Diagnostic larvae mea suring ≤10 mm in length appear in copious nasal discharge or vomitus. Another type of tongue worm, Armillifer armillatus, infects people who consume its eggs in contaminated food or drink or after han dling the definitive host, the African python. Larvae encyst in various organs, usually the liver or peritoneum, but rarely cause symptoms. Cysts may require surgical removal as they enlarge during worm molting, but they usually are encountered as an incidental finding at

CHAPTER 472 autopsy. Parasite-induced lesions may be misinterpreted as a malig nancy, with the correct diagnosis only confirmed histopathologically. Larva migrans–type syndromes caused by other pentastomes have been reported from Southeast Asia and Central America. Ectoparasite Infestations and Arthropod Injuries ■ ■LEECH INFESTATIONS Medically important leeches are annelid worms that attach to their hosts with chitinous cutting jaws and draw blood through muscular suckers. Medicinal leeches (Europe: Hirudo medicinalis and other Hirudo species; Asia:

Hirudinaria manillensis; North America: Macrob della decora) are still used occasionally for medical purposes to reduce venous congestion in surgical flaps or replanted body parts. This prac tice has been complicated by intractable bleeding, wound infections, myonecrosis, and sepsis due to Aeromonas hydrophila, which colonizes the gullets of commercially available leeches. Ubiquitous aquatic leeches that parasitize fish, frogs, and turtles readily attach to human skin—most often the nasal mucosa—and avidly suck blood. Attachment is usually painless, and the leeches will detach themselves when satiated with a blood meal. Hirudin, a pow erful anticoagulant secreted by the leech, causes continued bleeding after the leech has detached. Healing of a leech-bite wound is slow, and secondary bacterial infections are not uncommon. Several kinds of aquatic leeches in Africa, Asia, and southern Europe can enter the mouth, nose, and genitourinary tract and attach to mucosal surfaces at sites as deep as the esophagus and trachea. Leeches may detach on exposure to gargled saline or may be removed by forceps or medical suction. Arboreal land leeches, which live amid rain forest vegetation, are attracted by heat and can drop from a leaf onto one's skin. Externally attached leeches generally drop off after they have engorged, but removal is hastened by gentle scraping aside of the anterior and poste rior suckers the leech uses for attachment and feeding. Some authori ties dispute the wisdom of removing leeches with alcohol, salt, vinegar, insect repellent, a flame or heated instrument, or applications of other noxious substances. ■ ■SPIDER BITES All spiders are carnivores and most have venomous bites intended to immobilize and digest their prey. However, very few spiders are capable of biting humans, and the vast majority of putative spider bites have nothing to do with spiders. Of >45,000 recognized species of spiders, only about 60 are medically important to humans. In the United States,

only recluse spiders (Loxosceles spp.) and widow spiders (Latrodectus spp.) are considered medically important.

Most spider bites are painful but do not require medical attention. Identification of the offending spider is important because specific treatments exist. Except when the patient actually observes a spider bit ing them or fleeing from a site of sudden sharp pain, most acute tender noduloulcerative lesions reported as spider-bite reactions are due to an unrelated minor injury or caused by an acute bacterial infection, particularly by methicillin-resistant S. aureus (MRSA). Recluse Spider Bites and Necrotic Arachnidism Brown recluse spiders (Loxosceles reclusa) live mainly in the southcentral United States and have close relatives in Central and South America, Africa, the Mediterranean basin, and the Middle East. Recluse spi ders are not aggressive toward humans and bite only if threatened or pressed against the skin. They generally live beneath rocks and logs or in caves and animal burrows. They invade homes and seek dark and undisturbed hiding spots in closets, garages, crawl spaces, and attics; under furniture and rubbish in storage rooms; and in folds of cloth ing. Despite their impressive abundance in some homes, these spiders rarely bite humans. Bites tend to occur while the victim is donning clothing in which the spider has hidden itself and are sustained primar ily on the hands, arms, neck, and lower abdomen. A brown recluse spider's bite may cause minor injury with edema and erythema, and envenomation can cause severe necrosis of skin and subcutaneous tissue and, more rarely, systemic hemolysis. Initially, the bite is painless or may produce a stinging sensation. Within a few hours, the site becomes painful and sensitive to touch, with central induration surrounded by a pale ischemic zone that itself is encircled by a zone of erythema. In most cases, the lesion resolves without treatment in just a few days. In severe cases, systemics signs, such as fever, chills, weakness, headache, nausea, vomiting, myalgia, arthralgia, and leukocytosis, may soon develop. As the bite site evolves, the

erythematous zone expands, and the center becomes hemorrhagic or necrotic with an overlying bulla. A black eschar forms and sloughs several weeks later, leaving an ulcer that eventually may create a depressed scar. Healing usually takes place in ≤3 months. Local complications include injury to nerves and secondary bacterial infection. Reports of deaths attributed to bites of North American brown recluse spiders have not been verified. PART 14 Poisoning, Drug Overdose, and Envenomation The Mediterranean recluse spider (Loxosceles rufescens) is a widely invasive species in urban areas of both the Old and New Worlds. The dorsal surfaces of L. rufescens and L. reclusa are adorned with a fiddleshaped pattern. L. rufescens is warier than L. reclusa, is less likely to bite, and rarely causes necrosis. Misidentification of this spider may create spurious reports of L. reclusa activity outside the known range of that species. TREATMENT Recluse Spider Bites Initial management includes rest, ice, compression, and elevation (RICE). Analgesics, antihistamines, antibiotics, and tetanus pro phylaxis should be administered if indicated. Early debridement or surgical excision of the wound without closure delays healing. Routine use of antibiotics or dapsone lacks utility. Patients should be monitored closely for signs of hemolysis, renal failure, and other systemic complications. Widow Spider Bites The southern black widow spider (Latrodectus mactans) is common in the southeastern United States. Female bodies are ~1 cm in length, but the leg span may be ~5 cm across. Their bodies are shiny black with a red hourglass marking on the ventral abdomen. Other dangerous Latrodectus species occur elsewhere in temperate and subtropical parts of the world. The bites of the female widow spiders are notorious for their potent neurotoxins. Widow spiders spin their webs under stones, logs, plants, or rock piles and in dark spaces in barns, garages, and outhouses. Bites are most common in the summer and early autumn and occur when a web

is disturbed or a spider is trapped or provoked. The initial bite is per ceived as a sharp pinprick or may go unnoticed. Fang-puncture marks are uncommon. Envenomation does not cause local tissue damage, and some persons experience no further symptoms. α-Latrotoxin, the most active component of the venom, is a neuro toxin. It binds irreversibly to presynaptic nerve terminals and causes release and eventual depletion of acetylcholine, norepinephrine, and other neurotransmitters from those terminals. Painful cramps may spread within 60 min from the bite site to large muscles of the extremi ties and trunk. Extreme abdominal pain and rigidity may resemble peritonitis, but the abdomen is not tender on palpation and surgery is not warranted. The pain begins to subside during the first 12 h but may recur over the next few days or weeks before resolving spontane ously. A wide range of other sequelae, largely neurologic, may include salivation, diaphoresis, vomiting, hypertension, tachycardia, labored breathing, anxiety, headache, weakness, fasciculations, paresthesia, hyperreflexia, urinary retention, uterine contractions, and premature labor. Rhabdomyolysis and renal failure have been reported, and respi ratory arrest, cerebral hemorrhage, or cardiac failure may end fatally, especially in very young, elderly, or debilitated persons. TREATMENT Widow Spider Bites Treatment consists of RICE and tetanus prophylaxis. Hypertension that does not respond to analgesics and antispasmodics (e.g., ben zodiazepines or methocarbamol) requires specific antihypertensive medication. The efficacy and safety of antivenin made from equine immunoglobulins are controversial for black widow bites because of potential anaphylaxis or serum sickness. Antivenins made from monoclonal antibodies are in development. Tarantulas and Other Spiders Tarantulas are large hairy spiders of which 30 species are found in the United States, mainly in the South west. Several species of tarantulas have become popular household pets and are usually imported from Central or South America. Tarantulas bite people only when threatened and usually cause no more harm than a bee sting, but

on occasion, the venom causes deep pain and swelling. Several species of tarantulas are covered with urticating hairs that are brushed off in the thousands when a threatened spider rubs its hind legs across its dorsal abdomen. These hairs can penetrate human skin and produce pruritic papules that may persist for weeks. Failure to wear gloves or to wash the hands after handling the Chilean Rose tarantula, a popular pet spider, has resulted in transfer of hairs to the eye with subsequent devastating ocular inflammation. Treatment of bites includes local washing and elevation of the bitten area, tetanus prophylaxis, and analgesic administration. Antihistamines and topical or systemic glucocorticoids are given for exposure to urticating hairs. Atrax robustus, a funnel-web spider of Australia, and Phoneutria species, the South American banana spiders, are among the world's most dangerous spiders because of their aggressive behavior and potent neurotoxins. Envenomation by A. robustus causes a rapidly progres sive neuromotor syndrome that can be fatal within 2 h. The bite of a banana spider causes severe local pain followed by profound systemic symptoms and respiratory paralysis that can lead to death within 2–6 h. Specific antivenins for use after bites by each of these spiders are available. Yellow sac spiders (Cheiracanthium species) are common in homes worldwide. Their bites, though painful, generally lead to only minor erythema, edema, and pruritus. ■ ■SCORPION STINGS Scorpions are arachnids that feed on arthropods and other small animals. They paralyze their prey and defend themselves by inject ing venom from a stinger on the tip of the tail. Painful but relatively harmless scorpion stings need to be distinguished from the potentially lethal envenomations from about 30 of roughly 1000 known species, which cause several thousand deaths worldwide each year. Scorpions are nocturnal and remain hidden during the day in crevices or burrows

or under wood, loose bark, or rocks. They occasionally enter houses and tents and may hide in shoes, clothing, or bedding. Scorpions sting humans only when threatened. Of approximately 40 scorpion species in the United States, only bark scorpions (Centruroides sculpturatus/C. exilicauda) in the South west produce venom that is potentially lethal to humans. This venom contains neurotoxins that cause sodium channels to remain open. Such envenomations usually are associated with little swelling, but prominent pain, paresthesia, and hyperesthesia can be accentuated by tapping on the affected area (the tap test). These symptoms soon spread to other locations; dysfunction of cranial nerves and hyperexcitability of skeletal muscles develop within hours. Patients present with rest lessness, blurred vision, abnormal eye movements, profuse salivation, lacrimation, rhinorrhea, slurred speech, difficulty in handling secre tions, diaphoresis, nausea, and vomiting. Muscle twitching, jerking, and shaking may be mistaken for a seizure. Complications include tachycardia, arrhythmias, hypertension, hyperthermia, rhabdomyoly sis, and acidosis. Symptoms progress to maximal severity in ~5 h and subside within a day or two, although pain and paresthesia can last for weeks. Fatal respiratory arrest is most common among young children and the elderly. Envenomations by Leiurus quinquestriatus in the Middle East and North Africa, by Mesobuthus tamulus in India, by Androctonus species along the Mediterranean littoral and in North Africa and the Middle East, and by Tityus serrulatus in Brazil cause massive release of endoge nous catecholamines with hypertensive crises, arrhythmias, pulmonary edema, and myocardial damage. Acute pancreatitis occurs with stings of Tityus trinitatis in Trinidad, and central nervous toxicity complicates stings of Parabuthus and Buthotus scorpions of South Africa. In Iran and adjacent countries, Hemiscorpius lepturus causes the most scorpion envenomations. Its stings are relatively asymptomatic at first, but its cytotoxic venom causes pain, hemolysis, and tissue necro sis after the first day. Systemic complications include hemoglobinuria and subsequent acute kidney injury. Stings of most other species cause immediate sharp local pain fol lowed by

edema, ecchymosis, and a burning sensation. Symptoms typically resolve within a few hours, and skin does not slough. Allergic reactions to the venom may occur. TREATMENT Scorpion Stings Identification of the offending scorpion helps to determine the course of treatment. Stings of nonlethal species require at most ice packs, analgesics, or antihistamines. Because most victims experi ence only local discomfort, they can be managed at home with instructions to return to the emergency department if signs of cranial-nerve or neuromuscular dysfunction develop. Aggressive supportive care and judicious use of antivenom can reduce or elimi nate deaths from more severe envenomations. Keeping the patient calm and applying pressure dressings and cold packs to the sting site are measures that decrease the absorption of venom. A continu ous IV infusion of midazolam reduces the agitation and involuntary movements produced by scorpion stings. Treating people with neuromuscular symptoms with sedatives or opiates requires close monitoring due to potential respiratory compromise. Hyperten sion and pulmonary edema respond to nifedipine, nitroprusside, hydralazine, or prazosin. Dangerous bradydysrhythmia can be controlled with atropine. Commercially prepared antivenins are available in several coun tries for some of the most dangerous scorpion species. The FDA has approved equine-derived C. sculpturatus IgG F(ab')2 antivenin. IV administration of antivenin rapidly reverses cranial-nerve dysfunc tion and muscular symptoms. ■ ■HYMENOPTERA STINGS Bees, wasps, hornets, yellow jackets, and ants (all of the insect order Hymenoptera) sting in defense or to subdue their prey. Their venoms

contain a wide array of amines, peptides, and enzymes that cause local and systemic reactions. Although the toxic effect of multiple stings can be fatal to a human, nearly all of the ≥100 deaths due to hymenopteran stings in the United States each year result from type 1, immediate-type allergic reactions. Bee and Wasp Stings The stinger of the honeybee (Apis mellifera) is unique in being barbed. The stinging apparatus and attached venom sac tear loose from the honeybee's body, and muscular contractions of the venom sac continue to infuse venom into the skin. Other kinds of bees, ants, and wasps have smooth stinging mechanisms and can sting numerous times in succession. Generally, a person sustains just one sting from a bee or social wasp unless a nest was disturbed. African ized honeybees (now present in South and Central America and the southern and western United States) respond to minimal intrusions more aggressively. The sting of an Africanized bee contains less venom than that of its non-Africanized relatives, but victims tend to sustain far more stings and thus receive a far greater overall volume of venom. Most patients who report having sustained a "bee sting" are more likely to have encountered stinging wasps instead.

The venoms of different kinds of hymenopterans are biochemically and immunologically distinct. Direct toxic effects are mediated by mixtures of low-molecular-weight compounds such as serotonin, hista mine, acetylcholine, and several kinins. Polypeptide toxins in honeybee venom include mellitin, which damages cell membranes; mast cell– degranulating protein, which causes histamine release; the neurotoxin apamin; and the anti-inflammatory compound adolapin. Enzymes in venom include hyaluronidase and phospholipases. There appears to be little cross-sensitization between the venoms of honeybees and wasps. CHAPTER 472 Ectoparasite Infestations and Arthropod Injuries Uncomplicated hymenopteran stings cause immediate pain, a wheal-and-flare reaction, and local edema, all of which usually subside in a few hours. Multiple stings can lead to vomiting, diarrhea, general ized edema, dyspnea, hypotension, and nonanaphylactic circulatory collapse. Rhabdomyolysis and intravascular hemolysis may cause renal failure. Death from the direct (nonallergic) effects of venom has fol lowed stings of several hundred honeybees. Stings to the tongue or mouth may induce life-threatening edema of the upper airways. Large local reactions

accompanied by erythema, edema, warmth, and tenderness that spread ≥10 cm around the sting site over 1–2 days are not uncommon. These reactions may resemble bacterial cellulitis but are caused by hypersensitivity rather than by secondary infec tion. Such reactions tend to recur on subsequent exposure but are seldom accompanied by anaphylaxis and are not prevented by venom immunotherapy. An estimated 0.4–4.0% of the U.S. population exhibits clinical immediate-type hypersensitivity to hymenopteran stings, and 15% may have asymptomatic sensitization manifested by positive skin tests. Per sons who experience severe allergic reactions are likely to have similar or more severe reactions after subsequent stings by the same or closely related species. Mild anaphylactic reactions to insect stings, as to other causes, consist of nausea, abdominal cramping, generalized urticaria or angioedema, and flushing. Serious reactions, including upper airway edema, bronchospasm, hypotension, and shock, may be rapidly fatal. Severe reactions usually begin within 10 min of the sting and only rarely develop after 5 h. TREATMENT Bee and Wasp Stings Honeybee stingers embedded in the skin should be removed as soon as possible to limit the quantity of venom delivered. The stinger and venom sac may be scraped off with a blade, a fingernail, or the edge of a credit card or may be removed with forceps. The site should be cleansed and disinfected and ice packs applied to slow the spread of venom. Elevation of the affected site and administration of oral analgesics, oral antihistamines, and topical calamine lotion help relieve symptoms. Anaphylactic reactions to bee or wasp venom can be a lifethreatening emergency that requires prompt life-saving actions.

If the individual carries a bee-sting kit, then a subcutaneous injec tion of epinephrine hydrochloride (0.3 mL of a 1:1000 dilution) should be considered, with treatment repeated every 20–30 min as necessary. A tourniquet may slow the spread of venom. The patient should be transferred to a hospital emergency room where treat ment for profound shock, if required, can be administered safely. Such treatment may entail the use of IV epinephrine and other vasopressors, intubation or provision of supplemental oxygen, fluid resuscitation, use of bronchodilators, and parenteral administration of antihistamines. Patients should be observed for 24 h for recurrent anaphylaxis, renal failure, or coagulopathy.

Persons with a history of allergy to insect stings should carry an anaphylaxis kit with a preloaded syringe containing epinephrine for self-administration. These patients should seek medical attention immediately after using the kit. Prophylactic immunotherapy may greatly reduce the risk of lifethreatening reactions to bee and wasp stings. Repeated injections of purified venom produce a blocking IgG antibody response to venom and reduce the incidence of recurrent anaphylaxis. Hon eybee, wasp, and yellow jacket venoms are commercially available for desensitization and for skin testing. Results of skin tests and venom-specific radioallergosorbent tests (RASTs) aid in the selec tion of patients for immunotherapy and guide the design of such treatment. PART 14 Poisoning, Drug Overdose, and Envenomation ■ ■STINGING ANTS Stinging ants are an important medical problem in the United States. Imported fire ants (Solenopsis species) infest southern states from Texas to North Carolina, with colonies now established in California, New Mexico, Arizona, and Virginia. Slight disturbances of their mound nests have provoked massive outpourings of ants and as many as 10,000 stings on a single person. Elderly and immobile persons are at high risk for attacks when fire ants invade dwellings. Fire ants attach to skin with powerful mandibles and rotate their bodies while repeatedly injecting venom with posteriorly situated stingers. The alkaloid venom consists of cytotoxic and hemolytic piperidines and several proteins with enzymatic activity. The initial wheal-and-flare reaction, burning, and itching resolve in ~30 min, and a sterile pustule

develops within 24 h. The pustule ulcerates over the next 48 h and then heals in ≥1 week. Large areas of erythema and edema lasting several days are not uncommon and, in extreme cases, may compress nerves and blood vessels. Anaphylaxis occurs in <2% of victims; seizures and mononeuritis have been reported. Stings are treated with ice packs, topical glucocorticoids, and oral antihistamines. Pustules should be cleansed and then covered with bandages and anti biotic ointment to prevent bacterial infection. Epinephrine administra tion and supportive measures are indicated for anaphylactic reactions. Fire ant whole-body extracts are available for skin testing and immu notherapy, which appear to lower the rate of anaphylactic reactions. European fire (red) ants (Myrmica rubra) have recently become pub lic health pests in the northeastern United States and southern Canada. The western United States is home to harvester ants (Pogonomyrmex species). The painful local reaction that follows harvester ant stings often extends to lymph nodes and may be accompanied by anaphy laxis. The bullet or conga ant (Paraponera clavata) of South America is known locally as hormiga veinticuatro (“24-hour ant”), a designation that refers to the 24 h of throbbing, excruciating pain following a sting that delivers the potent paralyzing neurotoxin poneratoxin. ■ ■DIPTERAN (FLY AND MOSQUITO) BITES In the process of feeding on vertebrate blood and tissue fluids, adults of certain fly species inflict painful bites, inject saliva that may cause vaso dilation and produce local allergic reactions, and may transmit diverse pathogenic agents. Bites of mosquitoes (culicids), tiny “no-see-um” midges (ceratopogonids), and sand flies (phlebotomines) typically produce a wheal and a pruritic papule. Small humpbacked black flies (simuliids) lacerate skin, resulting in a lesion with serosanguineous discharge that is often painful and pruritic. Regional lymphadenopathy,

fever, or anaphylaxis occasionally ensues. The widely distributed deer flies and horseflies as well as the tsetse flies of Africa are stout flies that attack during the day and produce large and painful bleeding punctures. House flies (Musca domestica) do not consume blood but use rasping mouthparts to scarify skin and feed upon tissue fluids and salt. Beyond direct injury from bites of any kind of fly, risks include transmission of diverse pathogens and secondary bacterial infection of the lesion. TREATMENT Fly and Mosquito Bites Treatment of fly bites is symptom based. Topical application of antipruritic agents, glucocorticoids, or antiseptic lotions may relieve itching and pain. Allergic reactions may require oral antihista mines. Antibiotics may be necessary for the treatment of large bite wounds that become secondarily infected. ■ ■FLEA BITES Common human-biting fleas include the dog and cat fleas (Cteno cephalides species) and the rat flea (Xenopsylla cheopis), which infest their respective hosts and their nests and resting sites. Sensitized persons develop erythematous pruritic papules (papular urticaria) and occasionally vesicles and bacterial superinfection at the site of the bite. Symptom-based treatment consists of antihistamines, topical glucocor ticoids, and topical antipruritic agents. Flea infestations are eliminated by removal and treatment of animal nests, frequent cleaning of pet bedding, and application of contact and systemic insecticides to pets and the dwelling. Flea infestations in the home may be abated or prevented if pets are regularly treated with veterinary antiparasitic agents, insect growth regulators, or chitin inhibitors. Tunga penetrans, like other fleas, is a wingless, laterally flattened insect that feeds on blood. Also known as the chigoe flea, sand flea, or jigger (not to be confused with the chigger), it occurs in tropical regions of Africa and the Americas. Adult female chigoes live in sandy soil and burrow under the skin, usually between toes, under nails, or on the soles of bare feet. Gravid chigoes engorge on the host’s blood and grow from pinpoint to pea size during a 2-week interval. They produce lesions that resemble a white pustule with a central black depression and that may be pruritic or painful. Occasional complications include tetanus, bacterial infections,

and autoamputation of toes (ainhum). Tungiasis is treated by removal of the intact flea with a sterile needle or scalpel, tetanus vaccination, and topical application of antibiotics. ■ ■HEMIPTERAN/HETEROPTERAN

(TRUE BUG) BITES Most true bugs feed on plants, but some are predaceous or feed on blood. In order to feed or to defend themselves, they may inflict bites that produce allergic reactions and are sometimes painful. Bites of the cone-nose or “kissing bugs” (family Reduviidae) tend to occur at night and are painless. Reactions to such bites depend on prior sensitization and include tender and pruritic papules, vesicular or bullous lesions, extensive urticaria, fever, lymphadenopathy, and (rarely) anaphylaxis. Bug bites are treated with topical antipruritic agents or oral antihista mines. Persons with anaphylactic reactions to reduviid bites should keep an epinephrine kit available. Some reduviids transmit Trypano soma cruzi, the agent of New World trypanosomiasis (Chagas disease) (Chap. 234). The cosmopolitan and tropical bed bugs (Cimex lectularius and C. hemipterus) hide in crevices of mattresses, bed frames and other fur niture, walls, and picture frames and under loose wallpaper, actively seeking blood meals at night. These bugs are now a common pest in homes, dormitories, and hotels; on cruise ships; and even in medical facilities. Their bite is painless. Bites on persons without prior exposure to bedbugs may not be noticeable. Persons sensitized to bed bug saliva develop erythema, itching, and wheals around a central hemorrhagic punctum. Reactions may manifest within minutes of the bites, or they

may be delayed for days or even a week or more. Bed bugs are not known to transmit pathogens. ■ ■CENTIPEDE BITES AND MILLIPEDE DERMATITIS Two groups of myriapods (“many-footed” arthropods) can harm humans. Centipedes, with one pair of legs per body segment, are fast-moving, aggressive, and carnivorous. They stun and kill their prey—usually other arthropods, earthworms, and rarely small verte brates—with a venomous bite. The fangs of centipedes of the genus Scolopendra can penetrate human skin and deliver a venom that produces intense burning pain, swelling, erythema, and sterile lym phangitis. Dizziness, nausea, and anxiety are described occasionally, and rhabdomyolysis and renal failure have been reported. Treatment includes washing of the site, application of cold dressings, oral analgesic administration or local lidocaine infiltration, and tetanus prophylaxis. Millipedes, with two pairs of legs per segment, are slow-moving, docile, and feed mostly on decaying plant materials. They do not bite, but some secrete defensive fluids that may burn and discolor human skin. Affected skin turns brown overnight and may blister and exfoli ate. Secretions in the eye cause intense pain and inflammation that can result in corneal ulcers and even blindness. Management includes irrigation with copious amounts of water or saline, use of analgesics, and local care of denuded skin. ■ ■CATERPILLAR STINGS AND DERMATITIS Caterpillars of several moth species are covered with hairs or spines that produce mechanical irritation and may contain or be coated with venom. Contact with these caterpillars or their hairs may lead to eru cism (a pruritic urticarial or papular rash) or caterpillar envenomation. The response typically consists of an immediate burning sensation followed by local swelling and erythema and occasionally by regional lymphadenopathy, nausea, vomiting, and headache. A rare reaction to a South American caterpillar, Lonomia obliqua, can cause disseminated coagulopathy and fatal hemorrhagic shock. Dermatitis is most often associated with caterpillars of io, puss, saddleback, and browntail moths in North America and with the oak processionary moth in Europe. Even contact with detached hairs of FIGURE 472-4 Real (left) versus delusional (right) infestation: comparable images of the lower backs of two young adults with multiple lesions. Left: A young woman developed innumerable widespread lesions during a camping ecotour near Manaus, Brazil.

Note scattered clusters of irregularly spaced lesions, accompanied by dozens of single or isolated lesions, consistent with the semi-random feeding pattern of biting flies. Lesions appear to be in roughly the same stage of development, a feature indicating that they were acquired at roughly the same time. No lesions were present before her ecotour; none have arisen since. This patient scratches the intensely pruritic lesions and causes superficial erosions. Unexcoriated lesions are also present on her midback, where she cannot scratch. Right: A young man has innumerable widespread lesions that have accumulated for several years, with a few new lesions appearing several times a week. His lesions are in various stages of development (fresh, crusted, reepithelialized, pigmented, and scarred), a feature indicating a long-standing process. The lesions are distributed in a regular pattern consistent with periodic “excavations” to remove alleged parasites that he believes are crawling through his skin. Scarring is due to manipulations that create dermal ulcers rather than superficial excoriations and erosions. Parts of his upper midback, where he cannot scratch, are free of lesions.

other caterpillars, such as gypsy moth larvae, can later produce eru cism. Spines may be deposited on tree trunks or drying laundry or may be airborne and cause irritation of the eyes and upper airways. Treat ment of caterpillar stings consists of repeated application of adhesive or cellophane tape to remove the hairs, which can then be identified microscopically. Local ice packs, topical glucocorticoids, and oral anti histamines relieve symptoms.

Few adult moths cause human health problems. Adult yellowtail moths (Hylesia species), found mainly in coastal mangrove zones along the eastern coast of Central and South America, have bodies that are covered by fine hairs or setae. The hairs on the ventral surface can detach and, when in contact with human skin, cause an extremely pru ritic reaction called “Carapito itch.” This issue is especially problematic when the moths have population booms, creating swarms around coastal communities. ■ ■BEETLE VESICATION AND DERMATITIS Several families of beetles have independently developed the ability to produce chemically unrelated vesicating toxins. When disturbed, blister beetles (family Meloidae) exude cantharidin, a low-molecularweight toxin that produces thin-walled blisters (≤5 cm in diameter) 2–5 h after contact. The blisters are not painful or pruritic unless bro ken. They resolve without treatment in ≤10 days. Nephritis may follow unusually heavy cantharidin exposure. CHAPTER 472 The hemolymph of certain rove beetles (Paederus species, Staphylin idae family) contains pederin, a potent vesicant. When these beetles are crushed or brushed against the skin, the released fluid causes painful, red, flaccid bullae. These beetles occur worldwide but are most numer ous and problematic in parts of Africa (where they are called “Nairobi fly”) and southwestern Asia. Ocular lesions may develop after impact with flying beetles at night or unintentional transfer of the vesicant on the fingers. Treatment is rarely necessary, although ruptured blisters should be kept clean and bandaged. Ectoparasite Infestations and Arthropod Injuries Larvae of common carpet beetles are adorned with dense arrays of ornate hairs called hastisetae. Contact with these larvae or their setae results in delayed dermal reactions in sensitized individuals. The lesions are commonly mistaken for bites of bed bugs.

■ ■DELUSIONAL INFESTATIONS The groundless conviction that one is infested with arthropods or other parasites (Ekbom syndrome, delusory parasitosis, delusions of parasitosis, delusion of infestation, and perhaps Morgellons syndrome) is extremely difficult to treat and, unfortunately, is not uncommon (Fig. 472-4). Patients describe uncomfortable sensations of something moving in or on their skin. Excoriations and self-induced ulcerations typically accompany the pruritus,

dysesthesias, and imaginary insect bites. Patients often believe that some invisible or as-yet-undescribed creatures are infesting their skin, clothing, homes, or environment in general. Frequently, patients submit as evidence of infestation specimens that consist of plant-feeding and nonbiting peridomestic arthropods, pieces of skin, vegetable matter, lint, and other inanimate detritus. In the evaluation of a patient with possible delusional parasit osis, it is imperative to rule out true infestations and bites by arthro pods, endocrinopathies, sensory disorders due to neuropathies, opiate and other drug use, environmental irritants (e.g., fiberglass threads), and other causes of tingling or prickling sensations. Frequently, such patients repeatedly seek medical consultations, resist alternative expla nations for their symptoms, and exacerbate their discomfort by selftreatment. Recent evidence suggests that high levels of central nervous system dopamine, endogenous or pharmaceutical, may promote these delusions. Long-term pharmacotherapy with pimozide or other psy chotropic agents has been more helpful than psychotherapy in treat ing this disorder. Patients with delusory parasitosis often develop the unshakeable conviction that they are infested by a previously unknown pathogen, while their personal lives, family support, and employment collapse around them.

PART 14 Poisoning, Drug Overdose, and Envenomation

Acknowledgment Richard J. Pollack contributed to this chapter in previous editions and some material from that chapter has been retained here. ■ ■FURTHER READING Amanzougaghene N et al: Where are we with human lice? A review of the current state of knowledge. Front Cell Infect Microbiol 21:9,

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