Vaccine preventable diseases

Vaccines have been frequently cited as one of the most equitable low-cost, high-impact public health measures, saving millions of lives annually when programs are implemented on the national level. Over the last 40 years, the use of smallpox, measles, diphtheria, tetanus, pertussis, and poliomyelitis vaccines have eradicated smallpox and eliminated disease in those populations that have achieved and sustained programs with high implementation rates.

The eradication of smallpox was an outstanding display of concerted global action in a war against microbial invaders. The progress in expanding poliomyelitis and measles vaccination efforts and their elimination from many regions further demonstrates that vaccines are among the most powerful public health tools.

Polio

Poliovirus is most often transmitted fecal-orally among persons living in unsanitary and crowded conditions. Acute infections are caused by any one of three serotypes of poliovirus that initially replicate in the gastrointestinal tract. Exposure to poliovirus predominantly results in asymptomatic infections. It has been estimated that 24 percent of infections result in minor illness characterized by a few days of varying symptoms, including fever, malaise, drowsiness, headache, nausea, vomiting, constipation, and sore throat). In fewer cases (4 percent), infection leads to nonparalytic polio or aseptic meningitis, which manifests as fever, vomiting, malaise, and sore throat; meningeal irritation occurs one to two days later, characterized by soreness and stiffness of the neck, back, limbs, and severe headache. These symptoms can last up to 10 days, but recovery is usually rapid and complete. Paralytic polio, which affects less than 1 percent of those infected, is the most serious manifestation of the disease. This form of the disease presents initially as a minor fever with rapid progression to paralysis within a matter of days. Paralysis may affect the major muscles involved in respiration and therefore cause death if there is no appropriate rapid intervention.

Global efforts toward polio eradication have included vaccination campaigns and active surveillance. The annual incidence of paralytic polio was reduced from an estimated 350,000 in 1988 to about 1,000 from 2001 to 2004 (WHO 2004). Africa and South Asia are the last regions in the world where poliomyelitis is still endemic.

Paralytic poliomyelitis has been completely controlled in the United States and other developed countries through routine childhood immunization with either inactivated poliovirus vaccine (IPV), live-attenuated poliovirus vaccine (OPV), or both. However, as of 2014, the goal of worldwide poliomyelitis eradication has not yet been achieved, having been hampered by unforeseen scientific challenges, geopolitical unrest, and social disruption, among other factors.

Risk factors

Several preexisting factors and provocative events are known to influence the risk of poliovirus infection and, once infected with poliovirus, the risk of developing paralysis. Before puberty, poliovirus infections occur equally in boys and girls, although paralysis is more common in boys. Among adults, women are at greater risk of infection but are not necessarily at greater risk of paralysis. Both the incidence and severity of poliomyelitis may be increased in pregnant women. Not only are women of childbearing age more likely to be exposed to infections in young children, but also, late pregnancy may be associated with increased susceptibility to more serious illness. Strenuous exercise increases both incidence and severity of paralytic poliomyelitis. Exercise during the minor illness or the prodrome has no effect but is detrimental when it occurs during the first 3 days of the major illness. Both epidemiologic and experimental studies have confirmed that poliomyelitis tends to localize in a limb that has been the site of an intramuscular injection or injury within 2 to 4 weeks before the onset of infection. Provocation poliomyelitis has been observed with both wild-type and OPV viruses. Skeletal muscle injury in viremic PVR-expressing transgenic mice induces retrograde axonal poliovirus transport to the CNS.
Tonsillectomy increases the risk of bulbar poliomyelitis whether the procedure is performed just before poliovirus infection or in the remote past.Because the ninth and tenth cranial nerves supply the fauces, the spread of virus from damaged nerve endings may explain this effect.

Prognosis
Muscle paralysis usually progresses or extends for only 1 to 3 days after its onset but occasionally for as long as 1 week. Permanent weakness is observed in approximately two thirds of patients with paralytic poliomyelitis. Complete recovery is less likely when acute paralysis is severe and when patients require mechanical ventilation. An estimate of the eventual outcome can be made after 1 month, when most reversible damage has disappeared. Very little additional return of function can be expected beyond 9 months. Recovery from pharyngeal paralysis usually is evident by 10 days and is eventually complete. Bulbar poliomyelitis is rarely responsible for permanent sequelae in surviving patients. Available mortality figures date from the era of epidemic poliomyelitis, a period when critical care medicine was less advanced than it is today. The reported overall mortality for acute paralytic poliomyelitis during this period was approximately 5% to 10%.Mortality rates are higher among older individuals who are more likely to have combined spinal respiratory muscle and bulbar involvement.

Post-polio syndrome

Some patients who partially or fully recover from paralytic poliomyelitis experience a new onset of muscle weakness, pain, atrophy, and fatigue many years after the acute illness. Typically, the involved muscles are the same as those affected during the original illness, but weakness may also occur in previously unaffected limb muscles. Progression of new symptoms is gradual, and affected persons are seldom severely disabled. However, it may be dangerous in those with respiratory dysfunction or dysphagia. Population-based studies suggest that the syndrome affects 20% to 85% of previously paralyzed patients. The mean interval between acute poliomyelitis and the onset of postpoliomyelitis syndrome is 36 years. Although the cause is unknown, the leading theory is that late progression of muscle weakness is a result of physiologic attrition of motor units innervating muscles and muscle groups already less innervated as a result of earlier acute poliomyelitis.

Vaccine-Associated Paralytic Poliomyelitis

The only adverse reaction associated with OPV is the rare occurrence of VAPP, which affects approximately 1 person per 2.6 million OPV doses distributed. The WHO has estimated the incidence of VAPP to be 4 cases per 1,000,000 birth cohort per year in countries using OPV. Approximately 50% of VAPP cases are recent OPV vaccinees, most of whom develop paralysis 7 to 21 days after the first feeding of OPV.A similar number of VAPP cases occurs among parents, other family members, babysitters, or other household contacts who develop paralysis 20 to 29 days after the administration of OPV to a close contact. For immunocompetent patients, the clinical features and outcome of VAPP

differ little from disease caused by naturally occurring polioviruses. More than 80% of recipient and contact cases are associated with the first dose of OPV. OPV virus types 3 and 2 are more common causes of VAPP than type 1. Approximately 25% of reported VAPP cases occur in children and adults who are immune-deficient. Most of these patients have transient or hereditary B-cell immunodeficiency, severe combined immunodeficiency syndrome, or common variable immunodeficiency. The risk of VAPP in newborn infants with a congenital B-cell immunodeficiency disorder is approximately 2000-fold higher than for immunocompetent infants. However, there is little evidence that immunodeficiency states that predominantly affect T-cell function, rather than B-cell function, increase the risk of VAPP, and there is little evidence that HIV infection, hematopoietic malignancy, or solid-organ transplantation increases the risk of VAPP. Certain clinical features distinguish VAPP in B-cell immunodeficient patients. The interval between the last OPV dose and onset of neurologic disease is unusually long, with a typical range of 1 to 8 months, but it has been documented to be as long as 12 years.The illness is protracted and characterized by chronic meningitis; progressive neurologic dysfunction, suggesting involvement of both upper and lower motor neurons; and progression of paralysis over several weeks. Immunodeficient patients also have a much higher risk of dying from VAPP than immunocompetent patients. Although fewer than 20% of surviving VAPP patients excrete polioviruses for longer than 6 months, fecal excretion of virus was estimated to occur for as long as 19 years in one immunodeficient patient. Most VAPP cases in immunocompromised children and adults have been associated with type 2 OPV virus.

The mechanism whereby the OPV viruses cause rare cases of paralytic disease is not completely understood. It is well known that OPV virus readily undergoes mutation during the brief period of intestinal replication and that isolates can be recovered that are neurovirulent for primates. Most OPV recipients shed polioviruses that have reverted to the naturally occurring genotype in an analogous stem-loop structure in the 5′ noncoding region of the genome affecting initiation of transcription, which is strongly associated with attenuation for each of the three OPV serotypes. Additional mutational events, mostly within the protein coding region, probably contribute to reversion to full neurovirulence.

Polio Eradication

The WHO Global Poliomyelitis Eradication Initiative uses several major strategies to control and ultimately eradicate polio from most regions of the world, including encouragement of routine childhood immunization, supplementary immunization activities (SIAs), improvement of laboratory capabilities, intensified surveillance, and rapid response to identified outbreaks. SIAs are highly coordinated nationwide or region-wide events in which all persons within a targeted age group receive two doses of OPV given within a short interval. Seroconversion rates during these mass campaigns are higher than for routine immunization, possibly because of the spread of OPV virus or because they are conducted during the dry season, when diarrheal disease is less prevalent.

As of mid-May 2014, paralytic polio cases continue to be reported from three nations where polio is considered to be endemic (Pakistan, Afghanistan, Nigeria) and another five countries previously free of polio (Equatorial Guinea, Iraq, Cameroon, Syria, Ethiopia).The greatest challenges to global eradication remain in those countries where polio remains endemic and which contribute more than 97% of the world's cases. In April 2013, the WHO introduced the Polio Eradication and Endgame Strategic Plan 2013-2018, a 6-year plan for the global eradication of polio.

Pertussis

Pertussis, or whooping cough, is a highly contagious disease caused by the bacterium *Bordetella pertussis,* which is transmitted through respiratory excretions. Pertussis is characterized as spasms (paroxysms) of coughing followed by inspiratory "whooping." The paroxysms can vary in length and severity but may become so severe, especially among infants, that respiration is compromised,

resulting in hypoxia. In some cases this can cause neurological damage. Pneumonia can also be a complication of pertussis infection. Severe coughing in older persons can cause serious complications, ranging from rib fractures to pneumo-thorax, inguinal hernia, and herniated lumbar disks.

In more developed countries transmission from adults to young infants is common. Girls tend to have higher incidence rates of the disease than boys. Pertussis is highly contagious in its early stages and has a secondary attack rate in other household members as high as 90 percent.

Each year there are an estimated 20 million to 40 million cases of pertussis and another 200,000 to 400,000 deaths attributed to the disease, 90 percent of which occur in the developing world. The WHO believes that only 1 to 2 percent of cases are reported worldwide. Pertussis diagnosis is difficult for several reasons. Paroxysms among adults are less severe and often misdiagnosed as other respiratory illnesses. Misdiagnosis is common in areas without adequately trained personnel or technology. Rapid diagnosis of pertussis using PCR techniques together with serological assays can enhance diagnosis as well as surveillance of pertussis. Clinical diagnosis of pertussis by a trained physician has also proved to be a reliable diagnostic tool and is often characterized by a cough that lasts at least 14 days. These are important implications when considering diagnosis of pertussis in remote areas with limited laboratory resources and few trained health professionals.

The incidence rate of pertussis has declined drastically over the past half-century primarily because of the administration of the inactivated whole-cell pertussis vaccines. Due to neurological reactions associated with the whole-cell vaccines, new acellular vaccines have been developed. Either of these vaccines is usually administered with the diphtheria and tetanus toxoids (TTs). The whole-cell vaccine is cheaper than the acellular vaccine and is produced in many developing countries. A herd effect of vaccination not only protects immunized infants but decreases transmission rates to protect unvaccinated infants.

Bordetella pertussis is the pathogen that causes whooping cough or pertussis. *B. pertussis* strictly affects humans and has no known animal reservoir.
Despite vaccination, pertussis disease continues to be a problem in the developing and developed world. According to the World Health Organization (WHO), an estimated 16 million cases and 195,000 deaths occurred in 2008 because of *B. pertussis*. Casefatality rates in developing countries may be as high as 3% in infants. WHO recommended that a pertussis incidence of less than 1 case/100,000 population be achieved in Europe by 2000. Data from countries represented in the Global Pertussis Initiative (GPI) have indicated that this goal has not yet been achieved.
In the prevaccine era, pertussis was a major childhood illness and a leading cause of death. Pertussis disease has always been cyclical, with peaks occurring every 3 to 5 years. With the introduction of the whole-cell pertussis vaccine in the 1940s, pertussis rates dropped dramatically. The age distribution of pertussis disease has changed as well, with the most cases occurring in unimmunized infants younger than 1 year of age.

In recent years, there has been a resurgence in pertussis reported in many countries worldwide. The reason for this resurgence is likely to be multifactorial.One of the key factors is the finding that neither natural pertussis infection nor immunization produces lifelong immunity to pertussis. Different pertussis vaccines have had varying rates of success over the years.
This suggests that immunity after acellular pertussis vaccination may begin to decline after 4 to 5 years, indicating that a booster dose may be appropriate.
Epidemiologic studies have also shown that decreasing antibody levels to pertussis toxin, for

example, at a population level, can precede large pertussis epidemics,although long-term memory B cells in vaccinated children may persist despite waning antibody levels and provide protection against pertussis disease. Additional factors that may have contributed to the resurgence in reported pertussis include increased awareness and subsequent testing for pertussis with very sensitive molecular methods that can detect as little as one organism of *B. pertussis*, making it difficult to distinguish between colonization and disease (discussed further under "Carrier State").80 Finally, it is possible that the bacterium itself has evolved and changed over time in response to vaccination practices; additional detail regarding strain variation among *B. pertussis* is included in "Molecular Diagnosis."

Carrier state

In the past, based on knowledge obtained from traditional culture methods, there was not considered to be a carrier state for *B. pertussis* in the nasopharynx. However, this may no longer be true, according to studies done with more sensitive PCR methods. In addition to circulating among adults, there may also be transient nasopharyngeal carriage of *B. pertussis* in immunized children. A case control study described a laboratory-confirmed (primarily by PCR assay) outbreak of pertussis occurring in preschool-aged children. This was not a classic pertussis, as evidenced by a lower number of cases meeting a clinical case definition, a very low hospitalization rate of unimmunized infants, and a low secondary attack rate in households. High vaccine rates may have moderated the outbreak, and, with respiratory coinfection in a significant proportion of cases, a positive PCR result may simply have reflected transient nasopharyngeal carriage of *B. pertussis* in the absence of evidence of seroconversion.

Prevention

Immunization is the single most effective means of preventing pertussis disease . The history of pertussis vaccination is long and began with attempts to develop a vaccine from the whole *B. pertussis* organism. The difficulty lay in striking the right balance between making a vaccine with enough bacteria that it was immunogenic versus making a vaccine that was too reactogenic because of additional impurities. Before the 1940s, the efficacy of a vaccine could only be assessed in human trials until Kendrick and associates developed the mouse potency test. In the mouse potency test, the efficacy of a vaccine was determined by immunizing a mouse intraperitoneally and then infecting it intracerebrally with live *B. pertussis.* Survival was measured at 14 days, and a potency unit was calculated according to WHO criteria.

Rubella

Rubella (German measles) is an acute exanthematous viral infection of children and adults. The clinical illness is characterized by rash, fever,and lymphadenopathy and resembles a mild case of measles (rubeola). Although many infections with the agent are subclinical, this virus has the potential to cause fetal infection, with resultant birth defects, and(uncommonly but especially in adults) various forms of arthritis.
Rubella was not distinguished clinically from certain other exanthematous infections until the late 19th century. It was at one time termed *third disease*, when measles and scarlet fever were called *first disease* and *second disease*, respectively.

Before widespread vaccine use, the incidence of clinical cases of postnatal rubella in temperate climates was highest in the spring, and it was traditionally recognized to be most common in children 5 to 9 years of age. Rubella is only a moderately contagious illness, in contrast to measles. Therefore, in the prevaccine era, only 80% to 90% of adults were immune to rubella, whereas 98% were immune to measles.

Epidemics of rubella of minor proportions occurred in the prevaccine era every 6 to 9 years, and large-scale epidemics occurred at intervals of up to 30 years.
Since the licensure of a live-attenuated rubella vaccine in 1969, there have been no large rubella epidemics in countries where the vaccine is widely used. However, limited outbreaks continued to occur in settings such as workplaces, schools, and military camps, where groups of susceptible individuals had close contact with each other.
Mathematical models indicate that transmission of rubella ceases at 90% immunization levels. Globally, rubella remains a significant problem, although in 2009, a decrease of 82% in cases of rubella was reported by the World Health Organization, between 2000 and 2009.
Rubella virus is spread in droplets that are shed from respiratory secretions of infected persons. Patients are most contagious while the rash is erupting, but they may shed virus from the throat from 10 days before until 15 days after the onset of the rash. Patients with subclinical cases of illness may also transmit the infection to others. Infants with congenital rubella shed large quantities of virus from body secretions for many months and therefore may transmit the infection to those who care for them. These babies continue to excrete rubella virus despite high titers of neutralizing antibody, a puzzling phenomenon that has yet to be explained. The possibility of immune tolerance due to fetal infection has been raised.
Persons who receive rubella vaccine do not transmit rubella to others, although the virus may be transiently isolated from the pharynx. It may be that the quantity of virus shed is too small to be infectious.
After an attack of rubella, lifelong protection against the disease develops in most persons. Nevertheless, despite the presence of specific immunity to rubella virus, it appears that reinfection with rubella virus can occur. This had been long suspected on clinical grounds alone.Rubella reinfections have been documented by detection of a significant boost in rubella antibody titers in naturally immune persons after reexposure to the virus. Most reinfections are asymptomatic.It is likely that the virus can multiply locally in the upper respiratory tract but that viremia occurs infrequently because the host's immune response eradicates the virus before it can invade the blood. However, in rare instances, patients have been reported to have proven rubella reinfection occurring years after naturally acquired rubella, with symptoms indicative of viremia (e.g., arthritis, rash). Rubella reinfection occurring months or years after the receipt of rubella vaccine has also been observed. Several investigators have documented reinfections in up to 80% of persons who had received rubella vaccine previously and were subsequently exposed to rubella during an epidemic.Most of these reinfections were not characterized by clinical illness but were identified only by a rise in antibody titer. Viremia is probably extremely rare in such cases, although rubella virus has been recovered from throat secretions in reinfections.

Reinfections are more common among vaccinees than among persons who have experienced natural rubella, and they are most common among persons with hemagglutination inhibition (HAI) antibody titers of 1 : 64 or less. It has been suggested that there may also be qualitative differences in antibody between persons with vaccine-induced immunity and those with natural immunity because in one study, even with similar HAI titers, vaccinees were 10 times more likely to be reinfected than were those with natural immunity to rubella.
In summary, it appears that persons who are immune to rubella, either by having had the natural infection or by having received rubella vaccine, may be reinfected when reexposed. However, this reinfection is usually asymptomatic and detectable only by serologic means.
Viremia and congenital rubella in maternal reinfection appear to be very rare events.

The presence of large numbers of immune people in a community appears to be able to prevent rubella epidemics from occurring; this effect is termed *herd immunity*. Although it has been documented that herd immunity does not entirely eliminate the spread of rubella, it probably plays a major role in control of this infection, which is now rare in the United States.

The effects of rubella virus on the fetus are, to a large extent, dependent on the time of infection; in general, the younger the fetus when infected, the more severe the illness. During the first 2 months of gestation, the fetus has a 65% to 85% chance of being affected, with an outcome of multiple congenital defects or spontaneous abortion, or both.Rubella during the third month of fetal life has been associated with a 30% to 35% chance of developing a single defect, such as deafness or congenital heart disease. Fetal infection during the fourth month carries a 10% risk for a single congenital defect. On occasion, fetal damage (deafness alone) is seen if rubella occurs up to the 20th week of gestation.

Rubella virus was isolated in 1962 and attenuated in 1966; the liveattenuated vaccine was licensed for use in the United States in 1969.The rationale for use of the vaccine is to prevent congenital rubella by control of postnatal rubella. In the United States, the first strategy was to vaccinate prepubertal children so as to minimize exposure of susceptible pregnant women to rubella. More recently, there has been an emphasis on immunization of rubella-susceptible women of childbearing age who are not pregnant. Often, this is done just after delivery of an infant; nursing mothers who are vaccinated do not cause harm to their infants. In some other countries, the approach has been to vaccinate girls against rubella as they approach puberty.

The incidence of postnatal rubella fell to an all-time low in 1988, but by 1991, it had increased threefold. There was a concomitant increase in cases of congenital rubella syndrome during the same period, although there was still a decline of more than 98% in cases of rubella compared with the prevaccine era. The observed increase in cases was attributed to failure to immunize rather than vaccine failure.

There remains, however, a continued need to emphasize the importance of immunization of susceptible women of childbearing age who are not pregnant, hospital employees, as well as infants and children.

Since the introduction of rubella vaccine, the number of reported cases of clinical rubella has declined progressively. The vaccines available today, when properly administered, produce a seroconversion rate of about 95% after one dose. Seroconversion in response to rubella vaccine is not impaired in children with upper respiratory tract infections. Because antibody titers are lower after vaccination than after natural disease, the question has been raised as to whether the antibody titer, years after vaccination, will remain high enough to preventclinical rubella. Only time and continued surveillance will provide an answer to this question, but at present, there is little evidence of waning immunity, as reflected by the low incidence of rubella in the United States. Booster injections of rubella vaccine therefore are not routinely indicated.

The observed risk for congenital rubella after immunization therefore is reported as zero; however, the theoretical maximal risk could be as high as 1% to 2%. This is in contrast to a 20% or greater risk after maternal rubella in the first trimester. Of interest, the vaccine-type virus can cross the placenta, and rubella virus has been isolated from both deciduas and fetal tissue at abortion after inadvertent vaccination of pregnant women. Rubella virus was isolated from the fetus of a woman given rubella vaccine 7 weeks before conception. A single case of persistent infection of a fetus whose mother was inadvertently immunized in early pregnancy has been recorded; the infant had no signs or symptoms of the congenital rubella syndrome.

Based on an analysis of 293 normal infants born to rubella susceptible mothers vaccinated 1 to 2 weeks before or 4 to 6 weeks after conception, for whom the theoretical risk to the fetus is 1.3%, the CDC recommends that women avoid pregnancy for 28 days after rubella vaccination. Although it is not recommended that rubella vaccine be administered to women who are pregnant, the currently

recognized minimal theoretical fetal risk does not mandate automatic termination of a pregnancy. Many, if not most, of such vaccinated women may wish to carry their baby to term.

Measles

Measles, an acute infection caused by the rubeola virus, is highly contagious and usually seen in children. The illness is characterized by conjunctivitis, cough, coryza, fever, and a maculopapular rash that begins several days after the initial symptoms appear.
Measles has been largely controlled in many developed countries since the introduction of live-attenuated measles vaccine in 1963; it remains a serious problem in developing countries, but successful efforts are now being carried out for improved control of the disease.

Epidemiology

Measles is seen in every country in the world. Without a vaccine, epidemics of measles lasting 3 to 4 months could be predicted to occur every 2 to 5 years. Countries in which measles vaccine is widely used have experienced a marked decrease in the incidence of disease.
Measles continues to be a worldwide problem that primarily affects children in developing countries. In 2000, it was estimated that more than 750,000 deaths attributed to measles occurred globally. With the advent of immunization programs supported by the World Health Organization and the United Nations Children's Fund, the estimated deaths globally have been reduced by 60%. The largest reduction in deaths was observed in Africa. Measles continues to be a problem in Europe, where vaccine use may be spotty, and introduction of measles to the United States by air travel has resulted in measles outbreaks.Despite the many challenges in controlling measles, however, eventual elimination of this infection continues to be a goal.

At present, there is minimal published evidence that immunity induced by measles vaccine wanes significantly with time. The major reasons why measles has not fully been eliminated are failure to immunize all persons who qualify for vaccination, primary vaccine failure, and importation of measles from other countries.

Spread of Infection

The measles virion is very labile; it is sensitive to acid, proteolytic enzymes, strong light, and drying. The virus, however, remains infective in droplet form in air for several hours, especially under conditions of low relative humidity. This latter fact may account for the increased incidence of measles in winter. Measles is spread by direct contact with droplets from respiratory secretions of infected persons and also by the airborne route. It is one of the most communicable of the infectious diseases, most infectious during the late prodromal phase of the illness, when cough and coryza are at their peak; however, the disease is probably contagious from several days before until several days after the onset of rash. Measles virus has been isolated from respiratory secretions of patients with measles only until up to 48 hours after the onset of rash. Airborne spread of measles in physicians' offices and in a sports complex has been observed.

Prevention

Included in the group of persons for whom passive immunization is recommended are those who are at high risk for developing severe or fatal measles, are susceptible, and/or have been exposed to the infection. This includes children with malignant disease, particularly if they are receiving chemotherapy, radiotherapy, or both, and children with significant deficits in cell-mediated immunity, including patients with AIDS. Babies younger than 1 year (including newborns whose

mothers have measles) are also at increased risk after an exposure to measles. Because measles has been reported even after vaccination in HIV-infected children, it has been recommended that they also be passively immunized with immune globulin after a recognized exposure.
To be effective, passive immunization must be given within 6 days after an exposure; administration after 6 days would not be expected to influence the course of the disease.

For a healthy infant younger than 1 year who has been exposed to measles, the modifying dose of immune globulin is 0.25 mL/kg intramuscularly (IM). An infant passively immunized in this fashion should be given live measles vaccine at the age of 15 months.For immunocompromised exposed children, a larger dose of immune globulin is required. These children should be given immune globulin, 0.5 mg/kg IM, with a maximum of 15 mL.
Vaccination is not usually recommended for infants younger than 12 months because the induction of immunity may be suppressed by residual transplacentally acquired antibodies.