Poliomyelitis

The word polio (grey) and myelon (marrow, indicating the spinal cord) are derived from the Greek roots which describe the tissue most commonly affected in the spinal cord which leads to the classic manifestations of paralysis.

Although records from antiquity mention crippling diseases compatible with poliomyelitis, it was Michael Underwood from Britain who, in 1789, first described a debility of the lower extremities in children that was recognizable as poliomyelitis. The first outbreaks in Europe were reported in the early 19th century, and outbreaks were reported in the United States a few years later. For the next hundred years, epidemics of polio were reported from developed countries in the northern hemisphere each summer and fall. These epidemics became increasingly severe, and the average age of persons affected rose, which increased both the disease severity and number of deaths from polio. Polio reached a peak in the United States in 1952, with over 20,000 paralytic cases. Polio incidence fell rapidly following introduction of effective vaccines. The last case of wild-virus polio acquired in the United States was in 1979, and global polio eradication may be achieved within the next decade.

Poliovirus

Poliovirus is a member of the enterovirus subgroup, family Picornaviridae. Enteroviruses are transient inhabitants of the gastrointestinal tract, and are stable at acid pH. Picornaviruses are small, ether-insensitive viruses with an RNA genome.

There are three poliovirus serotypes (P1, P2, and P3). There is minimal heterotypic immunity between the three serotypes.

The poliovirus is rapidly inactivated by heat, formaldehyde, chlorine, and ultraviolet light.
**Pathogenesis**

The mouth is the portal of entry of the virus and primary multiplication of the virus occurs at the site of implantation in the pharynx and gastrointestinal tract. The virus is usually present in the throat and in the stools before the onset of illness. One week after onset there is little virus in the throat, but virus continues to be excreted in the stools for several weeks. The virus invades local lymphoid tissue, enters the blood stream, and then may infect cells of the central nervous system. Replication of poliovirus in motor neurons of the anterior horn and brain stem results in cell destruction and causes the typical manifestations of poliomyelitis.

**Clinical Features**

The incubation period for poliomyelitis is commonly 6 to 20 days with a range from 3 to 35 days.

The response to poliovirus infection is highly variable and has been categorized based on the severity of clinical presentation.

**Inapparent infection without symptoms**

Up to 95% of all polio infections are inapparent or subclinical. Estimates of the ratio of inapparent to paralytic illness vary from 50:1 to 1,000:1 (usually 200:1). Infected persons without symptoms shed virus in the stool, and are able to transmit the virus to others.

**Minor illness (abortive poliomyelitis)**

Approximately 5% (4%-8%) of polio infections consist of a nonspecific illness without clinical or laboratory evidence of central nervous system invasion and are characterized by complete recovery in less than a week. Three syndromes observed with this form of poliovirus infection are upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely, diarrhea), and influenza-like illness. These syndromes are indistinguishable from other viral illnesses.

**Nonparalytic poliomyelitis**

Nonparalytic aseptic meningitis (symptoms of stiffness of the neck, back, and/or legs) usually following several days after a prodrome similar to that of minor illness occur in 1%-2% of polio infections. Increased or abnormal sensations can also occur. Typically these symptoms will last from 2 to 10 days followed by complete recovery.
**Paralytic poliomyelitis**

Less than 2% of all polio infections result in a flaccid paralysis (usually less than 1%). Paralytic symptoms generally begin 1 to 10 days after prodromal symptoms and progress for 2 to 3 days. Generally, no further paralysis occurs after the temperature returns to normal. The prodrome may be biphasic, especially in children, with initial minor symptoms separated by a 1- to 7-day period from more major symptoms. Additional prodromal signs and symptoms can include a loss of superficial reflexes, initially increased deep tendon reflexes and severe muscle aches and spasms in the limbs or back. The illness progresses to flaccid paralysis with diminished deep tendon reflexes which reaches a plateau without change for days to weeks and is usually asymmetrical. Strength then begins to return. Patients do not experience sensory losses or changes in cognition.

Many persons with paralytic poliomyelitis recover completely and, in most, muscle function returns to some degree. Patients with weakness or paralysis 12 months after onset will usually be left with permanent residua.

Paralytic polio is classified into three types, depending on the level of involvement. **Spinal polio** is most common, and accounted for 79% of paralytic cases from 1969-1979. It is characterized by asymmetric paralysis that most often involves the legs. **Bulbar polio** accounts for 2% of cases and leads to weakness of muscles innervated by cranial nerves. **Bulbospinal polio** accounts for 19% of cases and is a combination of bulbar and spinal paralysis.

The death-to-case ratio for paralytic polio is generally 2%-5% in children and up to 15%-30% in adults (depending on age). It increases to 25%-75% with bulbar involvement.

**Laboratory Diagnosis**

**Viral isolation**

Poliovirus may be recovered from the stool or pharynx from a person with presumed poliomyelitis. Isolation of virus from the cerebrospinal fluid (CSF) is diagnostic but is rarely accomplished.

If poliovirus is isolated from a person with acute flaccid paralysis, it must be tested further, using oligonucleotide mapping (fingerprinting) or genomic sequencing, to determine if the virus is “wild-like” or “vaccine-like.”


**Serology**

Neutralizing antibodies appear early and may be at high levels by the time the patient is hospitalized and, therefore, a 4-fold rise may not be demonstrated.

**Cerebrospinal fluid (CSF)**

The CSF in poliovirus infection usually contains an increased number of white blood cells (10 to 200 cells/mm³, primarily lymphocytes) and a mildly elevated protein from 40 to 50 mg/100 ml.

**Epidemiology**

**Reservoir**

Humans are the only known reservoir of poliovirus, which is transmitted most frequently by persons with inapparent infections. There is no asymptomatic carrier state except in immune deficient persons.

**Transmission**

Person-to-person spread of poliovirus via the fecal-oral route is the most important route of transmission, although the oral-oral route may account for some cases.

**Temporal pattern**

Poliovirus infection typically peaks in the summer months in temperate climates. There is no seasonal pattern in tropical climates.

**Communicability**

Poliovirus is highly infectious, with seroconversion rates in susceptible household contacts of children nearly 100% and of adults over 90%. Cases are most infectious from 7 to 10 days before and after the onset of symptoms, but poliovirus may be present in the stool from 3 to 6 weeks.

**Predominant serotype**

Nearly all epidemics are due to type 1, whereas types 2 and 3 are more often isolated in vaccine-associated poliomyelitis.
Secular Trends in the United States

Before the 18th century, polioviruses probably circulated widely and initial infections to at least one type probably occurred in early infancy, when transplacentally acquired maternal antibodies were high. Exposure throughout life probably provided continual boosting of immunity and paralytic infections were probably rare. (This view has been recently challenged based on data of lameness studies in developing countries.)

In the immediate pre-vaccine era, improved sanitation allowed less frequent exposure and increased the age of primary infection. There was infrequent boosting of immunity from natural exposure, pooling of susceptibles, and ultimately the occurrence of epidemics, with 13,000 to 20,000 paralytic cases reported annually.

In the early vaccine era, the incidence dramatically decreased following IPV introduction in 1955. The decline continued following OPV introduction in 1961. In 1960, a total of 2,525 paralytic cases were reported, compared with 61 in 1965.

The last case of paralytic poliomyelitis caused by endemic transmission of wild virus in the United States was in 1979. This outbreak occurred among the Amish in several Midwest states. The virus was imported from the Netherlands.

From 1980 through 1996, a total of 142 confirmed cases of paralytic poliomyelitis were reported, an average of 8 cases per year. Six cases were acquired outside the United States and imported. The last imported case occurred in 1986. Two cases were classified as indeterminant (no poliovirus isolated from samples obtained from the patients, and these persons had no history of recent vaccination or direct contact with a vaccine recipient). The remaining 134 (94%) cases were associated with administration of oral poliovirus vaccine.

Outbreaks of poliomyelitis in the United States since 1970

In 1970, on the Texas-Mexico border, 22 cases occurred, all in children 4 years of age or less. In 1972, in a Christian Science school in Connecticut, eight cases of paralytic poliomyelitis and three of non-paralytic occurred in persons from 7 to 18 years of age. In 1979, among the Amish (two non-Amish) in Pennsylvania, Missouri, Iowa, and Wisconsin, ten paralytic and five non-paralytic cases of poliomyelitis occurred, with a mean age of 12 years.
Poliovirus Vaccine

Inactivated (Salk) poliovirus vaccine (IPV) was licensed in 1955 and was used extensively from that time until the early 1960s. In 1961, type 1 and 2 monovalent oral poliovirus vaccine (MOPV) was licensed, and in 1962, type 3 MOPV was licensed. In 1963, trivalent oral poliovirus vaccine (OPV) was licensed and largely replaced IPV use. OPV has been the vaccine of choice in the United States and most other countries of the world since 1963. An enhanced-potency IPV was licensed in November 1987, and first became available in 1988.

Inactivated poliovirus vaccine (IPV)

Two enhanced forms of inactivated poliovirus vaccine are currently licensed in the United States, but only one vaccine is actually distributed. This vaccine is produced in Vero cells and contains all three types of vaccine-related poliovirus.

IPV is highly effective in producing immunity to poliovirus, and protection from paralytic poliomyelitis. Ninety percent or more of vaccine recipients develop protective antibody to all three poliovirus types after 2 doses, and at least 99% are immune following 3 doses. Protection against paralytic disease correlates with the presence of antibody.

The most important advantage of IPV is that it is inactivated, so it cannot replicate, and cannot be shed in the stool of a vaccinated person. IPV cannot cause vaccine associated paralysis, and is safe to use in immunodeficient persons or in household contacts of immunodeficient persons.

The disadvantages of IPV are that it requires injection, and there are currently no combination vaccines that contain IPV licensed in the United States. It is also more expensive than OPV. The duration of immunity to IPV is not known with certainty, although it likely provides protection for many years after a complete series.

IPV appears to produce less local gastrointestinal immunity than does OPV, so persons who receive IPV are more readily infected with wild polio virus than OPV recipients. A person who received IPV could become infected with wild polio virus in an endemic area and could be shedding wild virus when he or she returned to the United States. The infected person would be protected from paralytic polio, but the wild virus being shed in his or her stool could spread to contacts and result in transmission to a contact.
**Oral poliovirus vaccine (OPV)**

Live oral poliovirus vaccine (OPV) has been the vaccine of choice for routine vaccination in the United States for the past 30 years. It remains the vaccine of choice for most countries in the world. OPV is highly effective in producing immunity to poliovirus. A single dose of OPV produces immunity to all three vaccine viruses in about 50% of recipients. Three doses produces immunity to all 3 poliovirus types in more than 95% of recipients.

The advantages of live oral poliovirus vaccine are that it is very easy to administer and is less expensive than IPV. OPV produces excellent intestinal immunity which helps prevent infection with wild virus. This characteristic is important, because it reduces the chance that a vaccinated person will become infected with wild virus if he or she is exposed while visiting a polio endemic country. Intestinal resistance to infection would also help to minimize spread in the United States if an importation of wild virus were to occur. As with other live virus vaccines, immunity from oral poliovirus vaccine is probably lifelong.

Live oral vaccine-related poliovirus may spread from the recipient to contacts. Polioviruses replicate in the gut and are shed in the stool, and to a lesser degree from the pharynx, so persons coming in contact with fecal material of a vaccinated person may be exposed and infected.

The spread of oral vaccine-related poliovirus from vaccinees to contacts has long been thought to contribute to herd immunity to poliovirus in the United States. However, new information suggests that contact spread of vaccine-related poliovirus in the United States may not be as important as we once believed. A study published in the *Journal of the American Medical Association* in 1996 found only a small increase in seroprevalence of polio antibody that could be attributed to secondary spread of vaccine virus. Most polio immunity attributable to secondary spread of vaccine virus occurred in children with one or no prior doses of OPV. Since most areas of the United States have very high coverage levels with three or more doses of poliovirus vaccine, spread of vaccine virus from vaccinees probably contributes relatively little to the level of polio immunity in the population.

Some experts believe that spread of vaccine-related poliovirus to contacts is actually a disadvantage rather than an advantage. They argue that spread from a vaccinated person makes it difficult to control unintended contact with the vaccine virus, and may occasionally result in vaccine-associated paralytic polio in contacts of vaccinees.
One disadvantage of oral poliovirus vaccine is that seroconversion to all three viruses does not occur with a single dose, and high seroconversion rates to all three vaccine viruses require more than one dose. This probably occurs because of intestinal interference between the three types of vaccine virus, and perhaps because of interference between vaccine viruses and other enteroviruses. A second disadvantage of OPV is a small risk of vaccine-associated paralytic polio in both vaccinees and in contacts of vaccinees.

**Vaccine-Associated Paralytic Poliomyelitis (VAPP)**

Vaccine-associated paralytic polio (VAPP) is a rare adverse event following live oral poliovirus vaccine. Inactivated poliovirus vaccine does not contain live virus, so it cannot cause VAPP.

The mechanism of VAPP is believed to be a mutation, or reversion, of the vaccine virus to a more neurotropic form. These mutated viruses are called revertants. Reversion is believed to occur in almost all vaccine recipients, but it only rarely results in paralytic disease. The paralysis that results is identical to that caused by wild virus, and may be permanent.

It is likely that the longer the vaccine virus replicates in the intestine, the more reversion occurs. The longer that revertants are present, the more likely it is that one will make its way into the central nervous system and cause damage.

VAPP is more likely to occur in persons ≥18 years of age than in children, and is much more likely to occur in immunodeficient children than in those who are immunologically normal. Compared with immunocompetent children, the risk of VAPP is almost 7000 times higher for persons with certain types of immunodeficiencies, particularly B lymphocyte disorders which reduce the synthesis of immune globulins (e.g., agammaglobulinemia and hypogammaglobulinemia). There is no procedure available for identifying persons at risk of paralytic disease, except excluding older persons and screening for immunodeficiency.

Vaccine-associated paralytic polio has been monitored by the CDC since oral poliovirus vaccines began wide use in 1963. While there has been some year to year variation, the number of vaccine-associated cases has remained relatively stable at 5 to 10 a year since that time. Between 1980 and 1996, 142 paralytic polio cases were reported. All but 8 of these cases have been caused by vaccine-related poliovirus.
The 125 VAPP cases reported between 1980 and 1994 have been thoroughly investigated and characterized. During this 15 year period, 49 (39.2%) cases were reported in healthy vaccine recipients, an average of 3 cases per year. The average age of this group was 3 months. Forty cases (32.0%) occurred in healthy contacts of vaccine recipients, an average of 3 cases per year. The average age of this group was 26 years. Six (4.8%) cases were classified as community acquired. In these cases, vaccine virus was recovered from the stool, but there was no known contact with a vaccinated person. The remaining 30 (24.0%) VAPP cases occurred in immunodeficient persons. Twenty-three of these persons (76.6% of immunodeficient VAPP cases) were vaccine recipients, none of whom were known to be immunodeficient before receiving the vaccine. The remaining 7 immunodeficient cases were contacts of vaccine recipients.

The risk of VAPP is not equal for all OPV doses in the vaccination series. The risk of VAPP is 7 to 21 times higher for the first dose than for any other dose in the OPV series.

From 1980 through 1994, 303 million doses of OPV were distributed and 125 cases of VAPP were reported, for an overall risk of VAPP of 1 case per 2.4 million doses. Forty-nine paralytic cases were reported among immunologically normal recipients of OPV from 1980 through 1994. The overall risk to these recipients was one VAPP case per 6.2 million OPV doses. However, 40 (81.6%) of these 49 cases occurred following receipt of the first dose. The risk of VAPP was 1 case per 1.4 million first doses. The risk for all other doses was one per 27.2 million doses.

Forty VAPP cases were reported among contacts of OPV recipients from 1980 through 1994. The overall risk for contacts was 1 in 7.6 million. Sixty-five percent of contact VAPP cases occurred following the first dose in the vaccinee, for a first dose risk of 1 case per 2.2 million doses. The risk for all subsequent doses was 1 case of VAPP per 17.6 million doses.

The reason for this difference by dose is not known with certainty, but is probably because the vaccine virus is able to replicate longer in a completely nonimmune infant. This prolonged replication increases the chance of the emergence of a revertant virus that may cause paralysis. The situation is similar for contacts. A nonimmune child may shed virus longer, increasing the chance of exposure of a contact.
**Vaccination Schedule**

Parents of children who are to be vaccinated should be informed of the poliovirus vaccines available, alternative immunization schedules, and the basis for poliovirus vaccination recommendations. The benefits and risks of the vaccines for individuals and for the community should be discussed.

**Sequential IPV-OPV schedule**

Vaccination schedules using IPV alone or OPV alone are both effective. Schedules using either vaccine alone are acceptable options for preventing poliomyelitis. However, the Advisory Committee on Immunization Practices (ACIP) recommends the use of IPV followed by OPV for primary vaccination of children in the United States.

The highest risk of VAPP is with the first dose of OPV. IPV, when given as the first two doses of the vaccination series is expected to induce antibodies to poliovirus in over 90% of recipients. These antibodies would reduce or eliminate the viremia that results from OPV. Reducing the viremia from live vaccine-related poliovirus would in turn reduce the risk of vaccine-associated paralysis.

The sequential IPV-OPV schedule is expected to produce a high level of individual protection from two doses of IPV and should reduce by 95% VAPP that occurs among OPV recipients. The sequential schedule may also reduce VAPP among household and community contacts of OPV recipients because IPV provides some degree of intestinal and pharyngeal immunity. Continued use of OPV in the series induces intestinal immunity among vaccinees, thereby enhancing community resistance to transmission of wild virus should it be re-introduced. With a sequential schedule, fewer injections are required in the second year of life than would be required if only IPV were used, making compliance with the overall childhood vaccination schedule easier. Finally, stocking of both poliovirus vaccines by health care providers will facilitate parental choice. In the future, licensure of combination products will reduce the number of injections needed to administer the complete series of recommended childhood vaccinations.
For infants, children, and adolescents through secondary school age (generally up to age 18 years), the primary sequential series of IPV and OPV consists of 4 doses.

The vaccination series may be started as early as 6 weeks of age. The primary series is administered at age 2 months (IPV), 4 months (IPV), 12-18 months (OPV), and 4-6 years (OPV). For persons of any age, the first three doses should be separated by at least 4 weeks, although an interval of 6-8 weeks is preferred. It is not necessary to repeat or add doses if the interval between doses is prolonged. Both IPV and OPV can be administered simultaneously with DTP or DTaP (diphtheria and tetanus toxoids and whole-cell or acellular pertussis vaccine), Hib (Haemophilus influenzae type b) vaccines, hepatitis B vaccine, varicella vaccine and measles-mumps-rubella (MMR) vaccine.

The risk of VAPP is highest following the first dose of OPV. In order to have the most impact on VAPP, IPV must be given before any dose of OPV. In addition, a single dose of IPV does not result in significant protection. At least 2 doses of IPV must be given prior to exposure to OPV for maximum impact. If 1 or more doses of OPV have already been given, there is little benefit in switching to IPV.

**OPV schedule**

A schedule using OPV alone is effective for the prevention of polio and is an acceptable alternative to the sequential schedule. An all-OPV schedule is preferred in some circumstances. An all-OPV schedule may be preferred if the child starts the immunization series late (>6 months of age) and the number of required injections might hinder compliance with the accelerated schedule. OPV may also be preferred if the child is going to visit or live in a polio endemic area, because a high level of intestinal resistance to infection with wild poliovirus would be desirable. A parent may refuse to allow the child to receive additional injections, in which case OPV is an acceptable alternative.
If an all-OPV schedule is used, the primary series consists of three doses of vaccine. For infants, the primary series is usually integrated with the other vaccines routinely administered at 2, 4, and 6-18 months of age. For routine vaccination, the usual interval between doses of OPV is 6-8 weeks. However, a minimum interval of 4 weeks may be used if an accelerated schedule is required (e.g., if the child is significantly behind schedule and requires rapid catch-up). If the third dose of OPV is administered before the fourth birthday, a fourth dose of OPV should be provided before school entry (at 4-6 years of age). The fourth dose is not needed if the third dose is given on or after the fourth birthday. It is not necessary to repeat or add doses if the interval between doses is prolonged. Because of an increased risk of VAPP, OPV should not be used for the primary immunization of persons ≥18 years of age.

**IPV schedule**

A schedule using IPV alone is effective for the prevention of polio and is an acceptable alternative to the sequential schedule. An all-IPV schedule is preferred for vaccination of an immunodeficient child or a child with an immunodeficient household contact. IPV is also preferred for primary vaccination of an adult over 18 years of age, such as a traveler to a polio endemic area. To completely eliminate the risk of VAPP, IPV may be preferred when parents do not object to additional injections.
If an all-IPV schedule is used, the primary series consists of three doses of vaccine. In infancy, these primary doses are integrated with the administration of other routinely administered vaccines. The first two doses are recommended at 2 and 4 months of age. The third dose should be given 6-12 months after the second and no earlier than 12-18 months of age. The first and second doses of IPV are necessary to induce a primary immune response, the third dose of IPV ensures “boosting” of antibody titers to high levels. The preferred interval between the second and third doses of IPV is 6 months. However, if accelerated protection is needed, the minimum interval between doses of IPV is 4 weeks. Children who receive three doses of IPV before the fourth birthday should receive a fourth dose before or at school entry. The fourth dose is not needed if the third dose is given on or after the fourth birthday. It is not necessary to repeat or add doses if the interval between doses is prolonged.

**Interchangeability of vaccines**

Completion of poliovirus vaccination with any of the three options (sequential IPV-OPV, OPV alone, or IPV alone) is acceptable. However, four doses of any combination of IPV or OPV by 4-6 years of age is considered a complete poliovirus vaccination series. A minimum interval of 4 weeks should separate all doses of the series.

**Options for reducing the number of injections**

The number of injections needed to administer all recommended childhood vaccines to children 2 and 4 months of age (IPV, DTP or DTaP, *Haemophilus influenzae* type b conjugate [Hib], hepatitis B) can be reduced to three if IPV and Hib combined with hepatitis B vaccine are administered. The number of injections can be reduced to two if OPV and Hib-hepatitis B combination vaccines are administered. An additional option to reduce the number of injections required at the 2 and 4 month visits is to administer hepatitis B vaccine on a schedule of birth, 1, and 6 months of age.
Polio Vaccination of Adults

Routine vaccination of adults (>18 years of age) who reside in the United States is not necessary because most adults are already immune and have a very small risk of exposure to wild poliovirus in the United States.

Some adults (>18 years of age) are at increased risk of infection with poliovirus. These include travelers to areas where poliomyelitis is endemic or epidemic, laboratory workers handling specimens which may contain polioviruses, and health-care workers in close contact with patients who may be excreting wild polioviruses. In addition, members of specific population groups with a current disease caused by wild polioviruses (e.g., during an outbreak), are also at increased risk.

Recommendations for poliovirus vaccination of adults in the above categories depending upon the previous vaccination history and the time available before protection is required, are as follows:

Unvaccinated adults

For adults at increased risk of exposure to poliomyelitis, primary immunization with IPV is recommended whenever feasible. IPV is preferred because the risk of vaccine-associated paralysis following OPV is slightly higher in adults than in children. The recommended schedule is two doses given at 1- to 2-month intervals, and a third dose given 6 to 12 months later.

In circumstances where time will not allow completion of this schedule (e.g., impending travel), the following alternatives are recommended.

If 8 weeks or more are available before protection is needed, three doses of IPV should be given at least 4 weeks apart. If 4-8 weeks are available before protection is needed, two doses of IPV should be given at least 4 weeks apart. If less than 4 weeks are available before protection is needed, a single dose of either OPV or IPV is recommended. In all instances, the remaining doses of vaccine should be given later, at the recommended intervals, if the person remains at increased risk.
**Adults previously given a complete primary course of OPV or IPV**

Adults who are at increased risk of exposure to poliomyelitis and who have previously completed a primary course of OPV may be given another dose of OPV. These adults are not at increased risk of VAPP. The need for further supplementary doses has not been established. Those adults who previously completed a primary course of IPV may be given a dose of either IPV or OPV.

**Incompletely immunized adults**

Adults who are at increased risk of exposure to poliomyelitis and who have previously received less than a full primary course of OPV or IPV should be given the remaining required doses of either vaccine, regardless of the interval since the last dose and type of vaccine previously received. It is not necessary to restart the series of either vaccine if the schedule has been interrupted.

**Household contacts of children receiving OPV**

Adults who have not been adequately immunized against poliomyelitis with OPV or IPV have a minimal risk for developing OPV-associated paralytic poliomyelitis when OPV is administered to children in their households. Since 1980, one or two cases of VAPP have occurred each year among adult household contacts of children who received OPV. During that time approximately 19 million doses of OPV were distributed yearly.

Because of the overriding importance of ensuring prompt and complete immunization, sequential IPV-OPV vaccination of children should begin regardless of the poliovirus vaccine status of adult household contacts. If unvaccinated or inadequately vaccinated persons are known to reside in the child's household, IPV alone should be used to complete the child's vaccination, thereby reducing the already minimal risk for VAPP among adult household contacts.

**Contraindications and Precautions to Vaccination**

Serious allergic reaction to a vaccine component, or following a prior dose of vaccine, is a contraindication to further doses of that vaccine. Since IPV contains trace amounts of streptomycin and neomycin, there is a possibility of hypersensitivity reactions in individuals sensitive to these antibiotics. Persons with anaphylactic hypersensitivity, hives, etc., should not receive IPV. Persons with allergies that are not anaphylactic, such as skin contact sensitivity, may be vaccinated.
Moderate or severe acute illness is a precaution for both IPV and OPV. However, mild illness, including mild diarrhea, is not a contraindication.

OPV should not be given to individuals or household contacts of individuals who have immune deficiency diseases, immune depression (due to disease or therapy), or if there is suspected familial immune deficiency. IPV may be substituted for OPV in these circumstances.

In general, neither OPV nor IPV should be given to pregnant women unless immediate protection is needed (in which case OPV is the vaccine of choice).

Invalid Contraindications

Breast feeding does not interfere with successful immunization against poliomyelitis with IPV or OPV. A dose of IPV may be administered to a child with diarrhea. A dose of OPV may be administered to a child with mild diarrhea. Minor upper respiratory illnesses with or without fever, mild to moderate local reactions to a previous dose of vaccine, current antimicrobial therapy, and the convalescent phase of an acute illness are not contraindications for vaccination with IPV or OPV.

Inadvertent administration of OPV to members of households with immunocompromised persons

If OPV is inadvertently administered to a household contact of an immunodeficient patient, the patient and the recipient of OPV should avoid close contact for approximately 4-6 weeks after vaccination. If this is not feasible, rigorous hygiene and hand washing after contact with feces (e.g., after diaper changing) and avoidance of contact with saliva (e.g., sharing food or utensils) may be an acceptable but probably a less effective alternative. Maximum excretion of vaccine virus occurs within 4 weeks after oral vaccination.

Regurgitation of OPV

Infants may not completely swallow OPV. If, in the judgement of the person administering the vaccine, a substantial amount of vaccine is regurgitated or vomited soon after administration (i.e., within 5-10 minutes), another dose can be administered during the same visit. If this repeat dose is not retained, neither dose should be counted, and the vaccine should be readministered during a later visit.
Adverse Events Following Vaccination

**IPV**

No serious side effects of enhanced-potency IPV have been documented. Because IPV contains trace amounts of streptomycin, polymyxin B, and neomycin, hypersensitivity reactions may occur among persons sensitive to these antibiotics.

**OPV**

In rare instances, administration of OPV has been associated with paralysis in healthy recipients and their contacts. No procedures are currently available for identifying persons, other than those with immunodeficiency, who are likely to experience such adverse reactions. Although the risk of vaccine-associated paralysis is minimal, vaccinees (or their parents) and their susceptible, close, personal contacts should be informed of this risk (see section on vaccine-associated paralytic poliomyelitis). OPV may very rarely cause death due to paralytic poliomyelitis.

Storage and Handling

**OPV**

The vaccine should arrive frozen on dry ice. It should be maintained at a temperature of 0°C (32°F) or lower and may be in either a frozen or liquid state. Unopened vaccine may be thawed and refrozen for a maximum of 10 freeze-thaw cycles, if the total cumulative duration of thaw does not exceed 24 hours and provided the temperature does not exceed 8°C (46°F) during the period of the thaw. Unopened vaccine may be used for up to 30 days if stored between 2°-8°C (35°-46°F). Opened multiple-dose vials of vaccine can be used for up to 7 days if stored at 2°-8°C. The vaccine should be pink or red in color.

**IPV**

The vaccine may be shipped without refrigeration provided it is delivered within 4 days. It should be maintained at 2°-8°C (35°-46°F). The vaccine should be perfectly clear and colorless. Any vaccine showing particulate matter, turbidity, or change in color, should be discarded.
Outbreak Investigation and Control

Collect preliminary clinical and epidemiological information (including vaccine history and contact with OPV vaccines) on any suspected case of paralytic polio. Notify the National Immunization Program, Centers for Disease Control and Prevention ([404] 639-8255) after all appropriate local and state health authorities have been notified. Intensify field investigation to verify information and collect appropriate specimens for viral isolates and serology.

Even one case of paralytic poliomyelitis demands immediate attention. If the evidence indicates vaccine-associated disease, then no outbreak control program is needed. If, however, evidence indicates wild virus (for example, two cases in a community), then all unvaccinated individuals in the epidemic area who are over 6 weeks of age and whose vaccine histories are uncertain should be vaccinated.

Polio Eradication

Following the widespread use of poliovirus vaccine in the mid-1950s, the incidence of poliomyelitis declined rapidly in many industrialized countries. In the United States, the number of cases of paralytic poliomyelitis reported annually declined from >20,000 cases in 1952 to <100 cases in the mid-1960s. The last indigenous transmission of wild poliovirus in the United States was in 1979.

In 1985, the member countries of the Pan American Health Organization adopted the goal of eliminating poliomyelitis from the Western Hemisphere by 1990. The strategy to achieve this goal included increasing vaccination coverage; enhancing surveillance for suspected cases (i.e., surveillance for acute flaccid paralysis); and using supplemental immunization strategies such as national immunization days (NIDs), house-to-house vaccination, and containment activities. Since 1991, when the last wild-virus-associated indigenous case was reported from Peru, no additional cases of poliomyelitis have been confirmed despite intensive surveillance. In September 1994, an international commission certified the Western hemisphere to be free of indigenous wild poliovirus. The commission based its judgment on detailed reports from national certification commissions that had been convened in every country in the region.
In 1988, the World Health Assembly (the governing body of the World Health Organization) adopted the goal of global eradication of poliomyelitis by the year 2000. Substantial progress toward meeting this objective has already been achieved in many WHO regions, including East Asia, the Middle East, Southern and Eastern Africa, and Europe. By the end of 1996, almost all polio-endemic countries outside the African region of WHO will have conducted NIDs, as had >50% of African countries. The number of reported cases of paralytic polio, as well as the number of countries reporting cases, has decreased significantly since the global eradication program began.

The polio eradication initiative is supported by a coalition of international organizations that includes WHO, the United Nations children’s Fund (UNICEF), and other bilateral and multilateral organizations. Rotary International has contributed more than $240 million to support the eradication initiative.

**Post-Polio Syndrome**

After an interval of 30-40 years, some persons (25%-40%) who contracted paralytic poliomyelitis in childhood may experience new muscle pain and exacerbation of existing weakness, or develop new weakness or paralysis. This disease entity is referred to as post-polio syndrome. Factors which enhance the risk of post-polio syndrome include increasing length of time since acute poliovirus infection, presence of permanent residual impairment after recovery from the acute illness, and female gender. The pathogenesis of post-polio syndrome is thought to involve the failure of oversized motor units created during the recovery process of paralytic poliomyelitis. Post-polio syndrome is not an infectious process, and persons experiencing the syndrome do not shed poliovirus.

Several support groups have been established to assist and provide information to persons with post-polio syndrome, and their families.

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St. Louis, MO 63110-1406
(314) 534-0475

**March of Dimes**
Birth Defects Foundation
Community Services Department
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White Plains, NY 10605
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**Polio Summary**
- Eliminated from United States
- Vaccine-associated paralysis rare
- Sequential IPV-OPV schedule recommended
- Global eradication
POLIOMYELITIS