Poliomyelitis is a highly contagious disease caused by three serotypes of poliovirus.

Epidemiology

Poliomyelitis became an epidemic disease in the United States at the turn of the century. Epidemics of ever-increasing magnitude occurred, with over 20,000 cases of paralytic poliomyelitis reported in 1952. Following the introduction of effective vaccines, first inactivated poliovirus vaccine (IPV) in 1955, and oral poliovirus vaccine (OPV) starting in 1961, the reported incidence of poliomyelitis in the United States declined dramatically to <100 cases in 1965 and to <10 cases in 1973. With the introduction and widespread use of OPV (containing live-attenuated poliovirus strains), vaccine-associated paralytic poliomyelitis (VAPP) was first recognized. By 1973, for the first time, more cases of vaccine-associated disease were reported than paralytic disease caused by wild poliovirus.

The last cases of indigenously transmitted wild poliovirus disease were reported in 1979. Since then, apart from six cases of imported poliomyelitis, only one of which has occurred since 1986, all reported cases of paralytic poliomyelitis in the United States have been vaccine-associated. VAPP is a very rare disease with an average of eight reported cases annually during 1980–1994, or one case reported for every 2.4 million doses of OPV distributed. The risk of VAPP is highest following the first dose of OPV and among immunodeficient persons.

Following the successful implementation of the polio eradication initiative in the Americas since 1985, the last case of wild poliovirus-associated disease was detected in Peru in 1991. The hemisphere was certified as free of indigenous wild poliovirus in 1994. Nevertheless, the potential for importation of wild poliovirus into the United States remains until worldwide poliomyelitis eradication is achieved. Because inapparent infection with wild virus strains no longer contributes to establishing or maintaining poliovirus immunity in the United States, universal vaccination of infants and children is the only means of establishing and maintaining population immunity against poliovirus to prevent poliomyelitis cases and epidemics caused by importation of wild virus into the United States.

Population-based surveys have confirmed that the prevalence of poliovirus antibody among school age children, adolescents and young adults in the United States is high (>90% to poliovirus types 1 and 2, and >85% to type 3). In addition, seroprevalence surveys in two inner-city areas of the United States--areas in which routine coverage is low--during 1990-1991 found that >80% of all children 12-47 months of age had antibodies to all three poliovirus serotypes. However, members of certain religious groups objecting to vaccination have remained susceptible to poliomyelitis. These groups appear to be highest risk for epidemic poliomyelitis. The last two outbreaks of poliomyelitis in the United States were reported among religious groups in 1972 (Christian Scientists) and in 1979 (Amish).

The incubation period is 7 to 14 days for paralytic cases, with a range from 3 to 35 days.
Clinical Description

Infection with poliovirus results in a spectrum of clinical manifestations from inapparent infection to non-specific febrile illness, aseptic meningitis, paralytic disease, and death. Two phases of acute poliomyelitis can be distinguished: a non-specific febrile illness (minor illness) followed, in a small proportion of patients, by aseptic meningitis and/or paralytic disease (major illness). The ratio of cases of inapparent infection to paralytic disease is usually in the range from 100:1 to 1000:1.

Following poliovirus exposure, viral replication occurs in the oropharynx and the intestinal tract. Viremia follows, which may result in infection of central nervous system cells. A specific receptor is needed for the virus to enter cells. Replication of poliovirus in motor neurons of the anterior horn and brain stem results in cell destruction and causes the typical clinical manifestations of poliomyelitis. Depending on the site of paralysis, poliomyelitis can be classified as spinal, bulbar, or spino-bulbar disease. Progression to maximum paralysis is rapid (2-4 days), usually associated with fever and muscle pain, and rarely continues after the temperature has returned to normal. Spinal paralysis is typically asymmetric, more severe proximally than distally, and deep tendon reflexes are absent or diminished. Bulbar paralysis may compromise respiration and swallowing. Between 2%-10% of cases of paralytic poliomyelitis are fatal. Infection with poliovirus results in lifelong, type-specific immunity.

Following the acute episode, many patients recover muscle functions at least partially, and prognosis for recovery can usually be established within 6 months after onset of paralytic manifestations.

Laboratory Tests

Laboratory studies, especially attempted poliovirus isolation, are critical to rule out or confirm the diagnosis of paralytic poliomyelitis.

Viral isolation
To increase the probability of poliovirus isolation, at least two stool specimens should be obtained 24 hours apart from patients with suspected poliomyelitis as early in the course of disease as possible (ideally within 15 days after onset).

The likelihood of poliovirus isolation is highest from stool specimens, intermediate from pharyngeal swabs, and very low from blood or spinal fluid. The isolation of poliovirus from stool specimens contributes to the diagnostic evaluation but does not constitute proof of a causal association of such viruses with paralytic poliomyelitis. Isolation of virus from the cerebrospinal fluid (CSF) is diagnostic but is rarely accomplished. To increase the probability of poliovirus isolation, at least two stool specimens and two throat swabs should be obtained 24 hours apart from patients with suspected poliomyelitis as early in the course of the disease as possible (i.e., immediately after poliomyelitis is considered as a possible differential diagnosis), but ideally within the first 15 days after onset of paralytic disease. Specimens should be sent to the state or other reference laboratories for primary isolation. Laboratories should forward isolates to CDC for intratypic differentiation to determine whether the poliovirus isolate is wild or vaccine-related.

Isolation of wild poliovirus constitutes a public health emergency and appropriate control efforts must be initiated immediately (in consultation between health care providers, the state and local health departments, and CDC).
Serologic testing

Serology may be helpful in supporting or ruling out the diagnosis of paralytic poliomyelitis. An acute serum specimen should be obtained as early in the course of disease as possible, and a convalescent specimen should be obtained 3 weeks later. A four-fold rise between the acute and convalescent specimens suggests poliovirus infection. Non-detectable antibody titers in both specimens may help rule out poliomyelitis, but may be falsely negative in immunocompromised persons, who are also at highest risk for paralytic poliomyelitis. In addition, neutralizing antibodies appear early and may be at high levels by the time the patient is hospitalized; thus, a four-fold rise may not be demonstrated. One of the limitations of serology is the inability to distinguish between antibody induced by vaccine-related poliovirus and antibody induced by wild virus. Serologic assays to detect anti-poliovirus antibodies are available in most commercial and state public health laboratories.

CSF analysis

The CSF usually contains an increased number of leukocytes — from 10 to 200 cells/mm$^3$ (primarily lymphocytes) and a mildly elevated protein, from 40 to 50 mg/100 ml. These findings are non-specific and may result from a variety of infectious and noninfectious conditions.

Surveillance

Poliomyelitis is a condition reportable within 24 hours of diagnosis.

The poliomyelitis surveillance system serves to
1) detect importation of wild poliovirus into the United States,
2) characterize the epidemiology of VAPP, and
3) monitor changes in VAPP.

Enhanced surveillance for paralytic poliomyelitis is needed to evaluate the impact of the revised poliovirus vaccination schedule on the incidence of VAPP.

Case Definition

Clinical case definition
Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss.

Case classification
Probable: a case that meets the clinical case definition
Confirmed: a case that meets the clinical case definition and in which the patient has a neurologic deficit 60 days after onset of initial symptoms, has died, or has unknown follow-up status
Comment: All suspected cases of paralytic poliomyelitis are reviewed by a panel of expert consultants before final classification occurs. Confirmed cases are then further classified based on epidemiologic and laboratory criteria.

Confirmed cases are further classified based on epidemiologic and laboratory criteria.
Indigenous case. Any case which cannot be proved to be imported.

Imported case. A case which has its source outside the United States. A person with poliomyelitis (United States resident or other) who has entered the United States and had onset of illness within 30 days before or after entry.

Investigation

Rapid investigation of suspected poliomyelitis cases is critical to identifying possible wild poliovirus transmission. Rapid detection of wild virus-associated cases will permit the timely implementation of control efforts (mass vaccination with OPV) to limit the spread of imported wild poliovirus, and maintain the elimination of wild poliovirus from the United States. Moreover, rapid investigation of suspected cases will allow collection of specimens for poliovirus isolation, which are critical for ruling out or confirming paralytic poliomyelitis, whether wild virus-associated or vaccine-related.

Information to collect

Demographic, clinical, and epidemiologic information are collected to 1) determine whether the suspected case meets the case definition for paralytic poliomyelitis, and 2) determine whether the disease may be caused by wild poliovirus or is vaccine-related. Additional information may also be collected at the direction of the state health department.

- **Demographic information**
- **Vaccination status including**
  - Number of doses of poliomyelitis vaccine
  - Time since last dose of poliomyelitis vaccine
  - Type of vaccine (IPV or OPV)
- **Clinical details including**
  - Immunologic status of case-patient
  - Date of onset of symptoms
  - Complications and hospitalization
- **Laboratory information including**
  - Serologic test results
- **Travel and exposure history including**
  - Recent travel to polio endemic areas
  - Contact with persons recently returning from polio endemic areas
  - Contact with recent OPV recipient
  - Setting (i.e., is case-patient a member of a group objecting to vaccination)
- **Dates including**
  - Date reported to health department
  - Date of case investigation

Travel History
Because the last cases of paralytic poliomyelitis due to indigenously acquired wild poliovirus infection in the United States were reported in 1979, it is likely that wild poliovirus in a suspected case-patient is imported by, either the suspected patient directly or by a contact of the case-patient. Results of viral isolation and differentiation may not be available at the time of case investigation. Therefore, to rule out the possibility of imported wild poliovirus, a detailed travel history of suspected cases and of other household and non-household contacts should be obtained. Any history of contacts with visitors, especially those from polio-endemic areas, might be particularly revealing.

**Setting**

Because the last two outbreaks of poliomyelitis in the United States were reported among Christian Scientists in 1972 and the Amish in 1979, a suspected case of poliomyelitis reported from a group objecting to vaccination should be assigned the highest priority for follow-up and collection of specimens. In addition, isolation of wild poliovirus from residents of Canada in 1993 and 1996 demonstrates the potential for wild poliovirus importation into this continent. The strain isolated in 1993 was linked epidemiologically and by genomic sequencing to the 1992 poliomyelitis outbreak in the Netherlands, and the 1996 isolate was from a child who had recently visited India.

**Control**

No cases of paralytic polio due to indigenously acquired wild poliovirus have been reported in the United States since 1979. The goal of maintaining elimination of paralytic poliomyelitis due to indigenous acquisition of wild poliovirus has been established for each year until the goal of global eradication is met.

**Promoting awareness.** Because of the severity of poliomyelitis disease (i.e., paralytic disease), clinicians are often the first to suspect the diagnosis of poliomyelitis and they are the key to timely reporting of suspected cases. However, disease reporting by clinicians is often delayed because it is only after other differential diagnoses are ruled out that the diagnosis of poliomyelitis is considered. Efforts should be made to promote physicians’ awareness of the importance of prompt reporting of suspected cases to the state and local health department and the CDC, and the need to obtain stool and serum specimens early in the disease course.

**Ensuring laboratory capabilities.** Make sure that the state laboratory or other easily accessible laboratory facility is capable of performing, at a minimum, primary viral isolation and serologic testing for poliovirus.

**Obtaining laboratory confirmation.** Appropriate stool specimens (two specimens taken at least 24 hours apart during the first 15 days after onset of paralytic disease) should be collected.

**Active surveillance.** The diagnosis of a case of poliomyelitis, particularly in a member of a group that refuses vaccination (such as the Amish or Christian Scientists), should prompt immediate control measures (mass vaccination with OPV) as well as active surveillance activities. These activities should include active case finding at area hospitals or any other sources of acute medical care.

**Immunization**

Louisiana Office of Public Health – Infectious Disease Epidemiology Section- Infectious Disease Control Manual
In January 1997 the Advisory Committee on Immunization Practices (ACIP) recommended a sequential schedule of two doses of inactivated poliovirus vaccine (IPV) followed by two doses of oral poliovirus vaccine (OPV) for routine vaccination of children in the United States. Schedules of IPV only and OPV only are also acceptable options for poliovirus vaccination. The American Academy of Pediatrics (AAP) and American Academy of Family Practice (AAFP) have also changed their recommendations, and now recommend expanded use of IPV for routine poliovirus vaccination. These recommendations constitute the most substantive changes in poliovirus vaccination policy in the United States since the introduction of OPV in 1961. The recommendations for routine poliovirus vaccination have been revised for three principal reasons:
1) paralytic poliomyelitis attributable to indigenously acquired wild poliovirus has not occurred in the United States since 1979;
2) progress toward global polio eradication has reduced the risk for importation of wild poliovirus into the United States;
3) cases of vaccine-associated paralytic poliomyelitis (VAPP) continues to occur.

The risk for vaccine-associated paralytic poliomyelitis caused by OPV is now judged less acceptable because of the diminished risk of disease due to wild poliovirus (indigenous or imported). The new sequential schedule is expected to reduce the incidence of VAPP while maintaining the high levels of individual and population immunity against polioviruses necessary to prevent polio outbreaks if polio is reintroduced into the United States.

For unvaccinated adults and for persons in whom OPV is contraindicated, IPV is recommended. The primary series of IPV consists of three doses of vaccine. Two doses can be given at a 4–8 week interval; the third dose should follow 6–12 months after the second, (i.e., at 12–18 months of age for routine vaccination of children). In circumstances where accelerated protection is needed, the minimum interval between the doses of IPV is 4 weeks.

All children should complete primary immunization before entering school. The primary sequential IPV/OPV series consists of four doses, administered according to the schedule outlined above. All children who previously received a primary immunization series with OPV (three doses) or IPV (three doses) in early childhood should be given a fourth dose of OPV or IPV before entering school (e.g., between 4–6 years of age) to complete the recommended schedule. Persons considered to be at increased risk of exposure to poliovirus (e.g., travelers to polio-endemic areas, laboratory workers) and who have received primary immunization as a child should be given a single additional dose of OPV or IPV.

**Hospital precaution and isolation:** Contact precautions