# Chapter 9

# ARTHROPOD INFESTATIONS AND VECTORS OF DISEASE

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## INTRODUCTION

To an entomologist the word "insect" refers to the class Insecta, a group of organisms with six legs and three body segments: head, thorax, and abdomen. The layman and many in the medical community would also include spiders and mites (class Arachnida), both of which have eight legs and two body segments: head and abdomen. A more precise and inclusive term than insect is arthropod (phylum Arthropoda), comprising organisms from both classes, Insecta and Arachnida; these classes have as common features a hard, jointed exoskeleton and paired, jointed legs (Table 9-1). Some arthropods evolved into parasites, developing piercing-sucking mouth parts enabling them to obtain blood meals. Harwood and James[1] believe that in their evolution, arthropods began as scavengers of vertebrate-lair detritus. The host, in addition to providing a steady food source, provides warmth and shelter. Synanthropy, in contrast to parasitism, is a loose form of dependence between insects (eg, cockroaches, some fly and ant species) and vertebrates in which direct host feeding does not occur.[1]

Insects are estimated to have preceded human existence by at least 400 million years. Documentation of human afflictions with insects dates back to prehistoric times.[1] Legends, art, and speech have preserved our earliest knowledge of these most numerous pests. Stoneware from Mexico (AD 1200) and Peru (AD 400–900) display detailed depictions of fleas and tungiasis.[1] Variations of the story of Pandora's box attempt to explain human louse and flea infestations. In about AD 1200, Native American potters depicted mosquitoes poised for attack. In Exodus 8:24, the Bible refers to "a grievous swarm of flies into the house of Pharaoh, and into all the

**TABLE 9-1**

**ARTHROPODS KNOWN TO BE VECTORS OF HUMAN INFECTIOUS DISEASES**

| Class —Subclass | Order —Suborder | Families —Subfamilies | Common Names |
|---|---|---|---|
| Arachnida | | | |
| —Acari | Acarina | Ixodidae | Hard ticks |
| | | Argasidae | Soft ticks |
| —Acari | Acarina —Parasitiformis | Gamasidae | Mites |
| Insecta | | | |
| | Anoplura | Pediculidae | Head and body lice |
| | Pthiridae | Crab louse | |
| | Siphonaptera | Pulicidae | Human flea |
| | | Leptopsyllidae | Mouse flea |
| | Diptera | Muscidae | Tsetse fly |
| | | Culicidae | |
| | | —Culicinae | *Aedes* mosquito |
| | | —Anophelinae | *Anopheles* mosquito |
| | | Psychodidae | |
| | | —Phlebotominae | Sandflies |
| | | Simuliidae | Black flies |
| | Diptera | | |
| | —Brachycera | Tabanidae | Horse and deer flies |
| | Hemiptera | Reduviidae | Kissing bug |

Data sources: (1) Harwood RF, James MT, eds. *Entomology in Human and Animal Health*. 7th ed. New York, NY: Macmillan Publishing Co; 1979: 117–392. (2) Alexander JO. *Arthropods and Human Skin*. Berlin, Germany: Springer-Verlag; 1984: 303.

land of Egypt: the land was corrupted by reason of the swarm of flies." That insects were vectors of human disease was first suggested in 1577 by Mercurialis, who believed that flies carried the "virus" of plague.[1] Subsequent observations by numerous investigators up to the early 1900s helped better understand vector transmission of yaws, Carrion's disease, bubonic plague, malaria, and yellow fever. Medical entomology began in 1909 and was the direct result of the development of the microscope, rejection of the spontaneous generation theory, formulation of the germ theory by Louis Pasteur, and establishment of controlled experimentation by Walter Reed.[1,2]

Diseases caused by insects and arachnids can be the result of direct tissue injury, transmission of intermediate host parasites, or vector transmission of infectious and parasitic organisms.[1] Human diseases transmitted by insect vectors are some of the most ancient and deadly of all infectious disorders. During World War I, louse-borne typhus contributed to the collapse of the Russian and Balkan fronts.[1] Epidemics occurred also during World War II in Naples, Italy, and Germany; however, use of dichlorodiphenyltrichloroethane (DDT) greatly reduced the spread of disease.[1] War and natural disasters promote epidemics of these ancient disorders, as exemplified by the concerns that widespread epidemics would follow Operations Desert Storm and Desert Shield, 1991–1992.

## MITES AND TICKS

Three orders of medical importance are found within the class Arachnida: mites and ticks (Acarina), spiders (Araneida), and scorpions (Scorpionida).[3] Of these, only mites and ticks are significant vectors of human disease. Infestations by gamasid mites have been suspected to transmit a variety of rickettsial and viral diseases (Figure 9-1).[4] *Ornithonyssus bacoti* has been implicated in the transmission of endemic typhus, rickettsialpox, and Q fever (which are discussed in Chapter 11, Rickettsial Diseases), as well as relapsing fever, St. Louis encephalitis, and western equine encephalitis. *Ornithonyssus sylviarum* can transmit western equine encephalitis and St. Louis encephalitis.

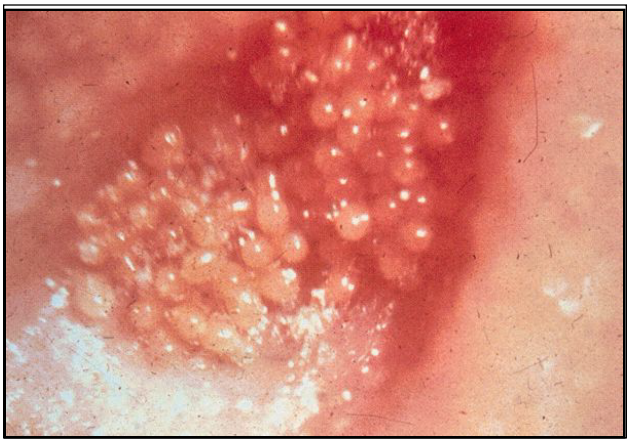

**Fig. 9-1.** Gamasid mites. These mites have a worldwide distribution, are known to attack man, and are suspect vectors of rickettsial and viral diseases.

*Ornithonyssus bursa* has been shown to transmit western equine encephalitis and is suspected to be a vector for Q fever. *Demanyssus gallinae* is a vector for the transmission of endemic typhus and St. Louis encephalitis. Rickettsialpox outbreaks in New York have been reported to be transmitted by *Liponyssoides sanguineus* (house mouse mite).[5] Gamasid mites are parasitic for domestic and wild birds, rats, and mice. They have a worldwide distribution and are known to attack humans.

Scabies is an infestation with *Sarcoptes scabiei*, an eight-legged human mite. The disease is most frequently transmitted through sexual intercourse. Chiggers, from the family Trombiculidae, are nonscabietic mites found on the ground or in grass. Their larvae can crawl up the legs as the host walks through infested vegetation. For a detailed discussion of mites and the dermatoses they transmit, see Chapter 8, Arthropod and Other Animal Bites.

Ticks are natural parasites of mammals, birds, reptiles, and amphibians (Table 9-2). Ticks consist of two groups: (1) the Ixodidae, which have a hard, chitinous, dorsal shield (ie, scutum), and (2) the Argasidae, or soft tick, which lacks a scutum. The natural life cycle is dependent on longer feeding cycles than those of other parasitic organisms such as flies, and ticks will feed until engorged with blood, reaching several times their original size (Figure 9-2).

As a group, most ticks fast for long periods because they cannot live on vegetable matter. A blood meal is acquired mostly by chance. Ticks climb to the top of grass stems or shrubs and await the passing of a suitable host. Sensory response to odor, vibration, air currents, interruption of inci-

**TABLE 9-2**

**TICKS AS VECTORS OF HUMAN INFECTIOUS DISEASES**

| Infectious Disease | Tick Vector | Distribution |
|---|---|---|
| Lyme disease | *Ixodes ricinus*, *I pacificus*, *I scapularis* | Worldwide |
| Relapsing fever | *Ornithodoros* spp | Africa, Asia, United States, Europe, Near East, Mediterranean, Canada, Central and South America |
| Tularemia | *Dermacentor andersoni*, *Ixodes* spp | Americas, Europe, Japan, Israel, Africa |
| Arboviruses | | |
| Omsk hemorrhagic fever | *Dermacentor pictus*, *D marginatus*, *Ixodes persulcatus*, *I apronophorus* | Siberia |
| Russian spring-summer encephalitis | *Ixodes persulcatus* | Central Europe, former USSR |
| Louping ill | *Ixodes ricinus* | British Isles, Spain |
| Powassan | *Dermacentor andersoni*, *Ixodes spinipalpis* | United States, Canada |
| Kyasanur forest disease | *Haemaphysalis* spp, *Ixodes* spp *Rhipicephalus turanicus* | India |
| Crimean-Congo hemorrhagic fever | *Hyalomma marginatum* | Asia, Europe, former USSR, Mediterranean, China, Middle East, Africa |
| Colorado tick fever | *Dermacentor andersoni*, *Ixodes* spp | United States, Canada |
| Ungrouped or other viruses | | |
| Lymphocytic choriomeningitis* | *Amblyomma variegatum*, *Rhipicephalus sanguineus*,*Dermacentor andersoni* | Ethiopia, Canada |
| Rickettsia | | |
| Rocky Mountain spotted fever | *Ixodes* spp, *Dermacentor andersoni* | United States, Canada, Mexico, South America |
| Siberian tick typhus | *Ixodes* spp | Former USSR, Japan, Far East |
| Boutonneuse fever | *Rhipicephalus sanguineus*, *Haemaphysalis* spp, *Ixodes* spp, *Hyalomma* spp, *Amblyomma* spp, *Boophilus* spp, *Dermacentor* spp | Africa, Europe, Asia, Mediterranean, Turkey, Crimea, Israel |
| Queensland tick typhus | *Ixodes holocyclus* | Queensland, Australia |
| Epidemic (louse-borne) typhus | *Pediculus humanus corporis* | Ethiopia |
| Q fever* | *Hyalomma asiaticum* | Europe, Mediterranean, Black Sea, Asia, Africa, North America, Australia |
| Tick-bite fever* | *Haemaphysalis leachi* | South Africa |
| Sennetsu fever* | Tick spp | Japan |
| Ehrlichiosis* | Tick spp | United States |
| Piroplasmosis | | |
| Human babeosis | *Ixodes scapularis* | Eastern and midwestern United States |

*Association with human transmission by tick vectors yet to be proven.

Data sources: (1) Harwood RF, James MT, eds. *Entomology in Human and Animal Health.* 7th ed. New York, NY: Macmillan Publishing Co; 1979: 371–416. (2) Samlaska CP. Arthropod-borne virus infections and virus hemorrhagic fevers. In: Demis DJ, ed. *Clinical Dermatology*. New York, NY: JB Lippincott; 1991: Unit 14-22; 1–15. (3) Gear JH, Wagner JM, Dyssel JC, et al. Severe tick-bite fever in children. *S Afr Med J*. 1990;77:84–87.

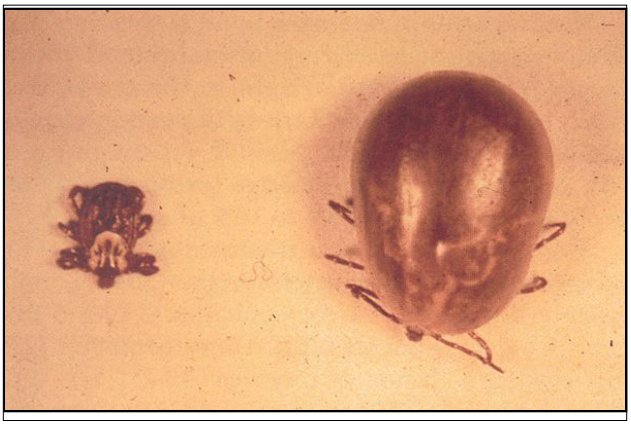

**Fig. 9-2**. Normal (on left) and engorged (on right) eastern wood ticks. When engorged with blood, ticks expand to several times their normal size.

dent light, warmth, and even moisture signal the presence of a potential host. The tick lacerates the host's skin with rigid, toothed chelicerae. Salivary solutions soften the surrounding tissues. As the tick penetrates the skin, it assumes a more vertical position (Figure 9-3). A cementlike substance is secreted into the wound, securing the hypostome into place. Frequently, hemorrhage takes place around the hypostome due to the cytolytic and anticoagulant action of secreted saliva. Feeding is usually complete within 6 to 7 days, but the tick can remain attached to the host for an unspecified period. Some ticks can live for 15 to 16 years.[3]

Ticks require a blood meal before they can lay eggs, with the number of eggs laid ranging from 300 to 7,000 depending on the species. There are four stages of development: egg, larva, nymph, and adult. The interval of development between the various stages can be considerable (66–359 d).

Caution should be exercised when removing ticks; at no time should they be forcibly removed. Forceful removal promotes breaking off of mouthparts and the subsequent development of a foreign-body granuloma. The preferred method for tick removal is by forceps.[6,7] The forceps are pressed to the sides of the tick's mouth parts and with a levering and lifting motion the tick is detached. Care should be taken to avoid squeezing the body of the tick.

Numerous other methods of tick removal are available.[3] Application of liquid paraffin to the tick results in blocking tick respirations. This technique requires 2 or more hours to work. Applying chloroform or ether to the tick results in quicker removal; however, there is a distinct risk of burn injury if unexpectedly ignited. Application of a warm match or match flame also works quickly, but has the same risk of igniting hair. Excisions or punch biopsies under local anesthesia are quite successful but are more invasive than other methods.

Because rapid deployment and high mobility are required of today's military, regional control of tick vectors for military personnel in the field is unlikely to prove effective. Therefore, individual preventive measures are most important. Use of permethrin-based repellents on clothing and applications of diethyltoluamide (DEET) on the skin are helpful. A recent study performed by the U.S. Army Environmental Hygiene Agency[8] clearly demonstrated that military uniforms impregnated or sprayed with permethrin more successfully controlled tick infestations than DEET applied to uniforms or skin. Duration of tick attachment has been shown to correlate with the probability of disease transmission; therefore, diligent daily searches for ticks should be performed.[9] In endemic areas, prophylactic antibiotics can be administered to soldiers who have been bitten by a tick.[9]

**Lyme Disease**

Lyme disease is an inflammatory disorder caused by the spirochete *Borrelia burgdorferi* and transmitted by Ixodes ticks. An early manifestation of the disease is an annular cutaneous lesion known as erythema chronicum migrans; however, the nervous system, heart, and joints may also become

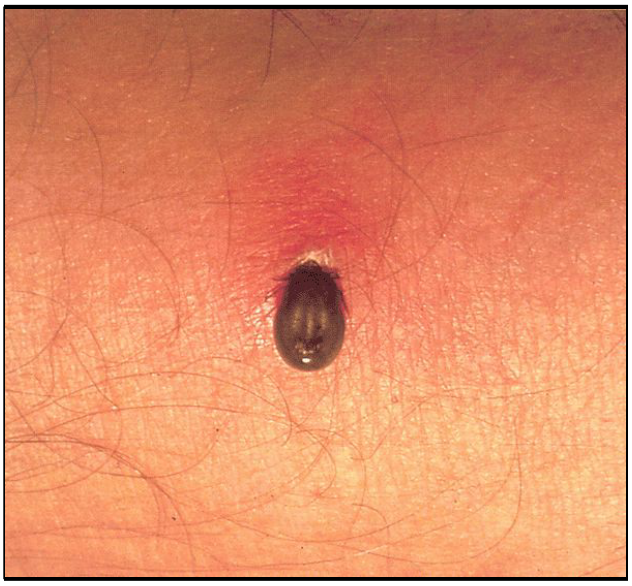

**Fig. 9-3**. Tick feeding on human subject. Note vertical positioning of the engorged tick body.

involved. For a detailed discussion of Lyme disease, see Chapter 13, Bacterial Skin Diseases.

**Relapsing Fever**

Relapsing fever is either endemic and transmitted by ticks or epidemic and transmitted by lice. Endemic tick-borne relapsing fever is caused by many species of *Borrelia* and is transmitted to humans by the soft *Ornithodoros* ticks. Relapsing fever transmitted by ticks is endemic in the western United States, where infected ticks reside in dead wood or are carried by wild rodents and domestic animals. Infection occurs during the summer months in the United States. Tick bites are often not recognized because they are painless; moreover, *Ornithodoros* ticks are night feeders and drop off quickly. The infective agents of tick-borne relapsing fever can vary their major surface antigens and thus evade the host's immune response.[10] *Borrelia* organisms infesting tick vectors are maintained in nature in the tick, which frequently has a long life (*Ornithodoros* ticks can live up to 13 y), by trans-ovarian transmission in some tick species, and by transmission to rodent reservoir hosts.[11]

Epidemic louse-borne relapsing fever is caused by *Borrelia recurrentis* and is transmitted to humans by the body louse *Pediculus corporis.*[10] Louse-borne relapsing fever was associated with large epidemics during World War II, with over 50,000 reported deaths.[9] Lice remain infective for their lifetime, and have no apparent natural vertebrate hosts.

The clinical manifestations of tick-borne and louse-borne relapsing fevers are the same. After a short incubation period of 3 to 10 days, the patient presents with sudden onset of fever associated with constitutional symptoms of cough, headache, lethargy, myalgias, and arthralgias. The fever terminates abruptly after a few days but is followed 3 to 10 days later by another febrile episode; this pattern persists, particularly in the tick-borne form, as new major surface antigens appear. The organisms are found in the blood and other bodily fluids during these febrile episodes. The severity and duration of the attacks decrease with time.[9,11]

An erythematous papular or petechial eruption may occur on the trunk during the last 1 to 2 days of the initial febrile episode. Up to 30% of patients develop neurological complications including cranial nerve palsies, meningitis, seizures, hemiplegia, and coma. Eye complications such as iritis and iridocyclitis occur in 15% of reported cases. Severe involvement resulting in death from fulminant liver failure and myocarditis have also been reported.[9]

The diagnosis of relapsing fever is confirmed on demonstration of *Borrelia* organisms in blood smears during febrile episodes. Wright- or Giemsa-stained dried blood smears, or dark-field examination of wet blood smears, yield positive results in up to 70% of patients. False-positive serologic tests for syphilis occur in up to 5% to 10% of cases.[9]

Antibiotics are effective treatment modalities for relapsing fever, including tetracycline (500 mg orally twice a day for 10–21 d), penicillin G (0.5–5 mU every 6 h intravenously for 10 d), erythromycin (250 mg orally 4 times a day for 10–21 d), and chloramphenicol (500 mg orally 4 times a day for 10–21 d). Louse-borne disease can be treated with a single 500-mg dose of erythromycin or tetracycline.

**Rickettsial Diseases**

*Rickettsia* is a Gram-negative genus of bacteria capable of causing a number of diseases in humans. These disorders are reviewed in detail in Chapter 11, Rickettsial Diseases; however, some of the more common human infections are worth mentioning briefly here.

Tick-bite fever is caused by *Rickettsia conorii* and is believed to be transmitted to humans by the dog tick, *Haemaphysalis leachi*. The disease affects predominantly children in South Africa and is usually benign and self-limited. In adults and some children, however, the disease may be fulminant, with a profuse maculopapular eruption, hepatitis, renal failure, and a hemorrhagic diathesis manifested as petechial hemorrhages, thrombocytopenia, and disseminated intravascular coagulation.[12]

Ehrlichiae are members of the family Rickettsiaceae, first isolated from infected dogs in 1935.[6] Until recently, the only known human disease was Sennetsu fever, caused by *Ehrlichia sennetsu*. The disease was first described and the organism first isolated in the 1950s in Japan. Characteristic features of the illness include remittent fever, generalized lymphadenopathy, hepatosplenomegaly, and increased circulating mononuclear cells and atypical lymphocytes similar to those observed in infectious mononucleosis. Other features of mononucleosis, such as fatigue, anorexia, chills, headache, and myalgias, have been reported. Cutaneous eruptions are unusual for this disorder. Although yet unproven, the disease is believed to be transmitted to humans through tick bites.[6]

During the Vietnam conflict in the late 1960s, a fatal epizootic hemorrhagic illness (called tropical canine pancytopenia) caused the deaths of many dogs attached to U.S. forces in the region.[13] The

illness correlated with heavy infestations of the tick *Rhipicephalus sanguineus*, and the causative organism was shown to be *Ehrlichia canis*. In 1987, the first case of human ehrlichiosis caused by *E canis* was reported.[14] Patient serum samples submitted for evaluation for suspected Rocky Mountain spotted fever are confirmed positive for *Rickettsia rickettsii* only 10% to 20% of the time.[6] Many of these seronegative cases are now believed to be due to Ehrlichiae. The majority of human ehrlichiosis cases have been reported in the southeastern, south central, and mid-Atlantic United States. The predominant clinical findings include fever (85%–99%); headache (83%–94%); myalgia (47%–82%); anorexia (81%–83%); nausea, vomiting, or both (42%–60%); rash (0%–60%); diarrhea (38%–50%); abdominal pain (19%–33%); confusion (12%–33%); and lymphaden-opathy (0%–19%).[6,15] When present, the cutaneous eruption may be petechial or macular and distributed over the trunk or extremities. Leukopenia, thrombo-cytopenia, anemia, and elevated aminotransferase levels may occur. Currently, confirmation of ehrlichiosis requires acute and convalescent sera for indirect immunofluorescence. Due to the retrospective nature of this form of confirmation, early diagnosis is based on clinical findings. Tetracycline (250 mg orally four times a day for 10 d) and tetracycline derivatives are the drugs of choice, although chloram-phenicol (500 mg orally four times a day for 10 d) has also been used successfully.[6,15]

**Tick Paralysis**

Tick paralysis in animals was first identified in Australia in 1824.[3] The first human case was reported by Bancroft[16] in 1884 in Queensland, Australia, and subsequent cases have been described in the United States, British Columbia, South Africa, France, Germany, Yugoslavia, and Crete (Table 9-3). The acute, ascending, lower-motor-neuron paralysis is due to a toxin produced by certain species of ticks. The toxin causes a conduction block at the myoneural junction similar to that observed with curare; it has been partially purified from the Australian tick *Ixodes holocyclus* and is resistant to digestion by pepsin, papain, and trypsin.[3,17] Parenteral injection into dogs results in paralysis within 48 hours. Tick paralysis results only from the bite of gravid female ticks.[17]

Children are more frequently afflicted with tick paralysis than adults. Although dark-haired individuals are said to be more susceptible, perhaps dark hair only makes the tick more difficult to find, and consequently the diagnosis is delayed.[17] Pain or numbness may precede the onset of motor weakness and, in children, restlessness, irritability, malaise, anorexia, and vomiting are not uncommon presenting complaints. Fever is not a feature of tick paralysis. The paralysis begins 2 or more days after the tick attaches, with weakness of the lower extremities progressing in hours to falling episodes and incoordination. True ataxia is rarely observed, and incoordination is attributed to motor weakness. Some cases of more proximal motor weakness have been described. Cranial nerve findings may ensue, manifested as dysarthria and dysphagia progressing to bulbar paralysis. Localized paralysis can sometimes occur, resulting in facial paralysis (tick attached to external auditory meatus), photophobia or blurred vision (tick attached behind the ear), or frontalis and orbicularis oculi paralysis (tick attached to frontal region).[3] The weakness is symmetrical and flaccid, and diffuse areflexia is present. The pupils remain reactive and sensory examination is normal. Patients usually succumb to respiratory failure.

Removal of the tick and supportive medical care, including respiratory support, form the cornerstone of therapy. The tick is usually found in the scalp. More than one tick may be present; therefore, a thorough examination is imperative. Clinical improvement usually begins within hours after tick removal, and full recovery occurs in 3 to 11 days for most patients. At times, however, recovery may be slower, requiring weeks to months. Permanent paralysis is exceedingly rare.

The differential diagnosis of tick paralysis includes Lyme disease, Guillain-Barré syndrome, myasthenia gravis, porphyria, botulism, and transverse myelitis.[7] The most common disease in this group is Guillain-Barré syndrome, which can be

**TABLE 9-3**

**GEOGRAPHIC DISTRIBUTION OF TICK VECTORS IMPLICATED IN HUMAN TICK PARALYSIS**

| Geographic Location | Ticks |
|---|---|
| Australia | *Ixodes holocyclus, I cornatus* |
| British Columbia | *Dermacentor andersoni* |
| United States | *Dermacentor andersoni* |
| South Africa | *Rhipicephalus evertsi evertsi* |

Data source: Kincaid JC. Tick bite paralysis. *Semin Neurol.* 1990;10:32–34.

differentiated from tick-bite paralysis by elevation of spinal fluid protein and slowed nerve-conduction velocities. Although Lyme disease may involve similar neurological complaints, they occur weeks to months after the tick bite. A negative enzyme-linked immunosorbent assay (ELISA) for *Borrelia burgdorferi* would also be helpful in differentiating the two diseases.

### Tick-Bite Alopecia

Tick-bite alopecia has been well established in the medical literature since Ross and Friede[18] described the first human case in 1955. The condition is believed to be due to direct toxic effects from tick saliva.[19] A history of tick bite is noted, or a tick may be found in the center of the site of alopecia. The hair usually begins to fall out 1 week after the tick bite, coinciding with induction of telogen hairs.[3] Necrosis at the site of the tick bite is frequently observed, surrounded by a 1- to 4-cm patch of alopecia.[20,21] Exclamation mark hairs similar to those observed in patients with alopecia areata have been reported.[20] Regrowth begins within 2 weeks and is complete within 2 months.[3] Scarring centrally from the original tick bite may result in residual scarring alopecia.

### Tick-Bite Reactions

The most frequent sites of tick-bite reactions in children are the head and neck; in adults, the trunk and legs.[3] Local effects include swelling, erythema, paresthesia, blistering, pruritus, ecchymosis, induration, nodule formation (prurigo-like lesions), and necrosis (Figure 9-4). *Ixodes racinus* bites may cause local gangrene, erysipelatoid swelling, generalized erythema, generalized urticaria, or psoriasiform eruptions. Healing of tick bites usually occurs in 2 to 3 weeks but may take longer. Systemic symptoms include nausea, vomiting, diarrhea, pulse irregularities, dyspnea, fever, gastrointestinal irritation, restlessness, muscular weakness, drooping eyelids, photophobia, delirium, hallucinations, and generalized pain of tick typhus. Some of these symptoms overlap with those reported for tick paralysis. Most of these complaints resolve with removal of the tick.[3]

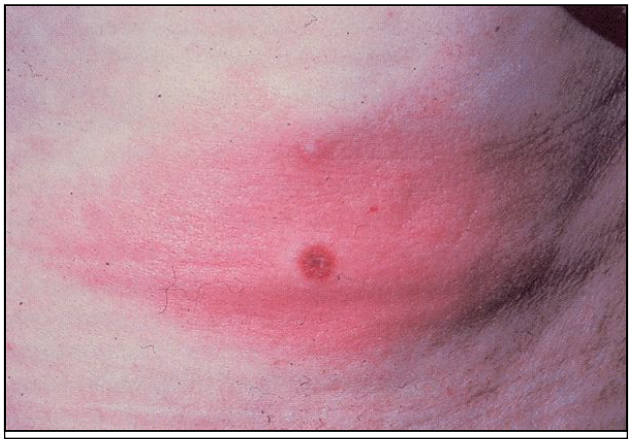

**Fig. 9-4**. Tick-bite reaction. These lesions are characterized by a large zone of erythema.

## FLEAS

Fleas evolved as highly specialized, bloodsucking parasites at least 60 million years ago.[22] Their ancestors may originally have had wings, but these would have inhibited movement through the host's fur; thus, jumping provided an alternative means of locomotion. Fleas are amazingly resilient: various species can jump 150-fold their own length (vertically or horizontally equivalent to a human's jumping 900 ft); survive months without feeding; accelerate 50-fold faster than the space shuttle; withstand enormous pressure; and remain frozen for a year, then revive. The amazing ability of the flea to jump so well is due to a superelastic protein located in the thorax known as resilin. The leg and thorax muscles compress a tiny pad of resilin, which is suddenly released, resulting in the explosive unleashing of kinetic energy.[22]

More than 2,400 species and subspecies have been described. The order Siphonaptera contains only 2 flea families of medical importance: Pulicidae (human, cat, dog, and bird fleas) and Sarcopsylidae (also called Tungidae), the sand flea, which causes tungiasis.[22,23] The human flea is *Pulex irritans*. Fleas are wingless, laterally compressed insects with a hard, shiny integument. The body has 3 regions: the head, thorax, and abdomen. Mouth parts are modified (paired maxillary palpi) for piercing and sucking. The head is applied directly to the three-segmented thorax (no neck). A "comb" may be present or absent at the posterior margin of the first segment and is a major feature for classification (Figure 9-5). A pair of legs is attached to each thoracic

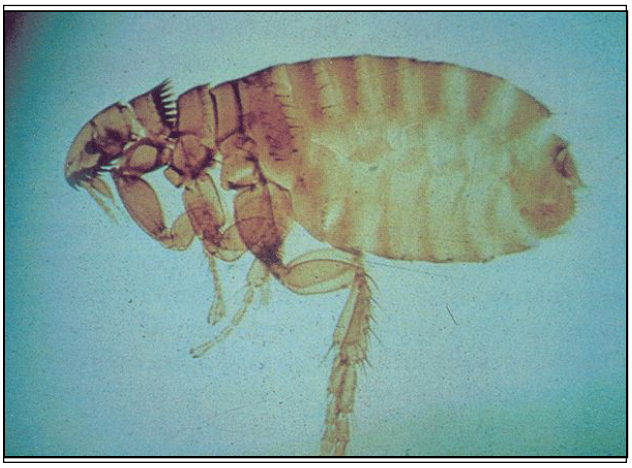

**Fig. 9-5**. *Pulex irritans*, the human flea. Note the comb at the posterior margin of the first segment, a major feature for classification.

segment, resulting in 3 pairs of legs. Each leg has 5 distinct parts. The abdomen has 9 to 11 segments.[23,24] Fleas require a blood meal to perpetuate their species. In some cases the blood meal must be from a specific host, but in others any suitable host is sufficient. Eggs are deposited on the ground. In some species, such as the rabbit flea (*Sipilpsyllus cuniculi*), the female flea responds to hormonal changes in the pregnant rabbit host, resulting in synchronized rabbit-flea procreation cycles. In animals with thick fur, fleas tend to infest for long periods of time, utilizing the warmth and protection of the fur. In humans, however, the flea is only a transient visitor for the purpose of feeding. The life cycle for adult fleas varies considerably: the human flea, *Pulex irritans*, lives 4 to 6 weeks; *Tunga penetrans* lives about 17 days. The individual stages can be modified by adverse conditions, extending the flea life cycle to beyond 200 days. Likewise, the pupal stage can vary from 7 days to 1 year. At all stages of development, the flea can withstand starvation for long periods. The stimulus for sudden, famished flea activity may be initiated by footstep vibrations. Fleas jump, on average, about 20 cm; however, when searching for food or attempting to escape enemies, they can reach a height of 2 m or more.[23] Fleas are important vectors of disease and transmit some of the most deadly infectious diseases ever known (Table 9-4).

**TABLE 9-4**

**FLEAS AS VECTORS OF HUMAN INFECTIOUS DISEASES**

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*Fleas are definite vectors.
†Fleas are possible vectors.
‡Ticks are usual vectors, but the infectious organism has been isolated from fleas.
Adapted with permission from Alexander JO. *Arthropods and Human Skin*. Berlin, Germany: Springer-Verlag; 1984: 159.

**Plague**

Plague is a zoonotic disease caused by *Yersinia pestis* and usually transmitted to humans by rodents and their fleas. The condition is characterized by a bubo, a mass of lymph nodes that become matted together and drain purulent material. A detailed discussion of plague can be found in Chapter 13, Bacterial Skin Diseases.

**Flea Bites**

Flea bites are the most common arthropod bites known. One flea can bite two to three times over a small area. It is not uncommon to see three flea bites in a row, described as breakfast, lunch, and dinner (Figure 9-6).[25] The bites produce irregular, pruritic, red wheals up to 1 cm in diameter. Some patients may present with a surrounding halo with a central papule, vesicle, or bulla. Others react by forming hemorrhagic macules, papules, vesicles, or bulla. In individuals immune to flea bites, an evanescent erythema with a central hemorrhagic punctum appears. A purpuric halo that persists for 3 to 4 days has also been observed. The lesions usually subside within 2 to 3 days. Bites usually are seen at points of access, such as the ankles or wrist; however, the first meal may occur at any site. The scalp and face are rarely affected.[23] Treatment is usually supportive with mild to moderate topical steroids.

**Papular Urticaria**

Chronic reinfestation by fleas may result in urticarial lesions that appear in irregular crops; these lesions are frequently observed on the limbs and around the waist. They have also been reported to involve the trunk, buttocks, neck, hands, and feet.[23] The distribution is characteristically bizarre, with lesions exhibiting two features: (1) new lesions appear in small groups, all in the same stage as earlier lesions, and (2) new lesions tend to cluster around the waistline, where tight clothing prevents further flea migration (Figure 9-7).[23] Postinflammatory changes and scarring from scratching are not uncommon. Papular urticaria has also been reported[23] to result from the bites of other arthropods, such as mites. Treatment involves removing the soldier from the infested area. In cases of severe pruritus, systemic steroids may be required.

**Tungiasis**

*Tunga penetrans*, also called sand flea, chigoe, and jigger flea, is the only member of the family Tungidae known to attack humans.[26] Tungiasis originated in Central and South America and was reported to infest sailors from Columbus' voyage in 1492.[23] It spread to Africa, where it was first recorded in

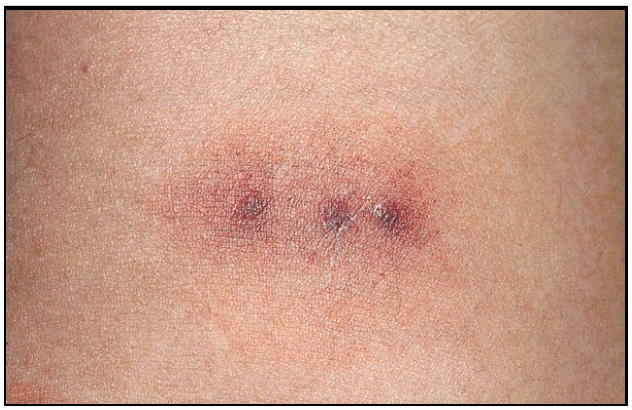

**Fig. 9-6**. These lesions resulted from flea bites. They display the characteristic "breakfast, lunch, and dinner" pattern.

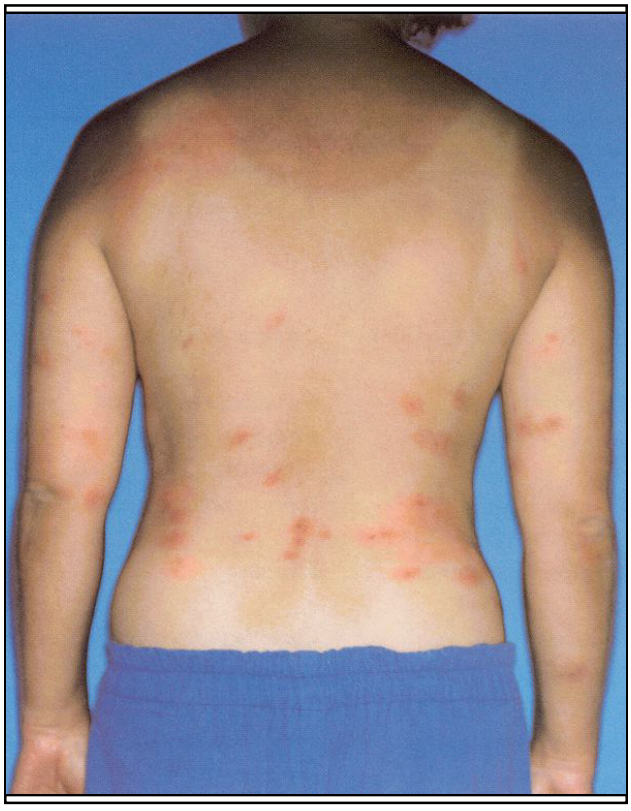

**Fig. 9-7**. Papular urticaria secondary to flea bites. These lesions tend to cluster around the waistline.

1634.[23] A major outbreak occurred in Brazil in 1872, and tungiasis was shortly thereafter introduced into Angola. It has been reported in Central and East Africa, Madagascar, the west coast of Pakistan, and India.[27]

Tungiasis is caused by the female sand flea, which burrows into human skin at the point of contact, usually the feet. The larva, adult males, and virgin females live in dry, sandy soil around pigsties, poultry runs, and stables, where they feed on vegetable matter. The sand flea is the smallest known flea (1 mm long) and has a 3- to 4-week life cycle. The male dies shortly after copulation. The gravid female jumps, reaching heights of 35 cm, in an attempt to find a suitable host. On contact with human or animal skin, the flea penetrates with its pointed head and burrows into the skin. The female enlarges to the size of a pea over a 7- to 14-day period. After discharging up to 300 eggs through the skin's surface, the female flea collapses and dies.[23,26]

Tremendous flea burdens can incapacitate an individual by causing pain and superinfection. Individual lesions consist of a firm, indurated, slightly tender nodule with a flat surface of whitish-yellow keratin (Figure 9-8). A minute black spot in the center (the posterior end of the flea abdomen) may be noted. Secondary infections may result in adenopathy, cellulitis, and erysipelas. Thrombo-phlebitis, bone necrosis, and autoamputation of toes have been reported.[23] Tetanus and gas gangrene are also potentially life-threatening complications.

Treatment of tungiasis involves surgical removal of the parasites. During the first 48 hours after the female imbeds herself, a sterile needle may be sufficient. Curettage and cautery frequently works for intermediate stages of development. However, the entire flea that has been in place for 7 to 14 days should be surgically removed. Antibiotic treatment may be required for secondary infections.[23]

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**Fig. 9-8**. Primary cutaneous lesion caused by *Tunga penetrans*. Note the white circular area with central black dot. Reprinted with permission from Zalar GL, Walther RR. Infestation by *Tunga penetrans*. *Arch Dermatol*. 1980;116:81. Copyright 1980, American Medical Association.

In areas where tungiasis is endemic, use of good protective footwear should be encouraged, and for the soldier in the field, boots are adequate. Individual cutaneous inspections should be encouraged. Known infested sites should be treated with lindane, dimethyl phthalate, or dimethyl carbamate. Use of flypaper low to the ground to collect jumping fleas is another measure that may help to curb disease, and also is a good test for determining the effectiveness of the insecticide being used.[23]

## OTHER INSECTS

### Lice

Pediculosis, or lice infestation, is caused by three types of lice: *Pediculus humanus capitis* (head louse), *Pthirus pubis* (pubic or crab louse), and *Pediculus humanus corporis* (body louse). After attaching to the skin, these flattened, wingless insects feed on human blood and can cause intense itching. For a detailed discussion of pediculosis, see Chapter 8, Arthropod and Other Animal Bites.

### Bedbugs

Members of the insect order Hemiptera include bedbugs, which are vectors for the transmission of *Trypanosoma cruzi*, the infective agent for Chagas' disease (also called South American trypanosomiasis), which is discussed in Chapter 12, Tropical Parasitic Infections.[28] Its best-known vectors are the assassin bugs, family Reduviidae, including *Rhodnius prolixus* (northeastern South America) and

*R pallescens* (Brazil and Panama). Other Hemiptera, such as *Triatoma infestans* (Argentina and Brazil), *T barberi* (Mexico), *T dimidiata* (Mexico, Ecuador, and Central America), *T maculata* (Venezuela), and *Panstrongylus megistus* (Brazil and Guyana) are also important vectors of Chagas' disease.[29]

**Biting Flies**

Infectious diseases may also be transmitted to humans by dipterous insects, which include biting flies and mosquitoes. A detailed list of mosquitoes as vectors of viral diseases is provided in Chapter 10, Viral Hemorrhagic Fevers. Myiasis (infestation with fly maggots) is discussed in Chapter 8, Arthropod and Other Animal Bites.

Bartonellosis (Carrion's disease), with its two clinical forms, Oroya fever and verruga peruana, is transmitted by sandflies, *Lutzomyia verrucarum* and *L colombiana* (Figure 9-9). The bacterial agent is *Bartonella bacilliformis*, which is restricted to endemic areas.[26] Both visceral (kala azar) and dermal leishmaniasis are transmitted to humans by sandflies, *Phlebotomus* species (Old World) and *Lutzomyia* species (New World). Leishmania organisms are flagellate protozoa in the genus *Leishmania* and are composed of two visceral forms (*Leishmania donovani* and *L infantum*) and three cutaneous forms (*L tropica*, *L braziliensis*, and *L mexicana*).[30]

The large collection of flies in the family Tabanidae contains over 3,000 species.[29] They are blood-sucking flies and include horseflies, deerflies, clegs, breeze flies, greenheads, and mango flies.[25] Tabanidae are important vectors for the spread of the microfilaria *Loa loa* (loiasis) in Africa, and the bacterial pathogens of anthrax (*Bacillus anthracis*) and tularemia (*Francisella tularensis*) in the United States.[25,4] They are also important vectors for the spread of animal trypanosomiasis worldwide. Glossinidae are composed of 22 species of the tsetse fly, which is well known for transmission of African trypanosomiasis.

**Fig. 9-9.** The sandfly is capable of transmitting bartonellosis as well as dermal and visceral leishmaniasis.

## SUMMARY

Organisms from the arthropod classes Arachnida and Insecta have a hard-jointed exoskeleton and paired, jointed legs. In most instances, arthropod bites are nothing more than a nuisance, resulting in localized, cutaneous reactions and pruritus. However, some of these organisms are medically important: fleas, lice, and ticks can transmit lethal epidemic disorders, such as bubonic plague, typhus, Crimean-Congo hemorrhagic fever, Rocky Mountain spotted fever, and boutonneuse fever.

Many of these vector-transmitted diseases are endemic in various regions of the world. Medical officers need to know which of these ancient afflictions are endemic to the region and treat infected troops appropriately. Medical officers also need to be aware of other medical complications of arthropod bites, such as tick-bite alopecia and tick paralysis. Tick paralysis may be particularly challenging, and when progressive, lower-extremity weakness is present, a vigorous search should be undertaken to find and remove the tick. This simple procedure can be lifesaving.

In a fast-moving wartime environment, regional control with pesticides is unlikely to control insect vectors. It is thus up to the individual soldier to minimize exposure. Reducing exposure can be accomplished through repeated use of permethrin-based repellents or diethyltoluamide (DEET) applied to clothing or the skin. In addition, permethrin-impregnated uniforms are even more effective than DEET applications to skin or uniforms. Therefore,

uniform impregnation with permethrin-based repellents should be strongly considered in field environments where the risk of vector-transmitted diseases is high.

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