16 May 2012

**RISK OF INFECTION WEST AFRICA**

![Map of West Africa](image)

### RECENT OUTBREAKS OF INFECTIOUS DISEASES

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MODE OF TRANSMISSION, SIGNS AND SYMPTOMS OF SELECTED DISEASES

AVIAN INFLUENZA

Transmission and etiology

Avian influenza is flu infection in birds. The virus that causes the bird infection can change (mutate) to infect humans. Such mutation could start a deadly worldwide epidemic. Human cases of avian influenza A (H5N1) have been reported in Asia, Africa, Europe, Indonesia, Vietnam, the Pacific, and the near East. Hundreds of people have become sick with this virus. Slightly more than 60% of those who became ill have died.

Clinical signs and symptoms

Symptoms of avian flu infection in humans depend on the strain of virus. Infection with the H5N1 virus in humans causes typical flu-like symptoms, which might include: cough (dry or productive, diarrhea, difficulty breathing, fever greater than 100.4°F (38°C), headache, malaise, muscle aches, runny nose and sore throat.
Prevention

The Food and Drug Administration has approved one vaccine to prevent infection with one strain of H5N1 bird flu virus. This vaccine isn't available to the public, but the U.S. government is stockpiling it and will distribute it in the event of an outbreak. It's intended to help protect adults ages 18 to 64 and could be used early in such an outbreak to provide limited protection until another vaccine, designed to protect against the specific form of the virus causing the outbreak, is developed and produced.

Recommendations for travelers

If you're traveling to Southeast Asia or to any region with bird flu outbreaks, consider these public health recommendations:

Avoid domesticated birds. If possible, avoid rural areas, small farms and open-air markets.

Wash your hands. This is one of the simplest and best ways to prevent infections of all kinds. Use an alcohol-based hand sanitizer containing at least 60 percent alcohol when you travel.

Ask about a flu shot. Before traveling, ask your doctor about a flu shot. It won't protect you specifically from bird flu, but it may help reduce the risk of simultaneous infection with bird and human flu viruses.

Avoid cross-contamination. Use hot, soapy water to wash cutting boards, utensils and all surfaces that have come into contact with raw poultry.

Cook thoroughly. Cook chicken until the juices run clear, and it reaches a minimum internal temperature of 165 F (74 C).

Steer clear of raw eggs. Because eggshells are often contaminated with bird droppings, avoid foods containing raw or undercooked eggs.

CHOLERA

Transmission and etiology

Cholera is an infection in the small intestine caused by the bacterium *Vibrio cholerae*. Cholera is typically transmitted by either contaminated food or water. In the developed world, seafood is the usual cause, while in the developing world it is more often water. Cholera has been found in only two other animal populations: shellfish and plankton. People infected with cholera often have diarrhea, and if this highly liquid stool, colloquially referred to as "rice-water" or "faucet butt", contaminates water used by others, disease transmission may occur. The source of the contamination is typically other cholera sufferers when their untreated diarrheal discharge is allowed to get into waterways, groundwater or drinking water supplies. Drinking any infected water and eating any foods washed in the water, as well as shellfish living
in the affected waterway, can cause a person to contract an infection. Cholera is rarely spread directly from person to person. Both toxic and nontoxic strains exist. Nontoxic strains can acquire toxicity through a temperate bacteriophage.

Coastal cholera outbreaks typically follow zooplankton blooms, thus making cholera a zoonotic disease.

Clinical signs and symptoms

For every symptomatic person, 3 to 100 people get the infection but remain asymptomatic. The primary symptoms of cholera are profuse, painless diarrhea and vomiting of clear fluid. These symptoms usually start suddenly, one to five days after ingestion of the bacteria. The diarrhea is frequently described as "rice water" may have a fishy odor. An untreated person with cholera may produce 10–20 liters of diarrhea a day with fatal results. Cholera has been nicknamed the "blue death" due to a patient's skin turning a bluish-gray hue from extreme loss of fluids. If the severe diarrhea is not treated with intravenous rehydration, it can result in life-threatening dehydration and electrolyte imbalances. The typical symptoms of dehydration include low blood pressure, poor skin turgor (wrinkled hands), sunken eyes, and a rapid pulse.

Prevention

Although cholera may be life-threatening, prevention of the disease is normally straightforward if proper sanitation practices are followed.

CRIMEAN CONGO HEMORRHAGIC FEVER (CCHF)

Transmission and etiology

Crimean-Congo Hemorrhagic fever is a viral hemorrhagic fever transmitted by ticks. Clinical disease is rare in infected mammals, but commonly severe in infected humans, with a 30% mortality rate. Sporadic infection of people is usually caused by Hyalomma tick bite. Ticks carry the virus to domestic animal stock. Sheep, goats and cattle develop high titers of virus in blood, but tend not to fall ill. Birds are generally resistant with the exception of ostriches. Clusters of illness typically appear after people treat, butcher or eat infected livestock, particularly ruminants and ostriches. Outbreaks of illness are usually attributable to handling infected animals.

Hyalomma tick
Clinical signs and symptoms

Typically, after a 1–3 day incubation period following a tick bite (5–6 days after exposure to infected blood or tissues), flu-like symptoms appear, which may resolve after one week. In up to 75% of cases, however, signs of hemorrhage appear within 3–5 days of the onset of illness in case of bad containment of the first symptoms: first mood instability, agitation, mental confusion and throat petechiae, then soon nosebleeds, bloody urine and vomiting, and black stools. The liver becomes swollen and painful. Disseminated intravascular coagulation may occur as well as acute kidney failure and shock, and sometimes acute respiratory distress syndrome. Patients usually begin to show signs of recovery after 9–10 days from when the symptoms appear, however 30% of the cases result in death on the second week of the illness. Patients usually begin to show signs of recovery after 9–10 days from when the symptoms appear, however 30% of the cases result in death on the second week of the illness.

Prevention

Crimean-Congo Hemorrhagic Fever (CCHF) can be prevented by avoiding tick bites. Adequate precaution should be taken when coming in contact with infected patients or animals. People exposed to domestic animals or those undergoing activities like hiking should wear protective gear in situations that could result in tick bites. These include using permethrin-impregnated clothing, wearing high boots with trousers tucked in, wearing light-colored clothes to identify ticks easily and using insect repellants like DEET on exposed skin. The skin should be regularly inspected for ticks.

DENGUE HEMORRHAGIC FEVER

Transmission and etiology

Dengue hemorrhagic fever is endemic in more than 110 countries. Dengue virus is primarily transmitted by Aedes mosquitoes, particularly A. aegypti. These mosquitoes usually live between the latitudes of 35° North and 35° South below an elevation of 1,000 metres (3,300 ft). They bite primarily during the day. Other Aedes species that transmit the disease include A. albopictus, A. polynesiensis and A. scutellaris. Humans are the primary host of the virus, but it also circulates in nonhuman primates. An infection can be acquired via a single bite. A female mosquito that takes a blood meal from a person infected with dengue
fever becomes itself infected with the virus in the cells lining its gut. About 8–10 days later, the virus spreads to other tissues including the mosquito's salivary glands and is subsequently released into its saliva. The virus seems to have no detrimental effect on the mosquito, which remains infected for life. *Aedes aegypti* prefers to lay its eggs in artificial water containers, to live in close proximity to humans, and to feed off people rather than other vertebrates. Dengue can also be transmitted via infected blood products and through organ donation. In countries such as Singapore, where dengue is endemic, the risk is estimated to be between 1.6 and 6 per 10,000 transfusions. Vertical transmission (from mother to child) during pregnancy or at birth has been reported. Other person-to-person modes of transmission have also been reported, but are very unusual.

**Clinical signs and symptoms**

Typically, people infected with dengue virus are asymptomatic (80%) or only have mild symptoms such as an uncomplicated fever. Others have more severe illness (5%), and in a small proportion it is life-threatening. The incubation period (time between exposure and onset of symptoms) ranges from 3–14 days, but most often it is 4–7 days. Therefore, travelers returning from endemic areas are unlikely to have dengue if fever or other symptoms start more than 14 days after arriving home. Children often experience symptoms similar to those of the common cold and gastroenteritis (vomiting and diarrhea), and generally have less severe symptoms than adults, but are more susceptible to the severe complications. The characteristic **symptoms of dengue are sudden-onset fever, headache (typically located behind the eyes), muscle and joint pains, and a rash.** The alternative name for dengue, "break-bone fever", comes from the associated muscle and joint pains. The course of infection is divided into three phases: febrile, critical, and recovery. The febrile phase involves high fever, often over 40 °C (104 °F), and is associated with generalized pain and a headache; this usually lasts two to seven days. At this stage, a rash occurs in 50–80% of those with symptoms. It occurs in the first or second day of symptoms as flushed skin, or later in the course of illness (days 4–7), as a measles-like rash. Some petechiae (small red spots that do not disappear when the skin is pressed, which are caused by broken capillaries) can appear at this point, as may some mild bleeding from the mucous membranes of the mouth and nose. The fever itself is classically biphasic in nature, breaking and then returning for one or In some people, the disease proceeds to a critical phase, which follows the resolution of the high fever and typically lasts one to two days. During this phase there may be significant fluid accumulation in the chest and abdominal cavity due to increased capillary permeability and leakage. This leads to depletion of fluid from the circulation and decreased blood supply to vital organs. During this phase, organ dysfunction and severe bleeding, typically from the gastrointestinal tract, may occur. Shock (dengue shock syndrome) and hemorrhage (dengue hemorrhagic fever) occur in less than 5% of all cases of dengue; however those who have previously been infected with other serotypes of dengue virus ("secondary infection") are at an increased risk. The recovery phase occurs next, with resorption of the leaked fluid into the bloodstream. This usually lasts two to three days. The improvement is often striking, but there may be severe itching and a slow heart rate. During recovery, a fluid overload may occur; if it affects the brain, it may cause a reduced level of consciousness or seizures. Dengue can occasionally affect several other body systems, either in isolation or along with the classic dengue symptoms. A decreased level of consciousness occurs in 0.5–6% of severe cases, which is attributable either to infection of the brain by the virus or indirectly as a result of impairment of vital organs, for example, the liver. Other neurological disorders have been reported in the context of dengue, such as transverse myelitis and Guillain-Barré syndrome, infection of the heart and acute liver failure. There are no approved vaccines for the dengue virus.

**Prevention**

Prevention thus depends on control of and protection from the bites of the mosquito that transmit virus.
DESYNTERY

Transmission and etiology

Dysentery results from viral, bacterial, or protozoan infections or parasitic infestations. These pathogens typically reach the large intestine after entering orally, through ingestion of contaminated food or water, oral contact with contaminated objects or hands, stomach pains and frequent passage of feces. Symptoms normally present themselves after one to three days and are usually no longer present after a week. The frequency of urges to defecate, the volume of feces passed, and the presence of mucus, pus and blood depend on the pathogen that is causing the disease.

Dysentery in the modern world is most likely to affect people in the less developed countries and travelers who visit these areas. In addition to the characteristic bloody and/or watery diarrhea and abdominal cramps of dysentery, the various types have somewhat different symptom profiles:

Clinical signs and symptoms

Bacillary dysentery. The symptoms of shigellosis may range from the classical bloody diarrhea and tenesmus (feeling of constantly needing to pass stools) characteristic of dysentery to the passage of nonbloody diarrhea that resembles the loose stools caused by other intestinal disorders. The high fever associated with shigellosis begins within one to three days after exposure to the organism. The patient may also have pain in the rectum as well as abdominal cramping. The acute symptoms last for three to seven days, occasionally for as long as a month. Bacillary dysentery may lead to two potentially fatal complications outside the digestive tract: bacteremia (bacteria in the bloodstream), which is most likely to occur in malnourished children; and hemolytic uremic syndrome, a type of kidney failure that has a mortality rate above 50 percent.

Amebic dysentery. Amebic dysentery often has a slow and gradual onset; most patients with amebiasis visit the doctor after several weeks of diarrhea and bloody stools. Fever is unusual with amebiasis unless the patient has developed a liver abscess as a complication of the infection. The most serious complication of amebic dysentery, however, is fulminant or necrotizing colitis, which is a severe inflammation of the colon characterized by dehydration, severe abdominal pain, and the risk of perforation (rupture) of the colon.

Dysentery caused by other protozoa. Dysentery associated with giardiasis begins about 1-3 weeks after infection with the organism. It is characterized by bloating and foul-smelling flatus, nausea and vomiting, headaches, and low-grade fever. These acute symptoms usually last for three or four days. The symptoms of cryptosporidiosis are mild in most patients but are typically severe in patients with AIDS. Diarrhea usually starts between seven and 10 days after exposure to the organism and may be copious. The patient may have pain in the upper right abdomen, nausea, and vomiting, but fever is unusual.

Viral dysentery. Viral dysentery has a relatively rapid onset; symptoms may begin within hours of infection. The patient may be severely dehydrated from the diarrhea but usually has only a low-grade fever. The diarrhea itself may be preceded by one to three days of nausea and vomiting. The patient's abdomen may be slightly tender but is not usually severely painful.
Dysentery caused by parasitic worms. Patients with intestinal schistosomiasis typically have a gradual onset of symptoms. In addition to bloody diarrhea and abdominal pain, these patients usually have fatigue. An examination of the patient's colon will usually reveal areas of ulcerated tissue, which is the source of the bloody diarrhea.

MENINGOCOCCAL DISEASE

Transmission and etiology

Meningococcal disease is potentially fatal and should always be viewed as a medical emergency. Meningococcal meningitis is a bacterial form of meningitis, a serious infection of the thin lining that surrounds the brain and spinal cord. The bacteria are transmitted from person-to-person through droplets of respiratory or throat secretions from carriers. Close and prolonged contact – such as kissing, sneezing or coughing on someone, or living in close quarters (such as a dormitory), sharing eating or drinking utensils) with an infected person (a carrier), facilitates the spread of the disease.

Clinical signs and symptoms

The most common symptoms are a stiff neck, high fever, sensitivity to light, confusion, headaches and vomiting. Even when the disease is diagnosed early and adequate treatment is started, 5% to 10% of patients die, typically within 24 to 48 hours after the onset of symptoms. Bacterial meningitis may result in brain damage, hearing loss or a learning disability in 10% to 20% of survivors. A less common but even more severe (often fatal) form of meningococcal disease is meningococcal septicemia, which is characterized by a hemorrhagic rash and rapid circulatory collapse.

Prevention

Vaccination

There is a vaccine for the bacteria that causes meningococcal disease. However, available vaccines do not cover all serogroups (“strains”) of Neisseria meningitidis bacteria. Like with any vaccine, meningococcal vaccines are not 100% effective. This means that even if you have been vaccinated, there is still a chance you can develop a meningococcal infection. People should know the symptoms of meningococcal meningitis and meningococcal septicemia since early recognition and quick medical attention are extremely important.

Antibiotics

Sometimes Neisseria meningitidis bacteria spread to other people who have had close or lengthy contact with a patient with meningococcal disease. People in the same household, roommates, or anyone with direct contact with a patient's oral secretions (saliva) (such as a boyfriend or girlfriend) would be considered at increased risk of getting the infection. People who qualify as close contacts of a person with meningococcal disease should receive antibiotics to prevent them from getting the disease.
Previous infection
Protection from previous infection does not last a lifetime and is not perfect. Therefore, routine meningococcal vaccines are still recommended. If you get meningococcal disease twice, it is highly possible that you have an underlying immune deficiency, which your doctor should evaluate.

LASSA FEVER

Transmission and etiology

Lassa fever is an acute viral hemorrhagic illness caused by Lassa virus. The disease is endemic in the rodent population in parts of West Africa. Person-to-person infections and laboratory transmission can also occur, particularly in the hospital environment in the absence of adequate infection control measures. Diagnosis and prompt treatment are essential. The infection is endemic in West African countries, and causes 300,000–500,000 cases annually, with approximately 5,000 deaths. The primary animal host of the Lassa virus is the Natal Multimammate Mouse (*Mastomys natalensis*), an animal indigenous to most of Sub-Saharan Africa. The virus is probably transmitted by contact with the feces or urine of animals accessing grain stores in residences. Infection in humans typically occurs via exposure to animal excrement through the respiratory or gastrointestinal tracts. Inhalation of tiny particles of infective material (aerosol) is believed to be the most significant means of exposure. It is possible to acquire the infection through broken skin or mucous membranes that are directly exposed to infective material. Transmission from person to person has also been established, presenting a disease risk for healthcare workers.

Lassa fever can be also transmitted directly from one human to another. It can be contracted by an airborne route or with direct contact with infected human blood, urine, or semen. Transmission through breast milk has also been observed.

![Natal Multimammate Mouse](image)

Clinical signs and symptoms

In 80% of cases the disease is unapparent, but in the remaining 20% it takes a complicated course. It is estimated that the virus is responsible for about 5,000 deaths annually. The fever accounts for up to one third of deaths in hospitals within the affected regions and 10 to 16% of total cases. After an incubation
period of six to twenty-one days, an acute illness with multiorgan involvement develops. Non-specific symptoms include fever, facial swelling, and muscle fatigue, as well as conjunctivitis and mucosal bleeding. The other symptoms arising from the affected organs are: gastrointestinal tract (nausea), vomiting, bloody diarrhea, and constipation cardiovascular system (hyper- or hypotension, tachycardia), respiratory tract (cough, chest pain), nervous system (meningitis, seizures, uni- or bilateral hearing loss). About 15%-20% of hospitalized Lassa fever patients will die from the illness. It is estimated that the overall mortality rate is 1%, however during epidemics mortality can climb as high as 50%. The mortality rate is greater than 80% when it occurs in pregnant women during their third trimester; fetal death also occurs in nearly all those cases, unilateral or bilateral hearing deficit). When Lassa fever infects pregnant women late in their third trimester, it is necessary to induce delivery for the mother to have a good chance of survival. This is because the virus has an affinity for the placenta and other highly vascular tissues. The fetus has only a one in ten chance of survival no matter what course of action is taken; hence focus is always on saving the life of the mother. Abortion decreases the risk of death to the mother

Prevention

Avoid contact with rats, especially rat urine and feces.

• Put food away in rodent-proof containers.

• Keep the home clean and Products rodent proof.

• Trap rats in around home.

If found suspected rodents droppings, newer brush surface. Wear gloves and surgical mask, wet surface with chlorax solution (3 Tbsp. of Clorox® Regular-Bleach per gallon of water), soak surface for 3 min, and wipe contamination. Place used paper towels in airtight container before disposing

YELLOW FEVER

Transmission and etiology

Mosquitoes carry the virus from monkey or person to person. Yellow fever causes epidemics that can affect 20% of the population. When epidemics occur in unvaccinated populations, case-fatality rates may exceed 50%.

Clinical signs and symptoms

Yellow fever begins after an incubation period of three to six days. Most cases only cause a mild infection with fever, headache, chills, back pain, loss of appetite, nausea, and vomiting. In these cases the infection lasts only three to four days. In fifteen percent of cases, however, sufferers enter a second, toxic phase of the disease with recurring fever, this time accompanied by a jaundice due to liver damage, as well as abdominal pain. Bleeding in the mouth, the eyes and in the gastrointestinal tract will cause vomitus containing blood (giving the name black vomit). Most patients improve and their symptoms disappear after 3 to 4 days. However, 15% of patients enter a second, more toxic phase within 24 hours of the initial remission. High fever returns and several body systems are affected. The patient rapidly develops jaundice and complains of abdominal pain with vomiting. Bleeding can occur from the mouth, nose, eyes or stomach. Once this happens, blood appears in the vomit and feces. Kidney function
deteriorates. Half of the patients who enter the toxic phase die within 10 to 14 days, the rest recover without significant organ damage. Surviving the infection causes life-long immunity and normally there is no permanent organ damage.

Prevention

For journeys into affected areas, vaccination is highly recommended since mostly non-native people are affected by severe cases of yellow fever and lasts for at least 10 years (even 30 years later, 81% of patients retained the immunity). In about 20% of all cases, mild, flu-like symptoms may develop. In rare cases (less than one in 200,000 to 300,000), Vaccination can cause YEL-AVD (yellow fever vaccine-associated viscerotropic disease), which is fatal in 60% of all cases. YEL-AVD is due to a genetic defect in the immune system. No treatment beyond supportive care exists.

POLIOMYELITIS (POLIO)

Transmission and etiology

Poliomyelitis often called polio or infantile paralysis is an acute, viral, infectious disease spread from person to person, primarily via the fecal-oral route. Although approximately 90% of polio infections cause no symptoms at all, affected individuals can exhibit a range of symptoms if the virus enters the blood stream. In about 1% of cases, the virus enters the central nervous system, preferentially infecting and destroying motor neurons, leading to muscle weakness and acute flaccid paralysis. Different types of paralysis may occur, depending on the nerves involved. Spinal polio is the most common form, characterized by asymmetric paralysis that most often involves the legs. Bulbar polio leads to weakness of muscles innervated by cranial nerves. Bulbospinal polio is a combination of bulbar and spinal paralysis. Two basic patterns of polio infection are described: a minor illness which does not involve the central nervous system (CNS), sometimes called abortive poliomyelitis, and a major illness involving the CNS, which may be paralytic or nonparalytic. In most people with a normal immune system, a poliovirus infection is asymptomatic. Rarely, the infection produces minor symptoms; these may include upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely, diarrhea), and influenza-like illness. The virus enters the central nervous system in about 3% of infections. Most patients with CNS involvement develop nonparalytic aseptic meningitis, with symptoms of headache, neck, back, abdominal and extremity pain, fever, vomiting, lethargy and irritability. About one to five in 1000 cases progress to paralytic disease, in which the muscles become weak, floppy and poorly controlled, and finally completely paralyzed; this condition is known as acute flaccid paralysis. Depending on the site of paralysis, paralytic poliomyelitis is classified as spinal, bulbar, or bulbospinal. Encephalitis, an infection of the brain tissue itself, can occur in rare cases, and is usually restricted to infants. It is characterized by confusion, changes in mental status, headaches, fever, and less commonly, seizures and spastic paralysis.

Because Poliomyelitis is highly contagious via the oral-oral (oropharyngeal source) and fecal-oral (intestinal source) routes, in endemic areas, wild polioviruses can infect virtually the entire human population. Poliomyelitis is seasonal in temperate climates, with peak transmission occurring in summer and autumn. These seasonal differences are far less pronounced in tropical areas.

The time between first exposure and first symptoms, known as the incubation period, is usually six to 20 days, with a maximum range of three to 35 days. Virus particles are excreted in the feces for several weeks following initial infection. Factors that increase the risk of polio infection or affect the severity of the disease include immune deficiency, malnutrition, tonsillectomy, physical activity immediately following the onset of paralysis, skeletal muscle injury due to injection of vaccines or therapeutic agents,
and pregnancy. Although the virus can cross the placenta during pregnancy, the fetus does not appear to be affected by either maternal infection or polio vaccination. Maternal antibodies also cross the placenta, providing passive immunity that protects the infant from polio infection during the first few months of life. As a precaution against infection, public swimming pools were often closed in affected areas during poliomyelitis epidemics.

**Clinical signs and symptoms**

Early symptoms of paralytic polio include **high fever, headache, stiffness in the back and neck, asymmetrical weakness of various muscles, sensitivity to touch, difficulty swallowing, muscle pain, loss of superficial and deep reflexes, paresthesia (pins and needles), irritability, constipation, or difficulty urinating.** Paralysis generally develops one to ten days after early symptoms begin, progresses for two to three days, and is usually complete by the time the fever breaks. The likelihood of developing paralytic polio increases with age, as does the extent of paralysis. In children, nonparalytic meningitis is the most likely consequence of CNS involvement, and paralysis occurs in only one in 1000 cases. In adults, paralysis occurs in one in 75 cases. In children under five years of age, paralysis of one leg is most common; in adults, extensive paralysis of the chest and abdomen also affecting all four limbs (quadriplegia) is more likely. Paralysis rates also vary depending on the serotype of the infecting poliovirus; the highest rates of paralysis (one in 200) are associated with poliovirus type 1, the lowest rates (one in 2).

**Spinal polio**, the most common form of paralytic poliomyelitis, results from viral invasion of the motor neurons of the anterior horn cells, or the ventral (front) gray matter section in the spinal column, which are responsible for movement of the muscles, including those of the trunk, limbs and the intercostal muscles. Virus invasion causes inflammation of the nerve cells, leading to damage or destruction of motor neuron ganglia. When spinal neurons die, Wallerian degeneration takes place, leading to weakness of those muscles formerly innervated by the now-dead neurons. With the destruction of nerve cells, the muscles no longer receive signals from the brain or spinal cord; without nerve stimulation, the muscles atrophy, becoming weak, floppy and poorly controlled, and finally completely paralyzed. Progression to maximum paralysis is rapid (two to four days), and is usually associated with fever and muscle pain. Deep tendon reflexes are also affected, and are usually absent or diminished; sensation (the ability to feel) in the paralyzed limbs, however, is not affected.

The extent of spinal paralysis depends on the region of the cord affected, which may be cervical, thoracic, or lumbar. The virus may affect muscles on both sides of the body, but more often the paralysis is asymmetrical. Any limb or combination of limbs may be affected—one leg, one arm, or both legs and both arms. Paralysis is often more severe proximally (where the limb joins the body) than distally (the fingertips and toes).

Making up about 2% of cases of paralytic polio, **bulbar polio** occurs when poliovirus invades and destroys nerves within the bulbar region of the brain stem. The destruction of these nerves weakens the muscles supplied by the cranial nerves, producing symptoms of encephalitis, and causes difficulty breathing, speaking and swallowing. Critical nerves affected are the glossopharyngeal nerve, which partially controls swallowing and functions in the throat, tongue movement and taste; the vagus nerve, which sends signals to the heart, intestines, and lungs; and the accessory nerve, which controls upper neck movement. Due to the effect on swallowing, **secretions of mucus may build up in the airway, causing suffocation.** Other signs and symptoms include facial weakness, caused by destruction of the trigeminal nerve and facial nerve, which innervate the cheeks, tear ducts, gums, and muscles of the face, among
other structures; **double vision; difficulty in chewing; and abnormal respiratory rate, depth, and rhythm**, which may lead to **respiratory arrest**. **Pulmonary edema and shock** are also possible, and may be fatal.

Approximately 19% of all paralytic polio cases have both bulbar and spinal symptoms; this subtype is called respiratory or **bulbospinal polio**. Here, the virus affects the upper part of the cervical spinal cord (cervical vertebrae C3 through C5), and paralysis of the diaphragm occurs. The critical nerves affected are the phrenic nerve, which drives the diaphragm to inflate the lungs, and those that drive the muscles needed for swallowing. By destroying these nerves, this form of polio affects breathing, making it difficult or impossible for the patient to breathe without the support of a ventilator. It can lead to paralysis of the arms and legs and may also affect swallowing and heart functions.

**Prevention**

The, live, oral polio vaccine (OPV) and inactivated poliovirus vaccine (IPV) are available. Because OPV is inexpensive, easy to administer, and produces excellent immunity in the intestine (which helps prevent infection with wild virus in areas where it is endemic), it has been the vaccine of choice for controlling poliomyelitis in many countries. On very rare occasions (about one case per 750,000 vaccine recipients), the attenuated virus in OPV reverts into a form that can paralyze. Most industrialized countries have switched to IPV, which cannot revert, either as the sole vaccine against poliomyelitis or in combination with oral polio vaccine. Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on poliovirus grown in a type of monkey kidney tissue culture (vero cell line), which is chemically inactivated with formalin. After two doses of IPV (given by injection), 90% or more of individuals develop protective antibody to all three serotypes of poliovirus, and at least 99% are immune to poliovirus following three doses.

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