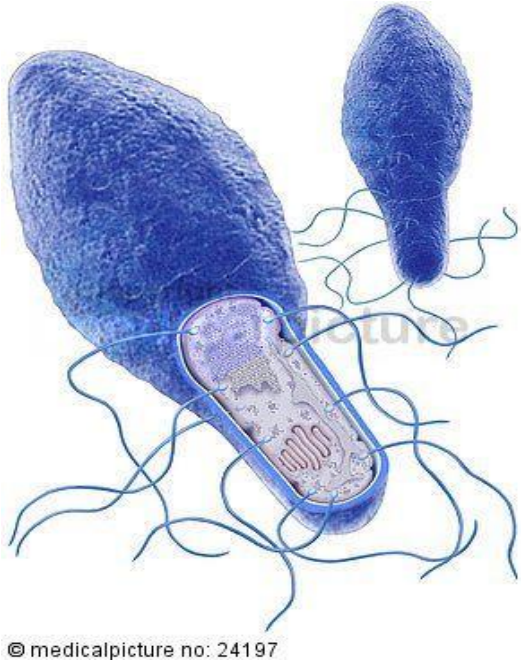
Morphology and Identification

Clostridium botulinum

C botulinum, which causes **botulism,** is worldwide in distribution; it is found in soil and occasionally in animal feces. Types of *C botulinum* are distinguished by the antigenic type of toxin they produce. Spores of the organism are highly resistant to heat, withstanding 100°C for several hours. Heat resistance is diminished at acid pH or high salt concentration.



Clinical Findings

Symptoms begin 18–24 hours after ingestion of the toxic food, with visual disturbances (incoordination of eye muscles, double vision), inability to swallow, and speech difficulty; signs of bulbar paralysis are progressive, and death occurs from respiratory paralysis or cardiac arrest. Gastrointestinal symptoms are not regularly prominent. There is no fever. The patient remains fully conscious until shortly before death. The mortality rate is high. Patients who recover do not develop antitoxin in the blood.

Diagnostic Laboratory Tests

Toxin can often be demonstrated in serum, gastric secretions, or stool from the patient, and toxin may be found in residual food. *C botulinum* may be grown from food remains and tested for toxin production, but this is rarely done and is of questionable significance. In infant botulism, *C botulinum* and toxin can be demonstrated in bowel contents but not in serum. Other methods used to detect toxin include ELISAs and PCR, but the latter may detect organisms that carry the gene but do not express toxin.

Treatment

Potent antitoxins to three types of botulinum toxins have been prepared in horses. Because the type responsible for an individual case is usually not known, trivalent (A, B, E) antitoxin must be promptly administered intravenously with customary precautions. Adequate respiration must be maintained by mechanical ventilation if necessary. These measures have reduced the mortality rate from 65% to below 25%. Although most infants with botulism recover with supportive care alone, antitoxin therapy is recommended.

Clostridium tetani

Clinical Findings

The incubation period may range from 4 to 5 days to as many weeks. The disease is characterized by tonic contraction of voluntary muscles. Muscular spasms often involve first the area of injury and infection and then the muscles of the jaw (trismus, lockjaw), which contract so that the mouth cannot be opened. Gradually, other voluntary muscles become involved, resulting in tonic spasms. Any external stimulus may precipitate a tetanic generalized muscle spasm. The patient is fully conscious, and pain may be intense. Death usually results from interference with the mechanics of respiration. The mortality rate in generalized tetanus is very high.

Diagnosis

The diagnosis rests on the clinical picture and a history of injury, although only 50% of patients with tetanus have an injury for which they seek medical attention. The primary differential diagnosis of tetanus is strychnine poisoning. Anaerobic culture of tissues from contaminated wounds may yield *C tetani*, but neither preventive nor therapeutic use of antitoxin should ever be withheld pending such demonstration. Proof of isolation of *C tetani* must rest on production of toxin and its neutralization by specific antitoxin.

Prevention and Treatment

The results of treatment of tetanus are not satisfactory. Therefore, prevention is all important. Prevention of tetanus depends on (1) active immunization with toxoids, (2) proper care of wounds contaminated with soil (3) prophylactic use of antitoxin, and (4) administration of penicillin. The intramuscular administration of 250–500 units of human antitoxin (tetanus immune globulin) gives adequate systemic protection (0.01 unit or more per milliliter of serum) for 2–4 weeks. It neutralizes the toxin that has not been fixed to nervous tissue. Active immunization with tetanus toxoid should accompany antitoxin prophylaxis. Patients who develop symptoms of tetanus

should receive muscle relaxants, sedation, and assisted ventilation. Sometimes they are given very large doses of antitoxin (3000–10,000 units of tetanus immune globulin) intravenously in an effort to neutralize toxin that has not yet been bound to nervous tissue. However, the efficacy of antitoxin for treatment is doubtful except in neonatal tetanus, in which it may be lifesaving. Surgical debridement is vitally important because it removes the necrotic tissue that is essential for proliferation of the organisms. Hyperbaric oxygen has no proven effect. Penicillin strongly inhibits the growth of *C tetani* and stops further toxin production. Antibiotics may also control associated pyogenic infection. When a previously immunized individual sustains a potentially dangerous wound, an additional dose of toxoid should be injected to restimulate antitoxin production. This "recall" injection of toxoid may be accompanied by a dose of antitoxin if the patient has not had current immunization or boosters or if the history of immunization is unknown.