Human anthrax is typically an ulcerative sore on an exposed part of the body. Constitutional symptoms are minimal, and the ulcer usually resolves without complications. If anthrax spores are inhaled, a fulminant pneumonia may lead to respiratory failure and death.

The isolation of *B. anthracis*, the proof of its relationship to anthrax infection, and the demonstration of immunity to the disease are among the most important events in the history of science and medicine. Robert Koch rose to fame in 1877 by growing the organism in artificial culture using pure culture techniques. He defined the stringent criteria needed to prove that the organism caused anthrax (Koch’s postulates), then met them experimentally. Louis Pasteur made a convincing field demonstration at Pouilly-le-Fort to show that vaccination of sheep, goats, and cows with an attenuated strain of *B. anthracis* prevented anthrax. He was cheered and carried on the shoulders of the grateful farmers of the district, an experience now, unhappily, largely restricted to winning football coaches.

**Epidemiology**

Anthrax is primarily a disease of herbivores such as horses, sheep, and cattle, who acquire it from spores of *B. anthracis* contaminating their pastures. Humans become infected through contact with these animals or their products in a way that allows the spores to be inoculated through the skin, ingested, or inhaled. In the 1920s, more than 100 cases occurred annually in the United States among farmers, veterinarians, and meat handlers, but the control of animal anthrax in developed countries has made human cases rare. A few endemic foci persist in North America and have been the source of naturally acquired disease. Another source is animal products such as wool, hides, or bone meal fertilizer that have been imported from a country where animal anthrax is endemic.

The real threat associated with anthrax comes from its continuing appeal to those bent on using it as an agent of biologic warfare or terrorism. The long life, stability, and low mass of the dried spores make the prospect of someone producing a “cloud of death” leading to massive pulmonary anthrax a chilling reality. A 1979 episode resulting in more than 60 anthrax deaths in the former Soviet Union is now attributed to an accidental explosion at a biologic warfare research facility that aerosolized more than 20 pounds of anthrax spores. United Nations inspection teams in Iraq uncovered facilities for the production of massive amounts of spores together with plans to create and spread infectious aerosols using missile warheads. The inhalation anthrax among postal workers after the September 11, 2001, terrorist attacks appears to have been due to the mailing of envelopes containing “weapons-grade” anthrax spores stolen from a biologic warfare research facility. Such spores had been treated to enhance their aerosolization and dissemination. The source of this outbreak is still unknown. The forms of anthrax are summarized in Figure 26–10.

**Pathogenesis**

When spores of *B. anthracis* reach the rich environment of human tissues, they germinate and multiply in the vegetative state. The antiphagocytic properties of the capsule aid in survival, eventually allowing production of large enough amounts of the exotoxins to cause

Pasteur produced animal vaccine with attenuated anthrax strain

Infection is through injection of spores derived from herbivores into the skin

Contaminated materials are imported from countries with animal anthrax

Use for biologic warfare is a continuing threat

Aerosols could spread pulmonary anthrax widely

Weapons-grade spores are specially treated

Antiphagocytic effect of d-glutamic acid capsule is required for virulence
disease. The tripartite nature of the anthrax exotoxin complex must play an important role, but the timing and relative importance of the components are not known. The EF adenylate cyclase activity is believed to correlate with the striking edema seen at infected sites.

IMMUNITY

The specific mechanisms of immunity against *B. anthracis* are not known. Experimental evidence favors antibody directed against the toxin complex, but the relative role of the components of the toxin is not clear. The capsular glutamic acid is immunogenic, but antibody against it is not protective.

ANTHRAX: CLINICAL ASPECTS

MANIFESTATIONS

Cutaneous anthrax usually begins 2 to 5 days after inoculation of spores into an exposed part of the body, typically the forearm or hand. The initial lesion is an erythematous papule, which may be mistaken for an insect bite. This papule usually progresses through vesicular and ulcerative stages in 7 to 10 days to form a black eschar (scab) surrounded by edema (Figure 26–9B and C). This lesion is known as the “malignant pustule,” although it is neither malignant nor a pustule. Associated systemic symptoms are usually mild, and the lesion typically heals very slowly after the eschar separates. Less commonly, the disease progresses with massive local edema, toxemia, and bacteremia.

Pulmonary anthrax is contracted by inhalation of spores. Historically, this has occurred when contaminated hides, hair, wool, and the like are handled in a confined space.