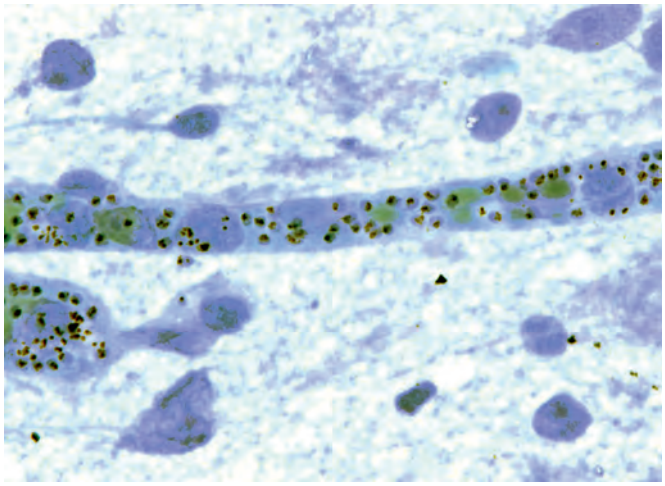

Figure 8-47 Field's stain of *Plasmodium falciparum*-infected red cells marginating within a capillary in cerebral malaria.

particularly in the spleen. Plasmodium falciparum infection leads to **splenomegaly**, due to both congestion and hyperplasia of the red pulp, and the spleen may eventually exceed 1000 gm in weight. In chronic infections, the spleen becomes increasingly fibrotic and brittle, with a thick capsule and fibrous trabeculae. The parenchyma is gray or black because the phagocytes contain granular, brown-black, faintly birefringent hemozoin pigment. Macrophages with engulfed parasitized red cells are also numerous.

With progression of malaria, the liver becomes enlarged and pigmented. Kupffer cells are heavily laden with malarial pigment, parasites, and cellular debris, while some pigment is also present in the parenchymal cells. Pigmented phagocytic cells may be found dispersed throughout the bone marrow, lymph nodes, subcutaneous tissues, and lungs. The kidneys are often enlarged and congested with a dusting of pigment in the glomeruli and hemoglobin casts in the tubules.

In **cerebral malaria** caused by *P. falciparum*, brain vessels are plugged with parasitized red cells (Fig. 8-47). Around the vessels there are ring hemorrhages that are probably related to local hypoxia incident to the vascular stasis and small focal inflammatory reactions (called **malarial** or **Dürck granulomata**). With more severe hypoxia, there is degeneration of neurons, focal ischemic softening, and occasionally scant inflammatory infiltrates in the meninges.

Nonspecific focal hypoxic lesions in the heart may be induced by the progressive anemia and circulatory stasis in chronically infected people. In some, the myocardium shows focal interstitial infiltrates. Finally, in the nonimmune patient, pulmonary edema or shock with disseminated intravascular coagulation may cause death, sometimes in the absence of other characteristic lesions.

Babesiosis

Babesia microti and *Babesia divergens* are malaria-like protozoans transmitted by the same deer ticks that carry Lyme disease and granulocytic ehrlichiosis. The white-footed mouse is the reservoir for *B. microti*, and in some areas, nearly all mice have a persistent low-level parasitemia. *B. microti* survives well in refrigerated blood, and several cases of transfusion-acquired babesiosis have been reported. *Babesia* parasitize red cells and cause fever and hemolytic anemia. The symptoms are mild except in debilitated or splenectomized individuals, who develop severe, potentially fatal parasitemias.

MORPHOLOGY

In blood smears, *Babesia* organisms superficially resemble *P. falciparum* ring stages, but lack hemozoin pigment, exhibit greater pleomorphism, and form characteristic tetrads (Maltese cross), which are diagnostic, if found (Fig. 8-48). The level of *B. microti* parasitemia is a good indication of the severity of infection (about 1% in mild cases and up to 30% in splenectomized persons). In fatal cases the anatomic findings are related to shock and hypoxia, and include jaundice, hepatic necrosis, acute renal tubular necrosis, adult respiratory distress syndrome, erythrophagocytosis, and visceral hemorrhages.

Leishmaniasis

Leishmaniasis is a chronic inflammatory disease of the skin, mucous membranes, or viscera caused by obligate intracellular, kinetoplast-containing (kinetoplastid) protozoan parasites transmitted through the bite of infected sandflies. Leishmaniasis is endemic throughout the Middle East, South Asia, Africa, and Latin America. It may also be epidemic, as is tragically the case in Sudan, India, Bangladesh, and Brazil, where tens of thousands of people have died of visceral leishmaniasis. Leishmanial infection, like infections by other intracellular organisms (mycobacteria, *Histoplasma*, *Toxoplasma*, and trypanosomes), is exacerbated by conditions that interfere with T-cell function, such as AIDS. Culture or histologic examination is used to diagnose the infection.

Pathogenesis. **The life cycle of *Leishmania* involves two forms: the promastigote, which develops and lives extracellularly in the sandfly vector, and the amastigote, which multiplies intracellularly in host macrophages.** Mammals, including rodents, dogs, and foxes, are reservoirs of *Leishmania*. When sandflies bite infected humans or animals, macrophages harboring amastigotes are ingested. The amastigotes differentiate into promastigotes, multiply within the digestive tract of the sandfly and migrate to the salivary gland, where they are poised for transmission by the fly bite. When the infected fly bites a person, the slender, flagellated infectious promastigotes

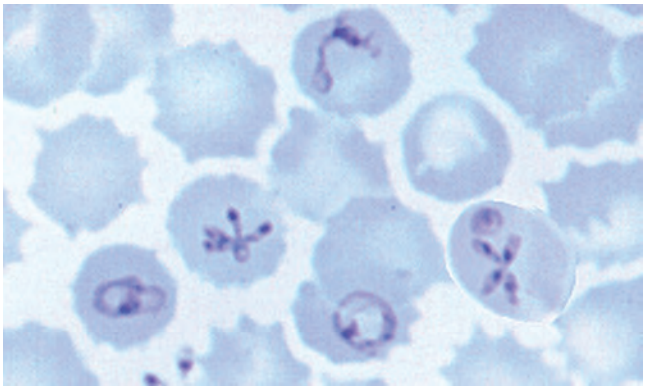

Figure 8-48 Erythrocytes with *Babesia*, including the distinctive Maltese cross form. (Courtesy Lynne Garcia, LSG and Associates, Santa Monica, Calif.)