The Story of Polio
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Presenting To
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Phoenix, AZ

Karen Lewis, M.D.| ADHS Medical Director for Immunizations
Childhood memories of polio fears
1789: Dr. Michael Underwood (Britain): Debility of the lower extremities
1840: Dr. Jacob von Heine (Germany)—First systematic investigation; theory contagious
1894 1st significant outbreak of infantile paralysis subsequently identified as polio is documented in the US
1907: Dr. Ivar Wickman (Sweden) categorizes different clinical types of polio
1908: Dr. Karl Landsteiner and Dr. Erwin Popper (Austria) hypothesize polio is virus
1916: NY polio epidemic killed 2,400 people (mostly children) and left thousands more with a life-long disability.

The first major U.S. polio epidemic occurred in 1894 in Vermont, with 132 cases. New York City experienced its first large-scale outbreak in 1916, with more than 27,000 cases and 6,000 deaths. Kept popping up mysteriously and worsening as sanitation improved. More likely to hit Enterovirus

Serotypes 1,2,3
Oral-fecal and oral-oral spread
Most infections not symptomatic
5% nonspecific fever, headache, sore throat
<1% paralysis
Deaths in 5-10% children, 15-30% adults

Withered legs on Egyptian carvings
1900s epidemics in Europe and the US
Almost all children infected with 1:200 developing paralytic polio 1952 had the worst polio outbreak with 57,879 total cases, >21,000 paralytic cases, and 3,145 deaths. Pink Book

1916, 1952 — Polio: Polio occurred primarily in July, August, and September and hit regardless of geographic region, economic status, or population density. Relatively few people showed any symptoms and even fewer died or experienced paralysis, but the physical effects were dramatic. Communities reacted with dread because no one understood how or why people got it, and because children were the most frequently affected. http://masterofpublichealth.org/2010/top-10-public-health-disasters-of-the-20th-century/ Posted July 9, 2010

Major polio epidemics were unknown before the 20th century; localized paralytic polio epidemics began to appear in Europe and the United States around 1900. The first report of multiple polio cases was published in 1843 and described an 1841 outbreak in Louisiana. A fifty-year gap occurs before the next U.S. report—a cluster of 26 cases in Boston in 1893. The first recognized U.S. polio epidemic occurred the following year in Vermont with 132 total cases (18 deaths), including several cases in adults. Numerous epidemics of varying magnitude began to appear throughout the country; by 1907 approximately 2,500 cases of poliomyelitis were reported in New York City.

On Saturday, June 17, 1916, an official announcement of the existence of an epidemic polio infection was made in Brooklyn, New York. That year, there were over 27,000 cases and more than 6,000 deaths due to polio in the United States, with over 2,000 deaths in New York City alone. The names and addresses of individuals with confirmed polio cases were published daily in the press, their houses were identified with placards, and their families were quarantined. Dr. Hiram M. Hiller, Jr. was one of the physicians in several cities who realized what they were dealing with, but the nature of the disease remained largely a mystery. The 1916 epidemic caused widespread panic and thousands fled the city to nearby mountain resorts; movie theaters were closed, meetings were canceled, public gatherings were almost nonexistent, and children were warned not to drink from water fountains, and told to avoid amusement parks, swimming pools, and beaches.

From 1916 onward, a polio epidemic appeared each summer in at least one part of the country, with the most serious occurring in the 1940s and 1950s. In the epidemic of 1949, 2,720 deaths from the disease occurred in the United States and 42,173 cases were reported and Canada and the United Kingdom were also affected.

Prior to the 20th century polio infections were rarely seen in infants before 6 months of age and most cases occurred in children 6 months to 4 years of age. Young children who contract polio generally suffer only mild symptoms, but as a result they become permanently immune to the disease. In developed countries during the late 19th and early 20th centuries, improvements were being made in community
sanitation, including improved sewage disposal and clean water supplies. Better hygiene meant that infants and young children had fewer opportunities to encounter and develop immunity to polio. Exposure to poliovirus was therefore delayed until late childhood or adult life, when it was more likely to take the paralytic form.[19] In children, paralysis due to polio occurs in one in 1000 cases, while in adults, paralysis occurs in one in 75 cases.[21] By 1950, the peak age incidence of paralytic poliomyelitis in the United States had shifted from infants to children aged 5 to 9 years; about one-third of the cases were reported in persons over 15 years of age.[22] Accordingly, the rate of paralysis and death due to polio infection also increased during this time.[1] In the United States, the 1952 polio epidemic was the worst outbreak in the nation's history, and is credited with heightening parents’ fears of the disease and focusing public awareness on the need for a vaccine.[23] Of the 57,628 cases reported that year 3,145 died and 21,269 were left with mild to disabling paralysis.[23][24] https://en.wikipedia.org/wiki/History_of_poliomyelitis

Polioviruses are human enteroviruses of the Picornaviridae family.
Higher standard of living, infants were not getting exposed while still had maternal antibodies.
Illustrations from Clip Art

<table>
<thead>
<tr>
<th>Percentage</th>
<th>Description</th>
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<tbody>
<tr>
<td>72%</td>
<td>No Symptoms</td>
</tr>
<tr>
<td>24%</td>
<td>Nonspecific Symptoms</td>
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<tr>
<td>1-5%</td>
<td>Aseptic Meningitis</td>
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<tr>
<td>&lt; 1%</td>
<td>Paralysis</td>
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Examining for Acute Flaccid Paralysis

This historic image depicts an Epidemic Intelligence Service (EIS) Officer using a muscle evaluation test for polio on this young patient. EIS is the country's critical epidemiology training service, combating the causes of major epidemics. Over the past 50 years, EIS officers have played pivotal roles in combating the root causes of major epidemics.
Images of Polio Infection

Right: Two children with polio receiving physical therapy. CDC. Immunize.org PHIL 2612
L. Images like this were used to encourage polio vaccination cdc. Immunize.org PHIL___________

The typical clinical manifestation of paralytic poliomyelitis is acute flaccid paralysis (AFP) affecting the limbs, principally the legs, usually asymmetrically; sensation remains intact.

Persistent paralysis and resulting deformities are common sequelae.
The case-fatality rates among paralytic cases range from 5% to 10% in children and from 15% to 30% in adolescents and adults, predominantly associated with bulbar involvement.

Post-polio syndrome, with symptoms appearing 15–30 years after recovery from the original paralytic attack, occurs in 25%–50% of cases; symptoms include acute or increased muscular weakness, pain in the muscles, and fatigue.

No specific anti-viral drugs are available for poliomyelitis.
Treatment consists of supportive, symptomatic care during the acute phase, including respiratory support in cases with respiratory muscle paralysis.
Neuromuscular sequelae are mitigated by physiotherapy and orthopaedic treatment.
WER Feb 28, 2014
Iron Lung

CDC PHIL 12009.
This 1960 photograph depicted a nurse caring for a victim of a Rhode Island polio epidemic, who was inside an Emerson respirator, also sometimes referred to as an “iron lung” machine. Devices such as these were used by polio patients whose ability to breath was paralyzed due to this crippling viral disease.

See Phil images 6524, 6525, 6526, 6527, 6529, 6530, 6531, 6533, and 6534 for pictures of an iron lung that was donated to the CDC’s Global Health Odyssey by the family of polio patient Mr. Barton Hebert of Covington, Louisiana, who’d used the device from the late 1950s until his death in 2003. Notice the mirror attached overhead that enabled the patient to see about his environment.

In 1959, there were 1,200 people using tank respirators in the United States, but by 2004 there were only 39.[29] By 2014, there were only 10 people left with an iron lung.[30] https://en.wikipedia.org/wiki/Iron_lung

The first iron lung used in the treatment of polio victims was invented by Philip Drinker, Louis Agassiz Shaw, and James Wilson at Harvard, and tested October 12, 1928, at Children's Hospital, Boston.[43] The original Drinker iron lung was powered by an electric motor attached to two vacuum cleaners, and worked by changing the pressure inside the machine. When the pressure is lowered, the chest cavity expands, trying to fill this partial vacuum. When the pressure is raised the chest cavity contracts. This expansion and contraction mimics the physiology of normal breathing. The design of the iron lung was subsequently improved by using a bellows attached
directly to the machine, and John Haven Emerson modified the design to make production less expensive.[43] The Emerson Iron Lung was produced until 1970.[44] Other respiratory aids were used such as the Bragg-Paul Pulsator, and the "rocking bed" for patients with less critical breathing difficulties.[45]

During the polio epidemics, the iron lung saved many thousands of lives, but the machine was large, cumbersome and very expensive:[46] in the 1930s, an iron lung cost about $1,500—about the same price as the average home.[47] The cost of running the machine was also prohibitive, as patients were encased in the metal chambers for months, years and sometimes for life:[44] even with an iron lung the fatality rate for patients with bulbar polio exceeded 90%.[48]

Polio Damages
Anterior Horn Cells in the Spine

Polio=Gray; Myelon=spinal or nerve tissue; it is=inflammation

Polio Serotypes 1, 2, 3
Prevaccine era polio was the leading cause of permanent disability in children
Virtually all children became infected by polioviruses with about 1 in 200 developing paralysis
Fecal-oral more common where sanitation poor; oral-oral transmission may be more common where sanitation is good. WER Feb 28, 2014
Paralytic poliomyelitis occurs when poliovirus enters the central nervous system and replicates in anterior horn cells (motor neurons) of the spinal cord and is experienced in <1% of poliovirus infections in children <5 years of age, varying with serotype and age.
The ratio of paralytic cases to infections was estimated per 100 infections at approximately 0.5 for serotype 1, 0.05 for serotype 2, and 0.08 for serotype 3, based on data from 15 countries.
Depending on the degree and extent to which motor neurons are affected, temporary or permanent paralysis of the affected muscles may ensue. In rare cases, viral destruction of bulbar cells results in respiratory paralysis and death. WER Feb 28, 2014
No specific anti-viral drugs are available for poliomyelitis.
Treatment consists of supportive, symptomatic care during the acute phase, including
respiratory support in cases with respiratory muscle paralysis. Neuromuscular sequelae are mitigated by physiotherapy and orthopaedic treatment. Immunocompetent individuals infected by poliovirus develop immunity through humoral (circulating antibody) and mucosal (secretory immunoglobulin A) immune responses. The presence of neutralizing antibody against polioviruses indicates protective immunity; for poliomyelitis, detectable antibody is an excellent correlate of protection against paralytic disease. However, immunity is serotype-specific with no cross-protection between serotypes. Mucosal immunity decreases the replication and excretion (shedding) of the virus, and thus provides a potential barrier to its transmission. Individuals with B-cell related immunodeficiency disorders are at increased risk for paralytic manifestations of poliomyelitis or prolonged excretion of virus.
Age 39 in 1921—Developed acute onset of paralysis after vigorous exercise and was diagnosed with polio
Established the National Infantile Paralysis Foundation that later became the March of Dimes
https://www.history.com/news/franklin‐roosevelts‐personal‐polio‐crusade
FDR developed polio in 1921 at age 39 (?GBS)
First—Birthday Balls
Basil O’Connor (former law partner) & Roosevelt created the National Foundation for Infantile Paralysis (Jan 3, 1938→soon renamed as the March of Dimes.
Despite the best efforts of the March of Dimes and others in the medical community, polio continued its destructive path, with a new outbreak seemingly every summer. In fact, it was the decade following Roosevelt’s death that saw the worst of the crisis—in 1949 alone more than 2,700 Americans died from the disease. The March of Dimes continued financial support for medical research finally paid off when Jonas Salk, a young doctor whose work was funded by a March of Dimes grant, developed a new vaccine to combat polio. In 1954, March of Dimes helped support a mass vaccination of more than 1.8 million schoolchildren, and just a year later Salk’s vaccine had been approved for even more widespread usage, leading to the virtual eradication of polio in the developed world.

Franklin D. Roosevelt proved instrumental in the vaccine’s development.
A year after his nomination as a Democratic vice presidential candidate, rising
political star Franklin D. Roosevelt contracted polio while vacationing at his summer home on Campobello Island in 1921. The disease left the legs of the 39-year-old future president permanently paralyzed. In 1938, five years after entering the White House, Roosevelt helped to create the National Foundation for Infantile Paralysis, later renamed the March of Dimes Foundation, which became the primary funding source for Salk’s vaccine trials. Employing “poster children” and enlisting the star power of celebrities from Mickey Rooney to Mickey Mouse, the grassroots organization run by Roosevelt’s former Wall Street law partner Basil O’Connor was raising more than $20 million per year by the late 1940s.
Poliovirus Serotypes

CDC PHIL 22498—3D demonstration of poliovirus
1931: Sir Macfarlane Burnet and Dame Jeam MacNamara identify several types of polio virus, types 1, 2 and 3
1948: Thomas Weller and Frederick Robbins successfully grow live polio virus in live cells
1955: Salk vaccine
1961: Sabin: OPV
1979: Last case of polio in the US
1988: World Health Assembly passes a resolution to eradicate polio by 2000. Global Polio Eradication Initiative is launched
1994: WHO region of the Americas certified polio-free
Polioviruses possess a single-stranded, RNA genome and a protein capsid. The 3 serotypes of polioviruses have slightly different capsid proteins. Polioviruses share most of their biochemical and biophysical properties with other enteroviruses. They are resistant to inactivation by many common detergents and disinfectants, including soaps, but are rapidly inactivated by exposure to ultraviolet light.
Viral infectivity is stable for months at +4 °C and for days at +30 °C.
Wer Feb 28, 2014
https://www.historyofvaccines.org/content/early-polio-vaccine-trials

1935: This year, two separate teams were at work developing and testing a polio
vaccine. Both projects came to disastrous ends.
At New York University, Maurice Brodie, MD (1903-1939), a young researcher, prepared a killed poliovirus vaccine, testing it on chimpanzees, on himself, and finally on children. He enrolled about 11,000 individuals (in both control and vaccine groups) in his trial.
Meanwhile, John Kolmer, MD, of Temple University in Philadelphia developed an attenuated poliovirus vaccine, which he tested in about 10,000 children. The tests proved a disaster. Several subjects died of polio, and many were paralyzed, made ill, or suffered allergic reactions to the vaccines.

**Although polio was the most feared disease of the 20th century, it was hardly the deadliest.**

“Polio was never the raging epidemic portrayed in the media, not even at its height in the 1940s and 1950s,” writes David M. Oshinsky in his Pulitzer Prize winning book “Polio: An American Story.” During those decades, 10 times as many children died in accidents and three times as many succumbed to cancer. Oshinsky notes that polio inspired such fear because it struck without warning and researchers were unsure of how it spread from person to person. In the years following World War II, polls found the only thing Americans feared more than polio was nuclear war.

http://polioeradication.org/polio‐today/history‐of‐polio

PHIL 1837 This transmission electron microscopic (TEM), negative stain image, reveals some of the ultrastructural features exhibited by a grouping of icosahedral-shaped polio virus particle.
PHIL 22498
This illustration provides a 3-dimensional (3D) graphical representation of a tightly packed icosahedral poliovirus particle that consists of 60 copies each of capsid polypeptides designated as VP (viral protein) 1 (Pink); VP2 (Green); VP3(Purple) and VP4 (not shown).; The particle is composed of units of four capsid polypeptides that interact in groups of five; resulting in a viral particle that has 5-fold (pentamer) and 12-fold symmetry.; A deep “canyon” on the capsid surface surrounds the apex of each pentamer of the virus.; The canyon together with the pentamer apex are used as the site for capsid binding to cellular receptors.
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WER Feb 28, 2014
While most scientists believed that effective vaccines could only be developed with live viruses, Salk developed a “killed-virus” vaccine by growing samples of the virus and then deactivating them by adding formaldehyde so that they could no longer reproduce.

By injecting the benign strains into the bloodstream, the vaccine tricked the immune system into manufacturing protective antibodies without the need to introduce a weakened form of the virus into healthy patients.

Many researchers such as Polish-born virologist Albert Sabin, who was developing an oral “live-virus” polio vaccine, called Salk’s approach dangerous. Sabin even belittled Salk as “a mere kitchen chemist.”

The hard-charging O’Connor, however, had grown impatient at the time-consuming process of developing a live-virus vaccine and put the resources of the March of Dimes behind Salk. https://www.history.com/news/8-things-you-may-not-know-about-jonas-salk-and-the-polio-vaccine

https://www.history.com/this-day-in-history/salk-announces-polio-vaccine

Salk tested the vaccine on himself and his family.

After successfully inoculating thousands of monkeys, Salk began the risky step of testing the vaccine on humans in 1952.

In addition to administering the vaccine to children at two Pittsburgh-area institutions, Salk injected himself, his wife and his three sons in his kitchen after boiling the needles and syringes on his stovetop.
March 26, 1953—Salk announced polio vaccine; CBS national radio evening of March 25; JAMA March 27, 1953

[https://www.history.com/this-day-in-history/children-receive-first-polio-vaccine](https://www.history.com/this-day-in-history/children-receive-first-polio-vaccine)

[https://www.history.com/this-day-in-history/polio-vaccine-trials-begin](https://www.history.com/this-day-in-history/polio-vaccine-trials-begin) April 26 1954

[https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1114166/pdf/1233.pdf](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1114166/pdf/1233.pdf) Over 600,000 schoolchildren were injected with vaccine or placebo and over a million others participated as “observed” controls

[https://www.history.com/this-day-in-history/salk-announces-polio-vaccine](https://www.history.com/this-day-in-history/salk-announces-polio-vaccine)

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On April 26, 1954, six-year-old Randy Kerr was injected with the Salk vaccine at the Franklin Sherman Elementary School in McLean, Virginia.

By the end of June, an unprecedented 1.8 million people, including hundreds of thousands of school children, joined him in becoming “polio pioneers.”

For the first time, researchers used the double-blind method, now standard, in which neither the patient nor person administering the inoculation knew if it was a vaccine or placebo.

Although no one was certain that the vaccine was perfectly safe—in fact, Sabin argued it would cause more cases of polio than it would prevent—there was no shortage of volunteers.

Over 600,000 school children were injected with vaccine or placebo.
From Encyclopedia of Twentieth-Century Journalists by William H. Taft.
This reference indicates that James Thomas Berryman (1902-1971) followed his father (Clifford Berryman: 1869-1949) as a political cartoonist on the Washington Star. His father was also a Pulitzer Prize winning political cartoonist who worked for the Washington Star, but he died in 1949 so his father could not have drawn this cartoon.


On April 12, 1955, the day the Salk vaccine was declared “safe, effective and potent,” legendary CBS newsman Edward R. Morrow interviewed its creator and asked who owned the patent. “Well, the people, I would say,” said Salk in light of the millions of charitable donations raised by the March of Dimes that funded the vaccine’s research and field testing. “There is no patent. Could you patent the sun?” Lawyers for the foundation had investigated the possibility of patenting the vaccine but did not pursue it, in part because of Salk’s reluctance. https://www.history.com/news/8-things-you-may-not-know-about-jonas-salk-and-the-polio-vaccine
What’s more, Salk’s choice of formalin to generate his polio vaccine was bold. Earlier, in the 1930s, Canadian scientist Maurice Brodie tested a formalin-killed polio vaccine in twelve children, with disastrous results. Several of the children developed paralytic poliomyelitis (4).
In April 1955 more than 200,000 children in five Western and mid-Western USA states received a polio vaccine in which the process of inactivating the live virus proved to be defective. Within days there were reports of paralysis and within a month the first mass vaccination programme against polio had to be abandoned. Subsequent investigations revealed that the vaccine, manufactured by the California-based family firm of Cutter Laboratories, had caused 40,000 cases of polio, leaving 200 children with varying degrees of paralysis and killing 10.
Born in Poland in 1906. Picture taken in 1953
Head of Pediatric Research at the University of Cincinnati
The first inactive polio vaccine (IPV), which required an injection administered by a doctor, was created in 1954 by Dr. Jonas Salk. During the same time period, Sabin discovered that the polio virus attacked and multiplied in the intestines, as opposed to the respiratory tract as was previously believed. He began testing a new polio vaccine on humans, starting with his own family. In 1956, Sabin reported that he had developed an oral polio vaccination (OPV), taken either as a syrup or a pill, that worked in the intestines to block the virus from entering the bloodstream. Many believed that Salk’s vaccine had eradicated polio, so health officials in the United States were hesitant to support Sabin’s new vaccine. However, whereas Salk’s vaccine was effective in preventing most of the complications of polio, it did not prevent the initial intestinal infection. Even with Salk’s vaccine, people could carry the virus and spread it without showing any signs of the disease. In response to the lack of support for his vaccine in the United States, Sabin took it to the USSR in 1959 where it was administered to more than 100 million people with excellent results.
Following Sabin’s success in the USSR, American health officials were prompted to begin testing his oral vaccine. Sabin’s vaccination had many benefits; it could be administered by volunteers instead of doctors, usually one dose of Sabin’s vaccine provided life-long immunity from the polio virus (furthermore, since it was a live-virus vaccine, immunity could be spread from person to person), it was less expensive, and provided better control over epidemic outbreaks of polio. In 1961, Sabin’s oral
vaccine was approved by the U.S. Public Health Service and replaced Salk's vaccine as the preferred inoculation in the United States. The last known case of polio in the United States was reported in 1979.

Once Sabin's oral vaccine finally became available in 1962, it quickly supplanted Salk's injected vaccine because it was cheaper to produce and easier to administer. Ultimately, both vaccines produced by the bitter rivals nearly eradicated the disease from the planet. According to the World Health Organization (WHO), there were only 416 reported cases of polio worldwide in 2013, mostly confined to a handful of Asian and African countries. Since Sabin's live-virus vaccine, which is responsible for about a dozen cases of polio each year, is seen as the final obstacle to eliminating the disease in most of the world, the WHO has urged polio-free countries to return to Salk's killed-virus vaccine.

OPV is composed of live attenuated polioviruses derived by passage of their parent wild-type poliovirus (WPV) strains in nonhuman cells to obtain the 3 vaccine strains (Sabin 1, 2, and 3). Attenuation of the virus in culture greatly reduces its neurovirulence and transmissibility.
IPV, first developed and licensed in 1955, is given by injection and is available only in trivalent form.
OPV was licensed in 1961 as a monovalent (mOPV) vaccine, followed by a trivalent version (tOPV) licensed for use in 1963.
Available data suggest differences in the epidemiology of VAPP in developing and industrialized countries.
In the latter, VAPP occurs mainly in early infancy associated with the first dose of OPV and decreases sharply (>10 fold) with subsequent OPV doses.
In lower-income countries, which experience relatively lower rates of vaccine seroconversion, this decline is more gradual and VAPP may occur with second or subsequent doses of OPV, with the age distribution concentrated among children aged 1–4 years.
Data from India and Iran suggest that in lower-income settings, the age at onset of VAPP is higher, and largely associated with second or subsequent OPV doses.
The main factors contributing to this difference are believed to be lower immune responsiveness to OPV and higher prevalence of maternally-derived antibody in populations in low-income settings.
Cases of VAPP are clinically indistinguishable from poliomyelitis caused by WPV. The incidence of VAPP has been estimated at 2–4 cases/million birth cohort per year.
in countries using OPV. VAPP occurs in both OPV recipients and their unimmunized contacts. OPV2 is the cause of 40% of cases of VAPP.
CDC. PHIL 8286. This historical image, which depicts workers creating a billboard in Columbus, Georgia, shows one of the communication modes, the billboard, used to promote public health awareness, in this case, polio vaccinations within a community. This campaign was produced by the former U.S. Department of Health, Education and Welfare's, Public Health Service, Bureau State Services, and what was the Communicable Disease Center, in cooperation with the Georgia Department of Public Health and the Muscogee Health Department. The billboard, as well as television, magazines, and pamphlets, are only some of the myriad of modalities implemented when information of this kind is disseminated throughout society, and across cultural barriers.

In the early 1950's, there were more than 20,000 cases of polio each year. After polio vaccination began in 1955, cases dropped significantly. Public health officials used every communications media available to promote the vaccination. By 1960, the number of polio cases dropped to about 3,000, and by 1979 there were only about 10.
Fecal oral more common with poor sanitation
As per WER Feb 28, 2014
Most people infected with poliovirus have no symptoms, with viral replication occurring in, and limited to the alimentary tract or pharynx. Approximately 25% of those infected develop minor symptoms, usually fever, headache and sore throat. The incubation period is usually 7–10 days (range 4–35 days). WER Feb 28, 2014
During the first 1–3 weeks following vaccination, the majority of non-immune vaccine recipients shed OPV in nasopharyngeal secretions and feces. In unvaccinated populations, these vaccine viruses are easily transmitted within and outside the household, with the collateral benefit of protecting non-immune individuals or boosting existing immunity in others.
While non-immune vaccine recipients shed virus after initial OPV vaccination, shedding is significantly reduced when the vaccine is administered to children who had previously received OPV.
IPV licensed 1955
1961 type 1 and type 2 licensed;
1962 type 3 licensed
Trivalent OPV licensed 1963
The effectiveness of OPV in controlling poliomyelitis and eliminating the circulation of wild polioviruses is amply demonstrated by the sharp decline in the number of poliomyelitis cases following the introduction of OPV in both industrialized and developing countries.
In the early 20th century, polio was one of the most feared diseases in industrialized countries, paralysing hundreds of thousands of children every year. Soon after the introduction of effective vaccines in the 1950s and 1960s however, polio was brought under control and practically eliminated as a public health problem in these countries.

It took somewhat longer for polio to be recognized as a major problem in developing countries. Lamineness surveys during the 1970s revealed that the disease was also prevalent in developing countries. As a result, during the 1970s routine immunization was introduced worldwide as part of national immunization programmes, helping to control the disease in many developing countries.

Left: Child with a severely deformed leg due to polio. Immunize.org PHIL 5578
Right: WHO. Immunize.org The wild poliovirus usually paralyses children under five years old

1988 Global Polio Eradication Initiative was established
annual global burden of paralytic poliomyelitis was estimated to be more than 350,000 cases reported in 125 countries
Last case of WPV type 2 occurred in India in 1999
No case due to WPV type 3 detected since Nov 10, 2012. WER Feb 28, 2014
In 1988, when the Global Polio Eradication Initiative began, polio paralysed more than 1000 children worldwide every day. Since then, more than 2.5 billion children have been immunized against polio thanks to the cooperation of more than 200 countries and 20 million volunteers, backed by an international investment of more than US$ 11 billion.

There are now only 3 countries that have never stopped polio transmission and global incidence of polio cases has decreased by 99%.

There has also been success in eradicating certain strains of the virus; of the three types of wild polioviruses (WPVs), the last case of type 2 was reported in 1999 and its eradication was declared in September 2015; the most recent case of type 3 dates to November 2012.

However, tackling the last 1% of polio cases has still proved to be difficult. Conflict, political instability, hard-to-reach populations, and poor infrastructure continue to pose challenges to eradicating the disease.

OPV has been the vaccine of choice for the GPEI and enabled the eradication of WPV2 globally in 1999.

OPV is administered as 2 drops (~0.1 mL), directly into the mouth. It is highly heat-sensitive and must be kept frozen for long-term storage or, after thawing, at temperatures between +2 °C and +8 °C for a maximum of 6 months. Vaccine vial monitors give a visual indication of whether the vaccine has been kept at correct temperature conditions.
A group of young schoolchildren, all dressed in their pink-colored school uniforms, who were standing outside a local vaccination center in Indonesia in 2013, about to receive their required dose of oral polio vaccine.

**Advantages of OPV (the Sabin vaccine)**
OPVs are all inexpensive (US $0.12-$0.18 for countries procuring through UNICEF in 2016).
OPVs are safe and effective and offer long lasting protection against the serotype(s) which they target. OPV stimulates good mucosal immunity, which is why it is so effective at interrupting transmission of the virus.
OPVs are administered orally and do not require health professionals or sterile needle syringes. As such, OPVs are easy to administer in mass vaccination campaigns.
For several weeks after vaccination the vaccine virus replicates in the intestine, is excreted and can be spread to others in close contact. This means that in areas with poor hygiene and sanitation, immunization with OPV can result in ‘passive’ immunization of people who have not been vaccinated.

**Disadvantages**
OPV is extremely safe and effective. However, in extremely rare cases (at a rate of approximately 2 to 4 events per 1 million births [1]) the live attenuated vaccine-virus in OPV can cause paralysis. In some cases, it is believed that this may be triggered by an immunodeficiency. The extremely low risk of vaccine-associated paralytic
poliomyelitis (VAPP) is well accepted by most public health programmes. Very rarely, when there is insufficient coverage in a community the vaccine-virus may be able to circulate, mutate and, over the course of 12 to 18 months, reacquire neurovirulence. This is known as a circulating vaccine-derived poliovirus. 
http://polioeradication.org/polio-today/polio-prevention/the-vaccines/opv/

This 2000 photograph depicted the District Immunization Officer of Gorakhpur, India as he was administering polio vaccine to a group of children during a National Immunization Day (NID). Living in a rural area made access to clinical services an unlikely possibility for these children to receive the polio vaccine. Thousands of children throughout the country receive a polio vaccine on each NID. Since its inception in the fall of 1998, the STOP Transmission of Polio (STOP) immunization initiative teams have worked in over 50 countries. In the first years of the program, most STOP team members worked primarily to bolster acute flaccid paralysis (AFP) surveillance, support “national immunization” days, and conduct polio case investigation and follow-up.

There are two kinds of polio vaccine: **IPV**, which is a shot given in the leg or arm, depending on age, which is recommended in the United States today, and a live, oral polio vaccine, or **OPV**, which is drops that are swallowed. IPV may be given at the same time as other vaccines. With regards as to who should receive the polio vaccine, and when, the following are the recommended protocols:

**Children:**

Most people should receive a polio vaccine when they are children. Children get 4 doses of IPV, at these ages:
- A dose at 2 months
- A dose at 6-18 months
- A dose at 4 months
- A booster dose at 4-6 years

**Adults (unvaccinated):** *(Without a written record of prior polio vaccination) at increased risk of exposure to poliomyelitis, primary immunization with IPV is recommended.)*

- The recommended schedule is two doses separated by 1–2 months
- a third dose given 6–12 months after the second dose.

**Safety of OPV**

The only rare serious adverse events associated with OPV are the occurrence of vaccine-associated paralytic poliomyelitis (VAPP) and the emergence of vaccine derived polioviruses (VDPVs).

All available evidence indicates that OPV is non-teratogenic and safe to administer to pregnant women and HIV-infected persons.

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Polio Vaccinators in India

CDC. PHIL 18240. This image was captured in 2011, by U.S. Centers for Disease Control and Prevention (CDC) Health Communication Specialist, Alan Janssen, M.S.P.H., during his tour of India, whereupon, he reviewed the communications elements of the polio eradication program in that region. These two young men have briefly stopped to pose for this snapshot while making their rounds as polio vaccinators. Motorcycles proved to be an integral mode of transportation, enabling eradication team members to access out of the way, remote areas, where many of the Indian inhabitants call their home.

Additional Information:
On March 27, 2014, former CDC Director Dr. Tom Frieden, and senior CDC immunization staff were present when India, along with the other 10 countries of the South East Asia Region, was certified polio-free. The country was once considered the most complex challenge to achieving global polio eradication. Four of the six regions of the World Health Organization have been certified polio-free: the Americas (1994), Western Pacific (2000), Europe (2002) and South East Asia (2014). 80% of the world’s people now live in polio-free areas.
1999. The UN Secretary General agrees to negotiate truces for immunization in the Democratic Republic of Congo. National Immunization Days are conducted in war-torn Liberia.
http://polioeradication.org/polio-today/history-of-polio/
2013 article http://polioeradication.org/news-post/a-year-without-type-3
Wild poliovirus type-2 was declared eradicated by the GCC (Global Commission for the Certification of Poliomyelitis Eradication) in 2015.
2012 The last case of wild poliovirus type 3 is recorded in Nigeria in November 2012.
http://polioeradication.org/polio-today/history-of-polio/ (info through 2016)
Several forms of OPV are currently in use: (i) trivalent (tOPV) against types 1, 2 and 3 which is used in many countries for routine or supplementary vaccination; (ii) bivalent OPV against types 1 and 3 (bOPV) and (iii) monovalent OPVs against type 1 (mOPV1) or against type 3 (mOPV3). Monovalent OPV against type 2 (mOPV2) has been licensed but is expected to be used primarily for outbreak response (see below) using the emergency stockpile.
In 1988, a global movement was started to ensure that every child is vaccinated against polio. At that time, every year more than 350,000 children were paralysed by the disease, in more than 125 countries.

2000: WHO Western Pacific Region certified polio free
2003: Northern Nigeria—unfounded rumors re OPV—new outbreaks
2011: Last case of wild polio reported in India
2012: Last case in Nigeria [not]
2014: South-East Asia declared polio free
2016: tOPV → bOPV

http://polioeradication.org/polio-today/history-of-polio/

• In 2016, only 37 cases were reported, from just 3 countries: Pakistan, Afghanistan and Nigeria. http://polioeradication.org/wp-content/uploads/2017/08/AR2016_EN.pdf

Overall, since the GPEI was launched, the number of cases has fallen by over 99%. More than 16 million people are able to walk today, who would otherwise have been paralysed.

Despite the overall success of the GPEI, in 2018, Pakistan and Afghanistan remain endemic for transmission of WPV type 1 (WPV1).
Circulating vaccine-derived poliovirus (cVDPV)

On very rare occasions, if a population is seriously under-immunized, there are enough susceptible children for the excreted vaccine-derived polioviruses to begin circulating in the community. If the vaccine-virus is able to circulate for a prolonged period of time uninterrupted, it can mutate and, over the course of 12-18 months, reacquire neurovirulence. These viruses are called circulating vaccine-derived polioviruses (cVDPV).

The lower the population immunity, the longer these viruses survive. The longer they survive, the more they replicate, change, and exchange genetic material with other enteroviruses as they spread through a community.

If a population is fully immunized against polio, it will be protected against the spread of both wild and vaccine strains of poliovirus.

Episodes of circulating vaccine-derived poliovirus are rare. Between 2000 and 2011 – a period in which more than 10 billion doses of oral polio vaccine were given worldwide – 20 cVDPV outbreaks occurred, resulting in 580 polio cases. In the same period, in the absence of vaccination with OPV, around 6 million children would have been paralysed by poliovirus.

Immunodeficiency-related vaccine-derived poliovirus (iVDPV)

Prolonged replication of VDPVs has been observed in a small number of people with rare immune deficiency disorders. Because they are not able to mount an immune response, these people are not able to clear the intestinal vaccine virus infection, which is usually cleared within six to eight weeks. They therefore excrete iVDPVs for
prolonged periods. The occurrence of iVDPVs is very rare. Only 111 cases have been documented worldwide since 1962. Of these, most stopped excretion within six months or died. 

**Ambiguous vaccine-derived poliovirus (aVDPV)**

aVDPVs are VDPVs that are either isolated from people with no known immunodeficiency, or isolated from sewage whose ultimate source is unknown. Very little is known about them.

**Implications and management of vaccine-derived polioviruses**

Circulating vaccine-derived polioviruses must be managed in the same way as wild poliovirus outbreaks. The solution is the same for all polio outbreaks: vaccinate every child several times with oral polio vaccine to stop polio transmission, regardless of whether the virus is wild or vaccine-derived. 

Vaccine-derived polioviruses appear to be less transmissible than wild poliovirus. Outbreaks are usually self-limiting or rapidly stopped with 2–3 rounds of high-quality supplementary immunization activities. Once wild poliovirus transmission has been stopped globally, the vaccine-viruses will be the only source of live polioviruses in the community and could potentially lead to the re-emergence of polio. Use of the oral polio vaccine in routine immunization programmes will therefore be phased out to eliminate the rare risks posed by vaccine-derived polioviruses. 

http://polioeradication.org/polio-today/polio-prevention/the-virus/vaccine-derived-polio-viruses/

cVDPVs were first recognized in 2000 during an outbreak in Hispaniola. The attenuated viruses in live OPV vaccines (Sabin viruses) may, through prolonged replication in an individual or in a community, re-acquire the neurovirulence and transmissibility characteristics of WPV. They may then become circulating vaccine-derived polioviruses that cause cases or outbreaks of paralytic poliomyelitis.

The behaviour of cVDPVs can be similar or identical to that of WPVs, with significant paralytic attack rates and sustained person-to-person transmission. They have lost the original attenuating mutations. Low vaccine coverage rates allowed CVDPV to get ahold in Hispaniola...and in other areas of the world. cVDPVs have the ability to become endemic (Nigeria, Egypt), and that VDPVs can be imported and spread in an under-vaccinated community in a developed country (Amish community, USA).

In 2012, 9 countries reported cases of paralytic poliomyelitis associated with cVDPVs, most of them Sabin 2. The largest numbers of such cases were reported in the Democratic Republic of the Congo (n=17) and Pakistan (n=16).
In 2013, 7 countries reported cases of paralytic poliomyelitis caused by cVDPV, all associated with Sabin 2, of which Pakistan reported the greatest number \( n=44 \). Cases of cVDPV also occur with type 1 and type 3.

VDPVs subdivided into 3 categories:
(1) circulating VDPVs (cVDPVs), when evidence of person-to-person transmission in the community exists;
(2) immunodeficiency-associated VDPVs (iVDPVs), which are isolated in rare cases from people with primary B-cell and combined immunodeficiencies (with defects in antibody production) who have prolonged VDPV infections (in individual cases excretion has been reported to persist for 10 years or more); and
(3) ambiguous VDPVs (aVDPVs), which are either clinical isolates from persons with no known immunodeficiency, or sewage isolates of unknown source.
Currently: Also Democratic Republic of the Congo (3 strains of serotype 2) and Papua New Guinea (serotype 1). 2017—Syria and Somalia Type 2 WPV eradicated in 2000 (last case 1999)
The eradication of WPV2 in 1999, coupled with the continuing problem of neurovirulent circulating type 2 vaccine-derived polioviruses (cVDPV2s) led to the recommendation that there should be coordinated global cessation of use of the type 2 component of OPV as soon as possible. [2016]
Monovalent OPV2 would then be used in the response to any type 2 outbreak after OPV2 cessation, e.g. as caused by an emergence of cVDPV2.

New case of AFP and vaccine-associated serotype 1 circulating in Papua New Guinea (April 2018)
Challenge: In 2017 VAP in Syria, Somalia, and Democratic Republic of the Congo Vaccination: PHIL 9408
TOPV→bOPV from WHO
All adults need a dose of IPV before international travel (in addition to MMR)
National immunization programmes currently use either or both of the 2 types of poliovirus vaccine, i.e. OPV or IPV.
IPV, first developed and licensed in 1955, is given by injection and is available only in trivalent form.
OPV was licensed in 1961 as a monovalent (mOPV) vaccine, followed by a trivalent version (tOPV) licensed for use in 1963.
Polio this week as of 10 July 2018
A Disease Outbreak News (DON) notification was issued on 10 July on the Democratic Republic of the Congo’s three concurrent circulating vaccine-derived poliovirus type 2 (cVDPV2) outbreaks.
Papua New Guinea prepares for the launch of large-scale immunization campaigns in Morobe, Madang and Eastern Highlands provinces, set to commence next week.
Summary of new cases this week: Last week’s advance notifications of one wild poliovirus type 1 (WPV1) case in Afghanistan and one cVDPV2 case in Somalia with a cVDPV2 positive community contact have been confirmed. Two new WPV1 positive environmental samples have been reported in Afghanistan, and four new WPV1 positive environmental samples have been reported in Pakistan. See country sections below for more details.
http://polioeradication.org/polio-today/polio-now/this-week/
Be fully immunized
Childhood—5 IPV, last ≥ 4 years old; Adult—3 doses; Adult immunized in childhood—Additional booster dose
% of Children With Personal/Religious Belief Vaccine Exemptions, AZ, 2000-18

% with Exemptions

- Child Care
- Kindergarten
- 6th/7th Grade

Health and Wellness for all Arizonans
Wonder Why My Parents Didn’t Give Me Salk Shots?

1957
Pulitzer Prize for
Editorial Cartooning
To Tom Little

- 20,000 cases paralytic polio in 1952