Mammalian Diseases and Arachnids

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Chapter 4

DISEASES OF MAN

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I. INTRODUCTION

Arachnids may cause various disorders in man. Spiders, ticks, and scorpions inject venom or toxins into the body, the first two by means of their chelicerae and the latter by a caudal sting. Among the mites of the groups Mesostigmata, Prostigmata, and Astigmata, the repeated contacts of both living and dead mites with the skin or the respiratory mucosa may produce an allergic reaction in these organs leading to contact dermatitis or bronchial asthma. Moreoever, in the Mesostigmata and Prostigmata, dermatitis may also result from the direct toxic action of the saliva injected by these mites by means of their piercing chelicerae. *Sarcoptes scabiei* is an obligate parasite colonizing the skin and producing mange. Several other Acari, especially among Prostigmata and ticks, are vectors of pathogenic agents, such as viruses, rickettsiae, bacteria, or protozoa.

II. SCORPIONS (SCORPIONIDA)

Scorpions have a more or less lobster-like appearance. The body is clearly divided into two parts: an anterior flat and broad region and a posterior narrow, cylindrical, tail-like portion. The broad part is formed by the fusion of the short nonsegmented cephalothorax with a longer segmented abdomen also called the preabdomen. Only the cephalothorax bears appendages, consisting of a small pair of chelicerae, a conspicuous pair of pedipalps ending in powerful pinchers, and four pairs of walking legs. The posterior part, or postabdomen, is divided in six segments; the last segment is bulbous and terminates in a curved hollow sting with sharp apex. This last segment is called the telson and contains a pair of venom glands. The size of scorpions varies from about 2 to 3 cm to about 15 to 25 cm. For more complete informations on the morphology see Reference 1 (for biology, Chapter 3, Volume I).

A. Geographical Distribution

Scorpions are widespread, but they are most common in warm countries. All of the most dangerous species for man belong to the family Buthidae and are distributed only in a few, desert or semidesert regions, especially South America, Mexico, North Africa, and some parts of the Middle East.

The genera containing these dangerous species for man are distributed in the following areas:[1-3]

1. Genus *Tityus*: Mexico, Central, and South America
2. Genus *Centruroides*: South of U.S. (Arizona, California), West-Indies (Cuba, Jamaica, Haiti, Puerto-Rico, etc.), and Central America
3. Genus *Buthus*: North Africa, parts of the Middle East, Egypt, Ethiopia, Somalia, and Manchuria

4. Genus *Parabuthus*: "parts" of South Africa
5. Genus *Buthotus*: North Africa and the Middle East
6. Genus *Buthacus*: North Africa, Senegal, and the Middle East
7. Genus *Androctonus*: from India to the Atlantic coast of Morocco, from the Middle East and Egypt to Senegal
8. Genus *Scorpio*: North Africa
9. Genus *Tamulus*: India

The most dangerous neotropical species are *Tityus serrulatus, T. bahiensis, T. trinitatis, T. trivittatus, Centruroides suffusus, C. limpidus, C. norius, C. sculpturatus*, and *C. vittatus*. In the Old World, the most dangerous species are *Buthus occitanus, Buthacus arenicola, Androctonus australis, A. amoreuxi, A. aeneas*, and *A. hoggarensis*.

B. Epidemiology

Most of the scorpions are nocturnal in their habits. During the daytime, they hide under rocks and stones, in cracks, or in various debris. Some species have "domiciliar habits" and may enter the houses where they hide among clothing, etc.

Scorpion stings occur generally at night, in houses, gardens, and in tents, when they are stepped on accidentally with bare feet. Therefore, the limbs are the parts of the body most frequently stung.

Mortality is especially high in very young children. In Brazil, *T. serrulatus* causes mortality of 0.8 to 1.4% in adults, 3 to 5% in school-age children, and 15 to 20% in very young children.[3] In Mexico the annual deaths from scorpion stings range from 1100 to 1900.[4]

C. Pathology and Symptomatology

The sting of the highly venomous scorpions causes a sharp local burning pain accompanied occasionally by a local swelling. Salivation, profuse sweating, nausea and vomiting, mydriasis, and tachycardia are generally observed. The temperature is variable and may be either higher or lower than normal. There can be either hyper- or hypotension. The patient is often agitated and the muscular tonus is increased and can result in contractures or spasms, especially of the limbs, the abdominal muscles, and the pharynx. Convulsions similar to strychnine poisoning are sometimes observed. This reaction occurs more frequently in young children. The respiratory rate is generally increased and the rhythm may be altered. A Cheyne-Stokes type is sometimes observed. Death is caused by paralysis of the respiratory system resulting from an action on the respiratory centers. Consciousness is not altered at the beginning, but in the hours following the envenomation, nervous or mental symptoms may appear, such as obnubilation, hallucination, temporary eye paralysis, hemiplegia, etc.

The evolution of the envenomation varies according to the species of scorpions involved. Death may occur in 2 to 20 hr. A sudden aggravation of the symptoms after an apparent recovery is frequently observed in scorpion envenomation and, therefore, a close surveillance of the patients must continue for at least 12 hr after disappearance of symptoms.[2] The venom of the scorpion is a mixture of various toxins, mainly neurotoxin, but also hemorrhagin, hemolysin, coagulant, etc.

The neurotoxin is responsible for paralytic and nervous symptoms leading to death by respiratory paralysis. The neurotoxin has also a selective stimulating action on the sympathetic and the parasympathetic centers of the hypothalamus, and its action resembles that of sympatheticomimetics (adrenaline) and parasympatheticomimetics (muscarine, acetylcholine, pilocarpine, and eserine).[2]

D. Treatment

Treatment is similar to that for snake bites. It should be started as soon as possible after

the bite. The patient should be placed completely at rest, a tourniquet applied, the skin incized, and sucking started. The tourniquet must be released after 30 min and replaced for another 30 min. The effectiveness of cryotherapy is still controversial.

Antivenin is generally available in regions where dangerous scorpions are present. It should be injected within an hour or two after the sting.

Analgesics, antihistaminics, and corticoids may be useful and reduce the symptoms. Artificial respiration should be utilized if respiratory paralysis is expected.[4-5]

III. SPIDERS (ARANEIDA)

The body in spiders is divided in two unsegmented segments: the cephalothorax and the abdomen. The cephalothorax bears a pair of chelicerae, a pair of pedipalps, and four pairs of walking legs. Each chelicera is composed of two segments, a thick basal segment and a curved claw or fang.

The venom glands are located in the anterior region of the cephalothorax except in the tarantulas where they are situated in the basal segment of the chelicerae. The poison duct traverses the claw and opens near its tip.[1]

Spiders, like scorpions, are carnivorous animals and they feed upon insects, spiders, and other small animals which they kill by injecting their venom. For a more complete description of the spider anatomy see References 6 and 7 (or Chapter 4, Volume I).

A. Geographical Distribution

The spiders dangerous to man belong to the following groups:

Widow spiders — They belong to the cosmopolitan genus *Latrodectus* (family Theridiidae). According to Levi,[8] this genus contains six species; the most important is *Latrodectus mactans*, the black widow. The typical form of this species is most abundant in the warmer parts of the Americas (from U.S. to Argentina and Hawaii). This species is also encountered in other parts of the world and is divided into four subspecies: *L. mactans tredecimguttatus*, in the Mediterranean region, Arabia, and Ethiopia; *L.m. hasselti*, from India to Australia and New Zealand; *L.m. menavodi*, in Madagascar; *L.m. cinctus*, in South and East Africa and Ethiopia.[8]

The five other species of *Latrodectus* are *L. pallidus*, in the eastern Mediterranean area and Middle East; *L. curacaviensis*, in the Americas; *L. geometricus*, the brown widow, is cosmotropical, mainly in Africa, but also in southern Florida; *L. hystrix*, in Yemen; and *L. dahli*, in Iran and the Sokotora Islands.

Tarantulas — In Europe the name "tarantula" is utilized for an indigenous harmless species *Lycosa tarentula* (family Lycosidae). In Italy during the Middle Ages, the bite of this species was supposed to cause a condition, known as tarantism (from the name of the Italian city Taranto), that could only be cured by frenzied dancing to exhaustion. This dance was known as the tarantella and was accompanied generally by manifestations of mass hysteria. In fact *L. tarentula* is an harmless spider and probably the general symptoms described in some cases of tarantism in Italy were caused by *Latrodectus mactans tredecimguttatus*, the malmignate, a much less conspicuous, but more dangerous spider. In the U.S., the name tarantula is applied to large or very large hairy mygalomorphs, and not aggressive spiders, of the genera *Avicularia*, *Grammostola*, etc. (family Theraphosidae). They are also called "bird-eating spiders" or "bird spiders". Most of these American tarantulas, from Mexico, Central America, and Trinidad, supposedly venomous, are harmless to man.[9] Only a few tropical tarantulas are poisonous to man, especially some species of the genera *Aphonopelma*, *Sericopelma*, and *Lampropelma*. In several megalomorphs, however, urticaceous hairs will produce marked dermatitis.

Funnel-web spiders or "Mygales fileuses" — The most important genus of this group

is *Atrax* (family Dipluridae) containing large mygalomorph, aggressive, and very toxic species. Some are capable of jumping. The most important species are *Atrax robusta* and *A. formidabilis*. This group is restricted to Australia and New Zealand.

Wandering or banana spiders — The most dangerous representatives of this group belong to the genus *Phoneutria* (family Ctenidae) and are found in South and Central America. The most important species is *Phoneutria nigriventer*. It is large, very aggressive, and very toxic.

***Harpactirella* species (family Barychelidae)** — These have caused fatalities in South Africa. They are aggressive and enter homes in Cape Town.

Violin spiders or brown spiders — They belong to the genus *Loxosceles* (family Sicariidae or Scytodidae) and are known from the Americas. The body is 10- to 15-mm long and brown in color. The most important species are the brown recluse *Loxosceles reclusa*, from the U.S., and *L. laeta* and *L. rufipes* from South America. *Loxosceles rufescens* has also been recorded from Australia probably introduced from the Mediterranean region.

Genus Lycosa sp. (family Lycosidae) — Only some species from Central and South America are dangerous.

Genus *Chiracanthium* spp. — These of the family Clubionidae are probably now worldwide in distribution. Some dozen species are known from Australia, several in the U.S. and Europe, and at least one in Hawaii (*Chiracanthium mordax* probably from Australia). Chiracanthism, similar but less severe than loxoscelism, is a common disease because these spiders usually seek out homes or nearby shrubbery for their living area[3] (see Chapter 4, Volume I).

B. Epidemiology

Among the 100,000 known species of spiders, only a few are dangerous to man. The term "arachnidism", or more conveniently "araneism", are given to the envenomation by spiders. More correct names are "latrodectism, loxoscelism, atraxism, etc." because they refer to the genus of the spider involved in the bite and thus to the particular symptoms they cause.[1]

Most of the poisonous spiders enter the houses and may hide in the clothing. The aggressiveness varies with the spider, the most aggressive belong to the genera *Atrax, Phoneutria*, and *Harpactirella*. Some spiders have a life span of more than 30 years. All are carnivorous.

C. Pathology and Symptomatology

1. Latrodectism or Neuromyopathic Araneism

Latrodectus mactans is a rather small spider; the body of the female is 10- to 13-mm long; it is coal black in color, with a scarlet or orange hourglass pattern on the ventral surface of the abdomen. Only the female is known to bite man. The venom has a neuromyopathic action containing a neurotoxin resembling that of the snake *Naja* and a toxin causing spasms.

The local symptoms consist of sharp burning pain with little or no swelling. Later, weakness and abdominal cramps develop. Muscles are very painful, rigid, and present spastic contractions, especially those of the abdomen and the chest. Other symptoms include nausea, vomiting, profuse perspiration, lacrimation, and difficulty in breathing and speech. Convulsions may be observed in children. Sometimes complete prostration occurs. Complications when present consist of lesions in the liver, spleen, and kidneys.

In the U.S. among 1291 cases of black widow bite from 1726 to 1943, 55 were fatal (4.25%).[10] According to Horen, "ninety per cent of the cases of latrodectism occurred in Texas in men who were bitten while using outdoor privies".[11] (Privies are now rare in Texas! Editor.)

More than 30 fatal cases have been recorded from the other parts of the world, some from

South America caused by *L. mactans mactans* and others from the Old World (Europe, South Africa, Madagascar, and Australia) caused by the other subspecies of *L. mactans*.[1]

Treatment — A tourniquet is useful only if it is applied within a few minutes after the bite. I.V. injection of calcium gluconate, hot baths, and injection of morphine can reduce the severe muscular spasms and pain. *Latrodectus* antivenom when available is effective against the different subspecies of *L. mactans*.

2. Necrotic Araneism (Especially Loxoscelism)

The spiders of the genus *Loxosceles* are exclusively nocturnal. During the daytime they hide under stones, bark of trees, and various other objects. They are typically found in houses where they may hide in clothing or in bedding.

Most bites occur in the home when the person is in bed or is dressing. This spider is not aggressive and the bites are accidental. The *Loxosceles* venom is cytolytic and hemolytic. The bite is followed by an immediate and severe pain. The bitten area becomes swollen and whitish by vasoconstriction. After a few hours, blisters appear and a reddish thick margin develops around this zone. Later, the center of the lesion becomes hemorrhagic and then necrotic. The skin plus the subcutaneous fatty tissues or muscles may be destroyed. Healing is slow and leaves a large scar.

Systemic reactions are inconstant, but in young children they may be severe. They consist of intravascular hemolysis and congestive hemorrhagic lesions, especially in liver and kidneys.

Chiracanthium spp. (see Section III.A) produce nearly identical envenomation and skin reactions although much less severe than in loxoscelism. Signs and symptoms are in all aspects reduced from that of loxoscelism and thus rarely severe (see Chapter 4, Volume I).

Treatment — The administration of corticosteroids for loxoscelism may be useful in the few hours after the bite.[5] Surgical excision of the bite area within the first day of the venomation is probably the most effective treatment. *Loxosceles* antivenom is produced by the Instituto Butantan, Sao Paulo, Brazil, but is not extensively used.[3] In even severe cases chiracanthism treatment with corticosteroids will probably be adequate (more research needed). To date mild cases need no treatment.

*3. Envenomation by Some Other Dangerous and Aggressive Spiders**

Phoneutria nigriventer — This very aggressive species is found in South and Central America. Adult females have an average of about 35 mm in total body length. The venom of this species has a neurotoxic action on the central and the peripheral nervous system. It causes intense pain, sweating, priapism, and respiratory paralysis. An antivenom is available from the Instituto Butantan, Sao Paulo, Brazil.[1]

***Atrax* spp.** — These spiders are very aggressive, especially the males, and fatalities have been recorded from their bites. Length of the body can attain 37 mm. The venom is mainly neurotoxic and is not antigenic; therefore, treatment is only symptomatic.[3]

***Harpactirella* spp.** — These spiders are feared in South Africa because they are very venomous and aggressive and enter homes.[1] The body is 20- to 30-mm long. The venom is mainly neurotoxic as in the genera *Phoneutria* and *Atrax*. Patients usually clear of symptoms after 24 hr without treatment (Chapter 2, Volume II).

Control of spiders — Spiders are sensitive to the usual insecticides.

IV. ACARI (ACARIDA**)

We are using here the approach proposed by Evans et al., who consider the Acari as a

* See also Chapter 2, Volume II.

** Editorial designation.

subclass of the Arachnida.[12] The Acari are further divided into two superorders, the Anactinochaeta and the Actinochaeta. The Anactinochaeta consist of four orders, of which two are medically important: the Metastigmata (or ticks) and the Mesostigmata. The Actinochaeta are also formed of two orders of medical importance: the Prostigmata and the Astigmata. It is customary to study separately the ticks (Metastigmata) and the mites (other groups of Acari). The differences between these two groups, however, are more practical than taxonomical as they are based mostly on the considerable economic importance of the ticks and to a lesser extent, their medical importance. Ticks are in fact important vectors and are very often the most efficient reservoirs of many pathogens. Other differences consist of the fact that ticks, unlike mites, form an homogenous group, all members of which are parasitic on vertebrates and are macroscopical in size, at least in the adult stage. In addition, ticks, in contrast to most of the mites, have a long life span, commonly a year, but very often much longer, up to 5 and 6 years in some species. This high longevity is of great importance for the maintenance of pathogens in nature and confers to ticks a particularly efficient role as reservoirs of many viruses, rickettsiae, and bacteria.

A. Ticks (Metastigmata)

Ticks transmit numerous viruses, rickettsiae, bacteria, and protozoa to animals and humans. Moreover, certain species of ticks may inoculate toxins in their hosts, causing paralysis or toxicosis. In animals, chiefly livestock, ticks also cause severe anemia and loss of body weight. We deal here with the disorders and diseases caused by ticks in man. Ticks are divided into two families: Argasidae, or "soft ticks", and Ixodidae, or "hard ticks". For discussion on the taxonomy of these families, we refer to the many publications of Hoogstraal (see Chapter 6, Volume I or Appendix 4).

1. Tick Paralysis and Toxicosis

Tick paralysis or toxicosis in animals or humans has been observed in various parts of the world.[13] About 43 species of ticks, belonging to 10 genera, are involved in these syndromes. Female ticks are more frequently involved with paralysis than males. In human paralysis, *Dermacentor andersoni* and *D. variabilis* are the most important species in North America, and *Ixodes holocyclus* in Australia.[14] Three human cases caused by *I. hexagonus* have also been recorded in France and in England.[3]

Symptomatology — Tick paralysis is essentially a symmetrical ascending flaccid and afebrile paralysis. The ticks producing paralysis in humans are generally hidden in the hair and may be overlooked. They must be attached for some time before paralysis develops. This incubation period corresponds with the feeding of the tick and lasts for 5 to 7 days. First symptoms are lassitude, difficulty of walking, headache, and vomiting. These are followed by progressive paralysis, beginning in the lower limbs. During the ensuing hours, the paralysis intensifies and extends to the trunk, upper limbs, and the head. The muscles of the face and the throat are involved and the patient is unable to move the arms and legs, and speech, swallowing, and breathing become more and more difficult. Paralysis is of the flaccid type and is accompanied by abolition of tendinous reflexes. The temperature is normal. The paralysis is reversible when the tick is removed in time. Some argasid ticks as *Ornithodoros* or *Argas* do not cause paralysis in man,[14] but their bite is painful and may produce severe reactions in this host.[3]

Mechanism of the paralysis — The paralysis is caused by a toxin present in the saliva of the tick. Experimental research has shown that the mechanism of paralysis varies according to the species of tick involved. In *Dermacentor* and *Argas* paralysis, there is a marked reduction in the conduction in the nerve fibers as well as a decrease of potentials of these nerves and of their corresponding muscles and an impairment of impulse propagation of afferent fibers. In *I. holocyclus*, the peripheric nerves are not impaired and the paralysis is

due to a direct temperature-dependent inhibition of evoked acetylcholine release at the neuromuscular junction.[14]

Prognosis and Treatment — A single female tick can completely paralyze and kill an adult human by respiratory paralysis if it is not removed in time. If the tick is removed promptly, the patient will recover in a few days. In cases of extensive paralysis, the recovery may take several weeks. Specific serum is indicated only for *I. holocyclus* paralysis. This serum is specific and is not active for tick paralysis caused by other ticks.[13-14]

2. Transmission of Tick-Borne Viruses

Ticks are efficient reservoirs for many viruses. Their life span is commonly a year or often much longer, and viruses may survive in their body for months or years. Transstadial passage of the virus, from larva to nymph to adult, is common, and transovarial passage from the infected female tick to its progeny is also possible, but less common. These biological properties greatly increase the maintenance and the spread of the virus in nature.

Numerous wild or domesticated vertebrates, either mammals or birds, are natural reservoirs for many tick-borne viruses. Some viruses, such as tick-borne encephalitis, normally transmitted by ticks, may proliferate in goat and bovine mammary glands and man may become infected by consuming infected milk or cheese.

Tick-borne viral diseases in man range from a mild fever to severe hemorrhagic fever or encephalitis with a high mortality rate. Up to now, 68 viruses have been isolated from 60 species of ixodid ticks and from 20 species of argasid ticks. Of these, 21 are pathogenic to man; quite all have been isolated from ixodid ticks.

A list of the viruses pathogenic to man with some epidemiological data for the more important ones is given here. These viruses fall into the following antigenic groups.[15-16]

Russian spring-summer encephalitis (RSSE) virus (Group B) — This complex is formed of 9 viruses. The disease caused by RSSE represents a complex of several closely related antigenic entities. The typical form is observed in the marshy taiga (Siberian forest), and *Ixodes persulcatus* is the chief vector of the virus. The incidence of natural infections in this tick is often high. Clinically it is characterized by a severe polio-encephalomyelitic syndrome with paralysis, but without hemorrhagic episodes. The mortality rate is high, up to 25 to 30%. Taiga encephalitis has also been recorded from Prussia and East Germany. In other regions of Asia, variants of RSSE may occur somewhat intermediate between RSSE and TBE. In these places (eastern U.S.S.R. and China), the disease may be transmitted by ingestion of infected milk, and other species of ticks have been found infected.

Central European tick-borne encephalitis (TBE) virus — The encephalitis caused by this virus is much milder than RSSE and has a low mortality rate. This disease is present within the range of *Ixodes ricinus*, which is the main vector. It has been recorded from forests or rural areas in Central Europe: western Russia, Finland, Czechoslovakia, Poland, Yugoslavia, Hungaria, Austria, Switzerland, Germany, and Sweden. The incidence of the virus in *I. ricinus* is low. Several other ticks, especially *D. marginatus* are secondary natural hosts of this virus. Infections of humans results from tick bite, but also frequently from ingestion of fresh goat's milk. This virus has been isolated from wild birds, rodents, hares, and insectivores in Central Europe. The mole *Talpa europea* is an important host for TBE virus in Czechoslovakia.

Other viruses of the RSSE complex — Louping ill (LI) virus causes high mortality in sheep and can also infect cattle and man. In humans, the disease resembles TBE encephalitis, Negishi encephalitis (NEGE) virus found in Japan; Omsk hemorrhagic fever (OHF) virus observed in Siberia; Kyasanur Forest disease (KFD) virus in India; Langat encephalitis (LANE) virus in Malaysia; Royal farm (RF) virus in Afghanistan; and Powassan encephalitis (POWE) virus in the U.S. and Canada.

Crimean-Congo hemorrhagic fever (CCHF) group — This virus may cause a fatal

disease in man. It has a wide distribution and has been found in Eurasia (from Bulgaria to the U.S.S.R. and Pakistan), Tropical Africa (from Senegal to Kenya), Egypt, and in India.

Kemerovo group — Two viruses of this group, Kemerovo (KEM) virus in the U.S.S.R. and Siberia and Tribeč (TRB) virus in Czechoslovakia and Hungary, have been isolated from humans.

Ganjam group — It contains two viruses causing fevers in man: Ganjam (GAN) virus, in India, and Dugbe (DUG) virus, in Tropical Africa.

Quaranfil group — With Quaranfil (QUA) virus, from Africa and Asia, it has caused fever in humans.

Hughes group — Among the six viruses of this group, only two have caused fever in humans: Punta Salinas (PUNS) virus, in Peru, and Zirga (ZIR) virus, in the Arabian Gulf.

Ungrouped viruses — Four viruses of this group have been found in humans: Colorado tick fever (CTF) virus, transmitted by *Dermacentor* spp. and causing a dengue-like fever in man in the western U.S.; Bhanja (BHA) virus in India, Nigeria, and Italy; Thogoto (THO) virus isolated from various ticks in Egypt, Nigeria, and Italy; and Nairobi sheep disease (NSD) virus transmitted transovarially by *Rhipicephalus appendiculatus* in Kenya and Uganda.

3. Transmission of Rickettsial Diseases by Ticks

Ticks are also vectors and reservoirs for several rickettsia organisms causing important diseases in man.[3,17]

Rocky Mountain spotted fever (tick-borne typhus, Mexican spotted fever, Sao Paulo fever, Tobia fever) — The disease is widely distributed from North to South America. The agent is *Rickettsia rickettsi*; the main vectors and reservoirs are *D. andersoni* and *D. variabilis*, but other ticks could be accessory vectors.[33] The transmission of the *R. rickettsi* in the tick is transstadial and also commonly transovarial. Small rodents and rabbits are possible reservoirs of this pathogen. Symptoms consist of an extensive rash appearing between the 2nd and the 5th day after onset, headache, intensive aching in the lumbar region, and fever. In severe cases, the temperature rises up to 40°C or higher. Mortality was high before the use of antibiotics. A vaccine is available.

Boutonneuse fever (Marseilles fever, South African tick typhus, Kenya tick typhus, Crimean tick typhus, Indian tick typhus, tick typhus) — This disease is specially widespread in Africa and in the Mediterranean regions. It has also been observed in India. The pathogenic agent is *Rickettsia conori*. Several ixodid ticks are vectors and reservoirs. Rodents, hares, and birds may also harbor this pathogen. In Africa and in the Mediterranean region, *Rhipicephalus sanguineus* is one of the most important vectors. Clinically there is an extensive rash, headache, and fever. At the site where the tick is attached to the skin, a characteristic lesion develops resembling a black button with necrotic center. This lesion has given its name to the disease ("boutonneuse"). Generally the lymph glands corresponding to the lesion are swollen. In spite of the acute evolution of the disease and the prolonged convalescence, the prognosis is good and fatalities are rare.

Siberian tick typhus (North Asian tick typhus) — This form of typhus occurs in Siberia and resembles both clinically and serologically the Rocky Mountain spotted fever, but has milder symptoms. The pathogen is *Rickettsia siberica* and several species of ticks are vectors or reservoirs, including the genera *Dermacentor* and *Haemaphysalis*.

Queensland tick typhus — This mild febrile condition resembles the rickettsial pox described from the U.S. This disease is known along coastal Queensland. Its agent is *Rickettsia australis*, and *I. holocyclus* is probably the main vector.

Q-Fever — The agent of this widely distributed and generally mild disease is *Coxiella burneti*. Q-fever is primarily a zoonosis, transmitted directly to man by inhalation of dust or ingestion of milk or more rarely by ticks. The latter may act as a reservoir for the pathogen.

4. Transmission of Bacteria by Ticks

a. Tick-Borne Relapsing Fevers

Soft argasid ticks of the genus *Ornithodoros* are vectors throughout the world of *Borrelia* spp., the agents of relapsing fevers in man. These diseases are endemic in Central and South Africa and in many parts of Asia and America. We list them here as: *Borrelia duttoni* transmitted by *Ornithodoros moubata* in Central and Southern Africa; *B. hispanica* by *O. erraticus* in North Africa; *B. persica* by *O. tholozani* in Uzbekistan and Kashmir to Cyprus and Tripoli; *B. crocidurae* by *O. erraticus sonrai* in Africa, the Near East and Central Asia; *B. caucasica* by *O. verrucosus* in the Caucasus mountains; *B. hermsii* by *O. hermsi* in the western U.S. and Canada; *B. turicata* by *O. turicata* in the western U.S., Kansas to Mexico; *B. parkeri* by *O. parkeri* in the western U.S.; *B. venezuelensis* by *O. rudis* in central and northern South America; and *B. mazzottii* by *O. talaje* in Mexico, Panama, Colombia, and Guatemala.[3]

Ornithodoros may act as vector and as reservoir. Transovarial passage may persist for several generations. Man becomes infected either by the bite of the tick or by the coxal fluid. The *Ornithodoros* spp. have a very long life span and can survive starvation for several years.

b. Tularemia (Rabbit Fever)

Tularemia is a disease of lagomorphs and rodents caused by a bacterium *Francisella tularensis* and is transmitted either by direct contact or by several arthropods, mainly ixodid ticks, which are also efficient reservoirs. Transstadial and transovarial transmission of the pathogen have been demonstrated experimentally in *D. andersoni*. Man may be infected by skinning rabbits or rodents or by the bite of ticks or deer flies. The disease is known from North and South America, Asia, Europe, and Africa. Symptomatology recalls plague, but mortality is low. Antibiotics, especially streptomycin, are effective.

5. Transmission of Protozoa

Human cases of babesiosis were first described in Europe in splenectomized persons. The disease clinically resembles malaria. The pathogen was *Babesia divergens*, a parasite of cattle transmitted by *Ixodes ricinus*. More recently, a series of new cases were described in nonsplenectomized persons originating from islands near Cape Cod, especially Nantucket Island, in the U.S. The pathogen is *B. microti*, a cosmopolitan parasite of rodents, very common in *Peromyscus* sp. in Nantucket Island. The vector is an *Ixodes* sp. similar to *I. scapularis*.

B. Mites (Meso-, Pro-, and Astigmata)

Mites may cause various types of disorders in man. Some species provoke a dermatitis and some cause scabies. Others are vectors of rickettsioses. A few years ago, it has been found that some species of mites living in house dust are important producers of allergic bronchial asthma.

1. Mites Causing Dermatitis

Dermatitis and scabies (or mange) are two different aspects of pruriginous irritation of the skin caused by the presence of mites on the human skin. In dermatitis the irritation is caused either by the repeated contact of living or dead mites with the skin (contact dermatitis) or by the toxic action of the saliva injected in the skin by biting mites. In both cases, the mites which produce dermatitis are accidental invaders and are unable to develop or to colonize permanently the human skin. In sensitive individuals, the dermatitis is more severe. Contact dermatitis is generally produced by Astigmatic mites (e.g., Acaridae and Glycyphagidae) whose short mouth parts are not able to pierce the skin. In the cases of dermatitis

produced by the injection of toxic saliva (e.g., chiggers, *Pyemotes* spp., Mesostigmata), the skin reaction develops more rapidly and is probably mainly toxic in nature.

Scabies (or mange) differs from dermatitis primarily by the fact that the mite (*Sarcoptes scabiei*) is a strict parasite that colonizes the corneous layers of the skin and is unable to live under other conditions. In man only one species is able to produce scabies, while a number of species may cause dermatitis. Baker et al. have reviewed the various species of mites involved in the production of dermatitis.[19] The mites causing dermatitis in man belong to three different orders of mites.

a. Mesostigmata

Genera *Dermanyssus* (Dermanyssidae) and *Ornithonyssus* (Macronyssidae)—

1. *Dermanyssus gallinae*, the cosmopolitan mite is common in fowls, especially chicken. In daytime the mites are hidden in crevices and debris of poultry houses. At night they become active and suck the blood of the birds. The mites also attack man, causing painful dermatitis. A spray of malathion 2% in the chicken houses may control the mite and is rather well supported by the chickens.
2. *Ornithonyssus sylviarum*, the northern fowl mite, is a common bird parasite in temperate climates. It may cause itching in humans.
3. *O. bursa*, the tropical fowl mite, replaces the former on tropical and subtropical birds and may cause dermatitis in humans.
4. *O. bacoti*, the tropical rat mite, was originally described from Egypt on *Rattus norvegicus*. Later it was found in several European countries where it sometime causes severe dermatitis in man. Its distribution is cosmopolitan.

b. Prostigmata

Family Trombiculidae — Larval trombiculids are ectoparasites of vertebrates, whereas the nymphs and adults are free-living. These larvae are known as chiggers, red bugs, or harvest mites. They are widespread and more than 3000 species have been described in the world. Unengorged larvae are 150- to 300-μm long and many species have a yellow-orange to reddish color. A certain number of species are able to cause dermatitis in man. The larvae attach to the skin and feed upon host tissue that has been partly digested by their salivary fluids. A feeding tube or stylostome develops around the mouth parts fixed in the skin. This tube is formed by a combined reaction of the host and the secretions of the mite. Associated with it is a small congestive elevation of the skin surrounded by a wheal. The lesion is accompanied by an intolerable painful itching. Chiggers causing dermatitis are known from all the continents. In Europe the most important species is *Trombicula (Neotrombicula) autumnalis*. Its larvae are common during the harvest season and are known in France as "lepte automnal" or "aoutat". The dermatitis is called "erythème automnal". This dermatitis has been recorded mainly from western Europe. In America three species are important agents of dermatitis. *T. (Eutrombicula) alfreddugesi* is the most common. It is known from North America (U.S. and Canada), Central America, and South America. *T.(E.) splendens* is widespread in the eastern U.S. The third species, *T.(E.) batatas*, is tropical and found from southern U.S. to Brazil. In Southeast Asia and Australasia *T.(E.) wichmanni* and in Australia *T.(E.) hirsti* are commonly found as producing dermatitis in man.

Treatment of the dermatitis — The mites attached to the skin may be removed individually with a needle. Antipruritic preparations such as phenolated solutions (0.5%), mentholated powder (1%), and calamine lotion may reduce the itching.

Prevention and control — Repellents, such as dimethylphthalate and diethyltoluamide (deet), can prevent the attack of chiggers. Impregnation of clothing with these products or with benzyl benzoate is recommended.[3] Restricted areas infested by chiggers may be treated

by insecticides; some compounds of the carbamate group (propoxur) have been utilized successfully against *T.(E.) alfreddugesi*.

Family Pyemotidae (straw, hay, or grain itch mites) — Some species of the genus *Pyemotes* feed on larvae of various insects (Coleoptera, Hymenoptera, and Lepidoptera), infesting grain, seed straw, or hay. They may cause severe dermatitis in man. The young female is elongate and pyriform. After fertilization, the eggs (about 200) are retained in the body and there transform into larvae and subsequently into adults. The abdomen of the female inflates monstrously and finally bursts and liberates the adults. The young females attach to the skin of man and cause a papular dermatitis accompanied by pruritis, headache, fever, and perspiration. The site of the bite is marked by a vesicle surrounded by a wheal. Infestation occurs in persons that come in contact with infested material, generally by sleeping on straw mattresses or while harvesting grain. *Pyemotes ventricosus* has been frequently recorded in cases of dermatitis from Europe (Italy, France, Bulgaria, and Yugoslavia), North Africa, India, Australia, and the U.S. Some authors believe that most of the cases attributed to that species were in fact caused by another more common species *P. tritici*, morphologically very close to *P. ventricosus*.[20]

Treatment of the dermatitis — See the chapter on chiggers (Prostigmata) (Chapter 7, Volume I). The infested material should be treated by insecticides in order to destroy the insect hosts of the mites.

Family Cheyletiellidae — Only the genus *Cheyletiella* contains species harmful to man. *Cheyletiella parasitivorax*, the rabbit fur mite, is a well-known parasite of the rabbit which was believed to cause dermatitis in dogs, cats, and man as well. It has been shown that in reality the mites infesting dogs and cats belonged to two different new species, *Ch. yasguri* and *Ch. blakei*, respectively.[21] Dermatitis in man is generally temporary and results from their bites or contact with the mites infesting animals, generally pets. Mites are rarely found on the skin of patients. Several cases of dermatitis in man caused by *Ch. yasguri* have been described during these last years.[22] The treatment of the infested pet by an acaricide will cure the dermatitis. For the treatment of the pruritus see Section IV.B.1.b on Trombiculidae.

Family Demodicidae — Two species of the genus *Demodex* are known in man: *Demodex folliculorum*, an elongate species that lives in the hair follicle, and *D. brevis*, with a much shorter body, that inhabits the sebaceous glands. The follicular species parasitize chiefly the region of the face and are very frequent in man. In some populations, the incidence may approach 100%. The pathogenic role of these mites is still controversial. It seems that both species are low-grade pathogens.[23]

c. Astigmata

The Astigmata causing dermatitis in man belong to the families Acaridae, Glycyphagidae, and Carpoglyphidae. Many of these species infest stored food (grains, dried fruit, cheese, flour, and seed) and are of great economic importance. Humans come in contact with the mites by handling these infested products. The skin reaction is a contact dermatitis and consists of a pruriginous erythema appearing at the sites which had been in contact with the mites. Small papules or vesicles may be present. The eruption is either acute or chronic. The exact cause of the pruritus and eruption is still unknown, but it seems that allergy plays an important role and that the repetition of the contacts increases the symptoms.

The skin reactions produced by these mites are generally considered as occupational acarine dermatitis. The most important are Grocer's itch caused by *Glycyphagus domesticus* and by *Tyrophagus putrescentiae*; Baker's itch and cheese mite dermatitis caused by *Acarus siro*; Copra itch caused by *Tyrophagus putrescentiae*; dried-fruit mite dermatitis caused by *Carpoglyphus lactis*; and wheat pollard itch caused by *Suidasia nesbitti*. Dermatitis is also reported by workers handling vanilla (vanillism) or tea.[19,24] Dermatitis is acquired often in the home, and can be the source or initiating agent of acarophobia (see Chapter 5, Volume

II). For the treatment of the itching, see Section IV.B.1.b. on dermatitis by Trombiculidae larvae.

2. Sarcoptic Mange or Scabies Caused by Sarcoptes scabiei

a. Epidemiology

Sarcoptes scabiei causes mange in man and in numerous mammals. More than 30 species and varieties have been described in the genus *Sarcoptes*, but all are based on variable characters and have thus no taxonomic value.[25] Scabies has increased noticeably in the world since 1963. This resurgence can be explained in part by the relaxation of standards of personal hygiene in certain groups of young people. Other causes are increased travel, importation of itinerant workers, adoption of foreign-born children, etc.[26] Infestation results generally by intimate contact with an infected person, usually by sleeping in the same bed (see Chapter 8, Volume I).

b. Morphology and Biology of the Mite

The adult female is ovoid and 350 to 450 μm long. The legs are very short, especially the posterior legs which terminate in long hairs. The dorsum of the mite is covered by cuticular scales which allow the mite to remain in the burrow. The life cycle from egg to ovigerous female takes from 10 to 14 days, and the life span of the female is approximately 2 months (see Figures 1 through 4; and Chapter 8, Volume I).

c. Pathology and Symptomatology

The fertilized female burrows a sinuous tunnel in the horny layer of the skin and deposits eggs and fecal pellets behind her. The length of this burrow varies from a few millimeters to 1 or 2 cm. The female is visible, at the blind end of the tunnel, as a minute whitish spot and may be removed with a needle. The majority of the mites are found on the hands and wrists. The next most infested site is the elbow, then the feet and genital organs, the buttocks, the axillae, and the breasts. The presence of the females in the skin provokes an intense itching which increases by warmth and perspiration. The mechanism responsible for the pruritus is questionable, but it seems that allergy plays an important role.

Norwegian or crusted scabies — This is a rare form of scabies characterized by the scaly and crusted aspect of the lesions. Burrows may be completely absent, and pruritus is often mild or absent. This form of scabies requires one or several years for development. The mites are extremely numerous in the skin, and this disease is highly contagious. Crusted scabies is generally observed in persons presenting a lowering in immunity or in some nervous syndrome producing analgesia where pruritus is absent and thus mechanical removal of the mites by scratching is suppressed.

Treatment — The most efficient scabicide is HCH or lindane. The prescription for an adult is 300 mg of lindane incorporated into 30 g of cream or lotion. This amount should be applied to the whole body except the head.[27]

3. Mites Causing House-Dust Allergy

a. General Considerations

A comprehensive review of the pathogenic action of the house dust mites was provided by Wharton.[28] The role of house dust in bronchial asthma has been established since 1922, however, the nature of the allergen responsible for the sensitization could not be determined at that moment. In 1964, it was demonstrated that a mite of the genus *Dermatophagoides* was the cause of the allergy.[29] This mite was specifically identified as *Dermatophagoides pteronyssinus* in 1966.[30] Investigations in various parts of the world have shown that this mite is almost universally present in homes. They have also shown the importance of other species of mites of the family Pyroglyphidae in the production of bronchial asthma.

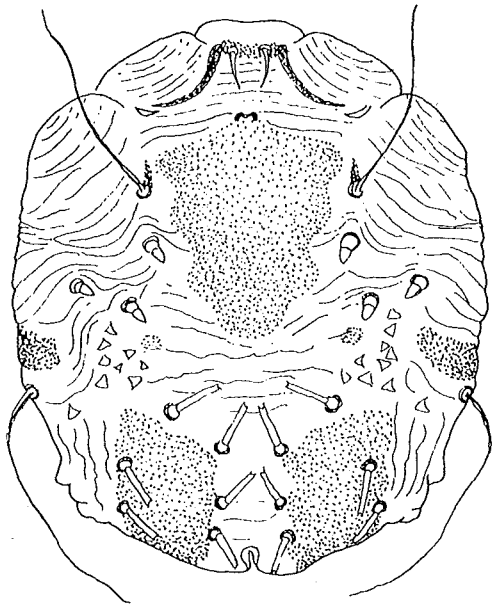

FIGURE 1. *Sarcoptes scabiei*, male; dorsum. (Redrawn from Fain, A., *Acta Zool. Pathol. Antverp.*, 47, 1, 1968. With permission.)

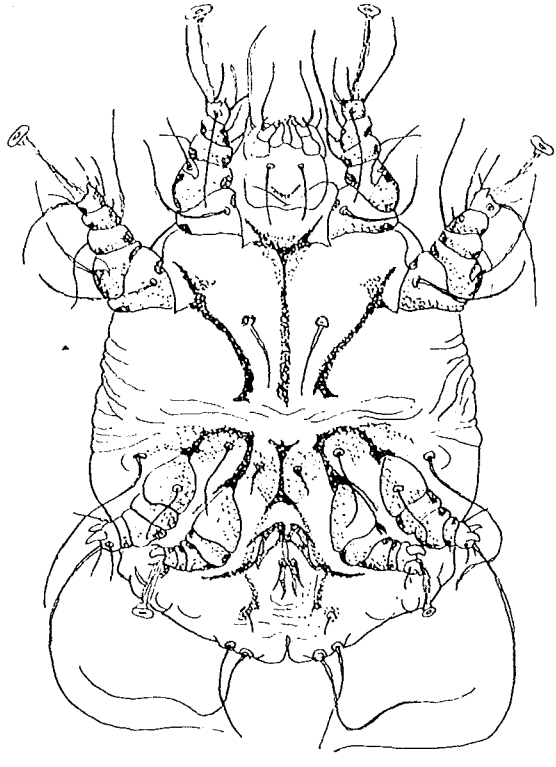

FIGURE 2. *Sarcoptes scabiei*, male; venter. (Redrawn from Fain, A., *Acta Zool. Pathol. Antverp.*, 47, 1, 1968. With permission.)

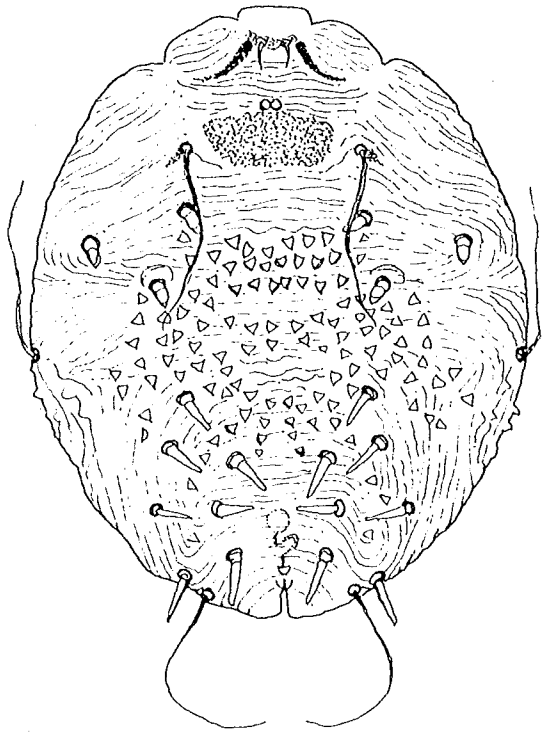

FIGURE 3. *Sarcoptes scabiei*, female; dorsum. (Redrawn from Fain, A., *Acta Zool. Pathol. Antverp.*, 47, 1, 1968. With permission.)

b. Mites Producing Respiratory Allergy

The genus *Dermatophagoides* belongs to the family Pyroglyphidae (Astigmata). This family presently contains 14 genera and 35 species. The most important species causing bronchial asthma are *D. pteronyssinus* and *D. farinae*.

Dermatophagoides pteronyssinus is the most frequent and the most widely distributed species. It is particularly abundant in regions of humid coastal climate, such as the coastal areas of western Europe and North America. On the other hand, *D. farinae* is more frequent in dryer areas, where the continental type of climate is prevalent such as central Europe and the central U.S.

Another species, also cosmopolitan and frequently found in houses, is *Euroglyphus maynei*. In Japan, *Hirstia domicola*, also a cosmopolitan species, is more frequent than *D. farinae*. In other countries, mostly tropical, several other local species are present.

The first studies have shown that pyroglyphid mites are more abundant on floors of the rooms occupied by humans, such as the living room and the bedroom. More recently, it has been demonstrated that the mattresses are the primary breeding places for *D. pteronyssinus*, *D. farinae*, and *E. maynei*. The total number of mites extract from a mattress may be as high as 5000/g of dust.[31]

In addition to the Pyroglyphidae, mites belonging to other families (Acaridae, Glycyphagidae, Cheyletidae, etc.) may be found in house dust. These mites are usually much less numerous in house dust than the Pyroglyphidae. They have, therefore, only a limited role in respiratory allergy.

c. House Dust Allergy

The most important syndrome associated with house dust is bronchial asthma. By using

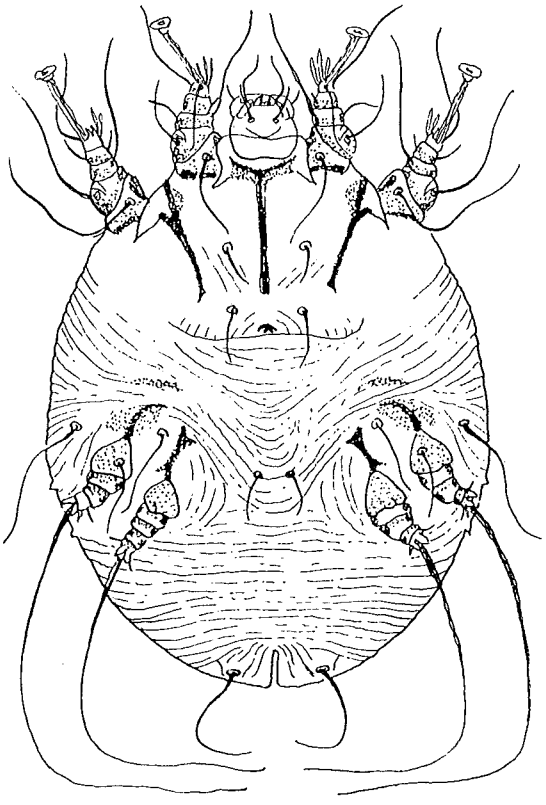

FIGURE 4. *Sarcoptes scabiei*, female; venter. (Redrawn from Fain, A., *Acta Zool. Pathol. Antverp.*, 47, 1, 1968. With permission.)

skin tests and bronchoprovocation, it has been shown that persons who react positively to house dust extract also react to mite extract, even at a high dilution.[29] The reaction is of the reagenic type (Ig E globulins). Recent investigations have shown that house dust may contain not only specific mite antigens, but also immunologically nonspecific molecules (premelanoidins) that could act as potential allergens in bronchial asthma.

d. Control of House Dust Mite Allergy

Desensitization of patients is possible by commercially available mite extracts. The mattresses should be covered with plastic sheets. Frequent cleaning of blankets and covers and vacuum cleaning of bedrooms is recommended.

4. Mites Vectors of Pathogens

Mites may transmit rickettsial diseases to man.

a. Mesostigmata

Allodermanyssus sanguineus, the house mouse mite, is a parasite of the house mouse and the house rat in the U.S. This mite is the vector and also a reservoir of *Rickettsia akari* causing rickettsial pox, a mild exanthematic disease of man. The disease has also been observed in the U.S.S.R. Man becomes infected by the bite of infected mites. The symptoms consist of fever, with a vesicular rash appearing 3 to 4 days after the onset of the fever. An eschar develops at the site of the infected bite and is accompanied by regional adenopathy.

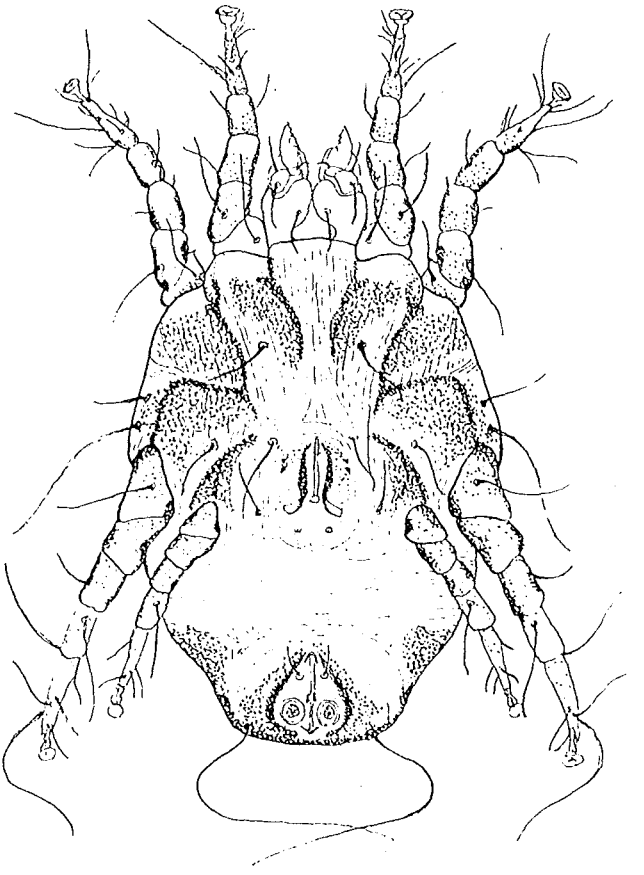

FIGURE 5. *Dermatophagoides pteronyssinus*, male; ventrally. (Redrawn from Fain, A., *Acarologia*, 8, 302, 1966. With permission.)

b. Prostigmata

Larvae of Trombiculidae — Several species of larval Trombiculidae are vectors of *Rickettsia tsutsugamushi* (= *R. orientalis*), the agent of the scrub typhus (Japanese river fever, Tsu Tsugamushi disease, etc.). Only the larvae of Trombiculidae are parasitic, while the nymphs and adults are free-living (see Section IV.B on Mites Causing Dermatitis). A comprehensive study on the epidemiology of this disease has been provided by Traub and Wisseman.[32]

This disease is endemic in many regions of southeastern Asia, in the West Pacific, and along the coast of north Queensland. The vectors are larval trombiculids of the genus *Leptotrombidium*. The most important species are *Leptotrombidium akamushi* in Japan and *L. deliensis* and *L. fletcheri* in southeastern Asia, but several other species can transmit the disease in more restricted areas. The main reservoirs are the mites in which the transmission is transovarial; rodents are transitory reservoirs. The disease is characterized mainly by fever, a maculo-papular rash, and severe general symptoms. At the site of the infective bite, an eschar generally develops. It begins by a papule which enlarges and becomes necrotic and black. Mortality in untreated cases depends on the regions. In Japan it varies from 15 to 60%; in some other regions, it is only 3%. Prognosis is favorable with the use of antibiotics, especially tetracycline. For the prevention and control of chiggers, see above.

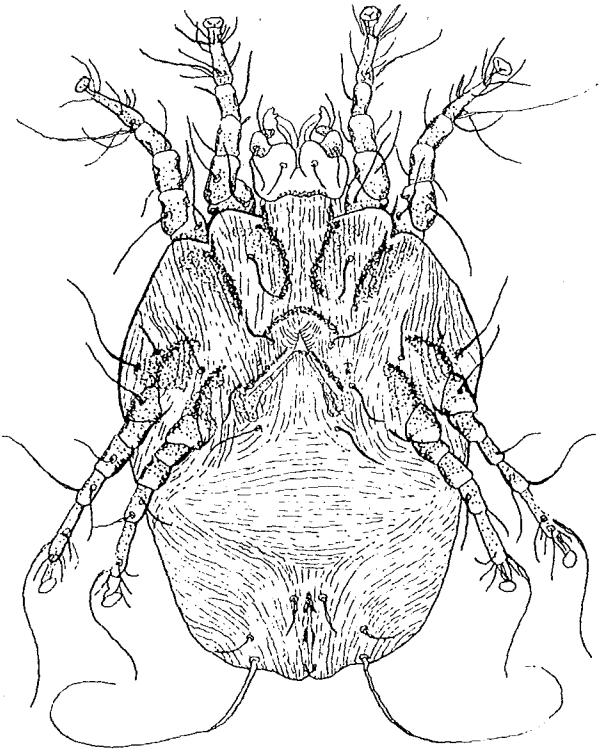

FIGURE 6. *Dermatophagoides pteronyssinus*, female; ventrally. (Redrawn from Fain, A., *Acarologia*, 8, 302, 1966. With permission.)

V. CONCLUDING REMARKS

Concluding this account, we note that new arachnid species inimical to man and new treatments even for the most damaging well-known species are discovered each year! An annual check of the *Journal of Medical Entomology*, *Acarologia*, *International Journal of Acarology*, and *Zoological Record*, Section 12 (Arachnida), for mites and *Index Medicus* for treatment should be used to update controls and treatments (see Appendixes 1 and 4).

Except for emergency situations (see Chapter 9, Volume I), it is very important, especially for spiders and scorpions, that species associated with lesions be captured, identified, and confirmed as soon as possible. With, for mites alone, approximately 3000 described species of chiggers and over 100 viruses found in these to date, diseases and arachnids will remain a challenge for scientific researches for the forseeable future.

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