<table>
<thead>
<tr>
<th>Program Name:</th>
<th>Immunization Competencies Education Program Module 2 - Vaccine-Preventable Diseases</th>
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<td>This continuing education lesson is designed primarily for community pharmacists and has been accredited by the Canadian Council on Continuing Education in Pharmacy (CCCEP) for 1.5 CEUs.</td>
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<td>CCCEP File Number: 1066-2010-092-I-P</td>
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<td>This online CME event is an Accredited Group Learning Activity (Section 1) as defined by the Maintenance of Certification program of the Royal College of Physicians and Surgeons of Canada. This program is recognized as 1 hour(s) of Continuing Professional Development.</td>
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<td>Family physicians may claim one (1) credit per hour of participation under Mainpro-M2.</td>
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<td>Course Expiration Date:</td>
<td>June 15, 2013</td>
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<tr>
<td>Sponsor:</td>
<td>This module is developed in collaboration with the Canadian Paediatric Society, the Public Health Agency of Canada and Health Canada.</td>
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Competency: Demonstrates an understanding of the rationale and benefit of immunization, as relevant to the practice setting.

Learning Objectives

Upon successful completion of this section the health professional will be able to perform the following:
1. Describe the key clinical features, including acute and long-term complications, of each vaccine-preventable disease.
2. Describe the key epidemiologic features of each vaccine-preventable disease.
3. Describe the historical impact of immunization on the epidemiology of vaccine-preventable disease.
4. For each of the vaccines administered in the practice setting, formulate a response to the question “Why should I be immunized when vaccine-preventable diseases are so rare in Canada?”
5. Explain why accurate diagnosis of vaccine-preventable diseases is important.

Test your Current Knowledge:
Based on your current knowledge, determine if the following statements are true or false.
1. In countries where vaccination rates are low, disease rates are high
   a. True
   b. False
2. In Canada approximately 20% of adults do not have protective levels of antibody to diphtheria
   a. True
   b. False
3. *Haemophilus influenza* type b is a major cause of otitis media, sinusitis and respiratory tract infections
   a. True
   b. False
4. The case fatality for Hepatitis B is greater than for hepatitis A
   a. True
   b. False
5. Most of the influenza outbreaks are caused by influenza type c
   a. True
   b. False
6. The complication risk from measles is very low, but we immunize due to its high transmission rate
   a. True
   b. False
7. Approximately 90% of susceptible family members will become infected with mumps following an illness in a child
   a. True
   b. False
8. Approximately 20-30% of infants with pertussis require hospitalization
   a. True
b. False
9. Only the conjugated form of the pneumococcus vaccine is effective in children < 2 years of age
   a. True
   b. False
10. Approximately 1 in 1000 patients infected with polio develop the paralytic polio complication
    a. True
    b. False
11. A tetanus infection kills approximately 10 % of infected people
    a. True
    b. False
12. Prior to immunization close to 95% of Canadians would be infected with the varicella zoster virus
during their lifetime
    a. True
    b. False

**Why Should I immunize when Vaccine Preventable Diseases are so Rare?**

Every clinician involved in immunization has been asked this question from parents. Crucial communication is required to help parents understand the risk and benefits of immunization for their child and for the population as a whole.

To effectively communicate risk, clinicians need to be familiar and comfortable with each vaccine preventable disease, their key epidemiology statistics, and their short-term and long-term sequelae. They should be able to communicate the benefits and risks associated with immunizing against these conditions.

This module focuses on providing the clinician with the information to answer this question. This will be covered through reviewing the main vaccine preventable diseases and the impact of immunization.

**Do vaccines work?**

Yes, vaccines work very well. We know that in countries where vaccination rates are high, disease rates are low. We also know that the opposite is true. In countries where vaccination rates are low, disease rates are high.¹

Here are some examples:

- In the United Kingdom, in 1974, the number of people who got the vaccine against pertussis (whooping cough) dropped. By 1978, the country had an epidemic of this disease. More than 100,000 people got it, and 36 people died.¹
- A diphtheria epidemic happened in the former Soviet Union because children stopped getting the vaccine and adults did not get booster shots. During there were over 140,000 diphtheria cases and 4,000 deaths reported.¹

Table 1 lists the impact of Canada’s immunization program on vaccine preventable diseases.
<table>
<thead>
<tr>
<th>Details</th>
<th>Disease</th>
<th>Pre-vaccine era</th>
<th>2000-2004</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5-year average annual incidence per 100,000</td>
<td>5-year average annual incidence per 100,000</td>
</tr>
<tr>
<td>Diphtheria</td>
<td>Diphtheria toxoid introduced in 1926, routine infant immunization since 1930, national notifiable diseases reporting began in 1924</td>
<td>1925-29 84.2</td>
<td>1925-29 9,010</td>
</tr>
<tr>
<td>Invasive Haemophilus influenzae type b (Hib) in children &lt;5 years of age</td>
<td>PRP vaccine introduced in 1986, currently approved Hib PRP-T and PRP-OMP conjugate vaccines introduced in 1991/92, national notifiable diseases reporting of invasive Hib disease began in 1986</td>
<td>1986-90 22.7</td>
<td>1986-90 526</td>
</tr>
<tr>
<td>Measles</td>
<td>Live vaccine approved in 1963, MMR universal infant program implemented in 1983, 2 dose MMR introduced 1996/97, no notifiable diseases reporting from 1959-68</td>
<td>1950-54 369.1</td>
<td>1950-54 61,370</td>
</tr>
<tr>
<td>Mumps</td>
<td>Vaccine approved in 1969, MMR universal infant program implemented in 1983, 2 dose MMR introduced 1996/97, no notifiable diseases reporting from 1960-85</td>
<td>1950-54 248.9</td>
<td>1950-54 43,671</td>
</tr>
<tr>
<td>Pertussis</td>
<td>Whole cell pertussis vaccine approved in 1943, acellular pertussis vaccine replaced whole cell in 1997-98, adolescent/adult acellular formulation approved in 1999</td>
<td>1938-42 156.0</td>
<td>1938-42 19,878</td>
</tr>
</tbody>
</table>
Diptheria Toxoid

Causal Agent

Diphtheria is an acute, communicable disease caused by exotoxin-producing strains of the bacterium Corynebacterium diphtheriae. C. diphtheriae is an aerobic gram-positive bacillus. Toxin production (toxigenicity) occurs only when the bacillus is itself infected (lysogenized) by a specific virus (bacteriophage) carrying the genetic information for the toxin (tox gene). Only toxigenic strains can cause severe disease.

Pathogenesis

Susceptible individuals may acquire toxigenic diphtheria bacilli in the nasopharynx. The organism produces a toxin that inhibits cellular protein synthesis and is responsible for local tissue destruction and membrane formation. The toxin produced at the site of the membrane is absorbed into the bloodstream and then distributed to the tissues of the body.

Clinical Notes from the Canadian Immunization Guide:

In Canada approximately 20% of adults do not have protective levels of antibody to diphtheria.

The potential for disease re-emergence if immunization levels are allowed to fall was demonstrated during the 1990s in the former Soviet Union, where over 140,000 cases and 4,000 deaths were reported.

Transmission

Transmission of the infection is by inhalation of aerosolized droplets and through contact with articles (such as clothing or bed linen) soiled by infected persons.

Transmission may occur as long as virulent bacilli are present in any discharge and lesions. This time is variable, but organisms usually persist 2 weeks or less, and seldom more than 4 weeks, without antibiotics. Chronic carriers may shed organisms for 6 months or more. Effective antibiotic therapy promptly terminates shedding. Approximately 3-5% of healthy persons are carriers of diphtheria. With immunization, only 0 to 5 isolates are found each year in Canada.

Signs and Symptoms

The most characteristic features of diphtheria affecting the upper respiratory tract are a membranous pharyngitis (often referred to as a pseudo-membrane) with fever, enlarged anterior cervical lymph nodes and edema of soft tissue giving a ‘bull neck’ appearance. The pseudo-membrane may cause respiratory obstruction.
Most complications of diphtheria, including death, are attributable to effects of the toxin. The severity of the disease and complications are generally related to the extent of local disease. The toxin, when absorbed, affects organs and tissues distant from the site of invasion. The most frequent complications of diphtheria are myocarditis and neuritis.³

Fatality rates are between 5–10% and higher in younger and older patients.²

Key Points:²

- Communicable disease caused by the infection with exotoxin producing bacteria
- Spread by direct physical contact or breathing aerosolized secretions of infected individuals
- Immunization introduced in 1930
- Peak incidence: 9,000 cases (1924). In Canada there is now 0 to 5 isolates per year.
- Fatality rate of 5–10%
- The vaccine provides antitoxic protection and is not antibacterial. Therefore a patient can still be infected by the bacteria but is protected from the potentially lethal systemic effects.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the diphtheria vaccine. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#diphtheria

**Haemophilus Influenza Type B (Hib)**

**Causal Agent**

*Haemophilus influenzae* type b (Hib) is a cause of bacterial infections that are often severe, particularly among
Pathogenesis

The organism enters the body through the nasopharynx. Organisms colonize the nasopharynx and may remain only transiently or for several months in the absence of symptoms (asymptomatic carrier).\(^3\)

In some persons, the organism causes an invasive infection. The exact mode of invasion to the bloodstream is unknown. Antecedent viral or mycoplasma infection of the upper respiratory tract may be a contributing factor. The bacteria spreads in the bloodstream to distant sites in the body. Meninges are especially likely to be affected.\(^3\)

Transmission

Hib is spread through coughing, sneezing or close contact with a carrier or an infected person.\(^4\) The organism enters the body through the nasopharynx.

Signs and Symptoms

Invasive disease caused by *H. influenzae* type b can affect many organ systems. The most common types of invasive disease are meningitis, epiglottitis, pneumonia, septic arthritis, and cellulitis.\(^3\) *H. influenzae* is also commonly associated with otitis media, sinusitis, bronchitis and other respiratory tract disorders. These infections are seldom caused by Hib and are not the target of the vaccine.

Table 2 lists some of the Signs and Symptoms of the different complications associated with Hib infection.

### Table 2 – Signs and Symptoms of Complications Associated with Hib Infection\(^3\)

<table>
<thead>
<tr>
<th>Complication</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meningitis</td>
<td>• Fever&lt;br&gt;• Decreased mental status&lt;br&gt;• Stiff neck&lt;br&gt;• Hearing impairment or other neurologic sequelae occur in 15%–30% of survivor</td>
</tr>
<tr>
<td>Epiglottitis</td>
<td>• Swelling of the epiglottis (tissue in the throat that covers and protects the larynx during swallowing)&lt;br&gt;• Epiglottitis may cause life-threatening airway obstruction</td>
</tr>
</tbody>
</table>
| Septic arthritis | • Fever  
| | • Severe pain  
| | • Swelling in the affected joint  
| Cellulitis | • Fever  
| | • Redness  
| | • Pain  
| | • Tenderness  

About 55% to 65% of affected children develop meningitis. The case-fatality rate of meningitis is about 5%. Severe neurologic sequelae occur in 10% to 15% of survivors and deafness in 15% to 20% (severe in 3% to 7%).

**Key Points:**

- *Haemophilus Influenzae* type b (HIB) was the most common cause of bacterial meningitis and a leading cause of other invasive infections in young children prior to the introduction of the Hib vaccine.
- Transmission occurs by direct contact with respiratory droplets from an infected patient.
- Peak incidence: 2.6 per 100,000 (1988) to 0.3 per 100,000 in 2004.
- 55-65% of children with Hib will develop meningitis, the remainder suffering from epiglottitis, bacteremia, cellulitis, pneumonia and septic arthritis.
- The case-fatality rate of meningitis is about 5%. Severe neurologic sequelae occur in 10% to 15% of survivors and deafness in 15% to 20% (severe in 3% to 7%).
- The most striking feature of Hib disease is age-dependent susceptibility. Hib disease is not common beyond 5 years of age.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the Hib vaccination. To find out more visit: [http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#hib](http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#hib)

**Hepatitis A (HAV)**

**Causal Agent**

Hepatitis A is caused by infection with Hepatitis A virus (HAV), a non-enveloped RNA virus. Depending on conditions, HAV can be stable in the environment for months. The virus is relatively stable at low pH levels and moderate temperatures but can be inactivated by high temperature.

Improved standards of living and hygiene have led to a marked fall in the incidence of hepatitis A infection. The similarities between the epidemiology of hepatitis A and poliomyelitis suggest that widespread vaccination of appropriate susceptible populations can substantially lower disease incidence, eliminate virus transmission, and ultimately, eliminate HAV infection.
Clinical Notes from the Canadian Immunization Guide:

In recent years 40% of all notified hepatitis A cases have been in travellers. Among those, 40% are staying for short periods of time in luxury hotels where meals are provided.

More than 5 million Canadians go to an HAV-endemic country in a year. Over 5 years, 30% of Canadians will travel to an endemic country, yet less than 15% of these travelers go to a travel clinic and receive hepatitis A vaccine.

Pathogenesis

HAV is acquired by mouth and replicates in the liver. After 10–12 days, virus is present in blood and is excreted via the biliary system into the feces. The incubation period of HAV is 15 to 50 days with a mean of 28 days.

Shedding of the virus is in feces and thus maximum infectiousness occurs during the latter part of the incubation period with peak levels in the 2 weeks before clinical illness. Infectiousness diminishes rapidly thereafter and ends shortly after the onset of jaundice. Most infected people no longer excrete virus in the feces by the third week of illness. Children may excrete virus longer than adults.

Transmission

HAV is most frequently transmitted by the fecal-oral route, through direct contact with infected people or indirectly through ingestion of contaminated water or foods. On rare occasions, transmission has been reported after exposure to HAV-contaminated blood or blood products. It also occurs through sexual activities that include direct or indirect oro-anal contact but not through exposure to saliva, semen or urine.

Signs and Symptoms

Infection usually causes clinical hepatitis in adults and school-aged children but is often asymptomatic in younger children. Typical symptoms of illness include anorexia, nausea, fatigue, fever and jaundice. Of note, jaundice develops in < 10% of children 6 years and under.

The severity of the illness increases with age. Recovery often takes 4 to 6 weeks but may take months. Recurrent hepatitis for up to a year occurs in about 15% of cases, but longer chronic infection is not known to occur. About 25% of reported adult cases require hospitalization. Fulminant disease with liver necrosis is rare but can be fatal.

The overall estimated case fatality rate associated with hepatitis A is 0.1% to 0.3%, but this rises to 1.8% in persons over the age of 50. It reaches 12.5% in patients over the age of 60 who are hospitalized because of the disease.

Key Points:

- Hepatitis A is an infection of the liver caused by hepatitis A virus
HAV is most frequently transmitted by the fecal-oral route, through direct contact with infected people or indirectly through ingestion of contaminated water or foods.

Immunization introduced in 1996

Peak incidence: 10.8 per 100,000 (1991) down to 2.2 per 10,000 in 2003.

Typical symptoms of illness include anorexia, nausea, fatigue, fever and jaundice.

Recurrence hepatitis for up to a year occurs in about 15% of cases, but longer chronic infection is not known to occur.

The overall estimated case fatality rate associated with hepatitis A is 0.1% to 0.3%, but this rises with age.

Hepatitis B (HBV)

Causal Agent

Hepatitis B virus (HBV) is a double-stranded DNA virus with three major antigens, known as hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg) and hepatitis B core antigen (HBcAg). Humans are the only known host for HBV. This virus is relatively resilient and, in some instances, has been shown to remain infectious on environmental surfaces for more than 7 days at room temperature.

An estimated 2 billion persons worldwide have been infected with HBV, and more than 350 million persons have chronic, lifelong infections. HBV infection is an established cause of acute and chronic hepatitis and cirrhosis. It is the cause of up to 80% of hepatocellular carcinomas.

Pathogenesis

A patient becomes infected through coming in contact with hepatitis B infected body fluids. The incubation period is 45 to 160 days with an average of 120 days. Hepatitis B surface antigens (HBsAg) can be detected in the serum 30 to 60 days after exposure and persist until the infection resolves. Any person testing positive for HBsAg should be considered infectious.

An individual with either acute symptomatic or asymptomatic HBV infection may become a chronic carrier. The risk of becoming a chronic carrier varies inversely with the age at which infection occurs (infants: 90% to 95%; children < 5 years: 25% to 50%; adults: 3% to 10%). The risk of becoming a chronic carrier is also greater in immunocompromised patients. Chronic carriers often do not have overt disease but over time are at increased risk of developing hepatic cirrhosis and primary hepatocellular carcinoma. All carriers should be considered infectious.

Persons with acute or chronic HBV infections should prevent their blood and other potentially infective body fluids from contacting other persons. They should not donate blood or share toothbrushes or razors with household members.

Clinical Note:

In routine clinical practice to prevent transmission of diseases, one assumes that blood, body fluids, and excretions and secretions from any person could contain pathogens and avoids direct contact.

Transmission
The virus is transmitted by parenteral or mucosal exposure to HBsAg-positive body fluids from persons who have acute or chronic HBV infection. The highest concentrations of virus are in blood and serous fluids; lower levels are found in other fluids, such as saliva and semen. There appears to be no transmission of HBV via tears, sweat, urine or stool.

**Modes of transmission include:**

- Any Sexual contact, with an infected person. Fecal-oral transmission does not appear to occur.
- Direct percutaneous inoculation of HBV by needles during injection-drug use. Transmission of HBV may also occur by other percutaneous exposure, including tattooing, ear piercing, and acupuncture, as well as needlesticks or other injuries from sharp instruments sustained by medical personnel.
- Contamination of mucosal surfaces with infective serum or plasma. This is seen in laboratories and hospitals that are in direct contact with these products (e.g. a lab technician being splashed in the face with infected fluid at a lab accident).
- Perinatal transmission from mother to infant at birth is very high.

A third of infections occur in people that do not have identified risk factors and do not know how they acquired hepatitis B infection.

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**Clinical Note:**

The introduction of school based immunization program was important as they protect children prior to potential sexual contact exposure and can decrease both HBV infections and complications.

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**Signs and Symptoms**

Initial infection with HBV may be asymptomatic in up to 50% of adults and 90% of children. When symptoms occur, they include an insidious onset of anorexia, vague abdominal pain, nausea, vomiting and jaundice. Acute illness may last up to 3 months and has a case fatality rate of 1% to 2%, which increases with age. While most acute HBV infections in adults result in complete recovery, fulminant hepatitis occurs in about 1% to 2% of acutely infected persons. Fulminant hepatitis and death may also occur in pregnant women and in infants born to infected mothers.

Around 20 to 25% of individuals with chronic HBV infection worldwide have progressive liver disease, leading to cirrhosis in some patients. The risk of progression is related to the level of active viral replication in the liver. Individuals with chronic hepatitis B infection, particularly those with an active inflammation and/or cirrhosis are at increased risk of developing hepatocellular carcinoma.

**Key Points:**

- Hepatitis B virus (HBV) is a double-stranded DNA virus with three major antigens, known as hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg) and hepatitis B core antigen (HBeAg).
- The virus is transmitted by parenteral or mucosal exposure to HBsAg-positive body fluids from persons who have acute or chronic HBV infection.
- Immunization introduced in 1982.
• Peak incidence: 20,000 new HBV infections occurred every year prior to immunization. At least 10% caused chronic infection that resulted in 400 deaths from cirrhosis each year and about 80 deaths from liver cancer.5
• HBV virus is the cause of up to 80% of hepatocellular carcinoma.
• Initial infection with HBV may be asymptomatic in up to 50% of adults and 90% of children. When symptoms occur, they include an insidious onset of anorexia, vague abdominal pain, nausea, vomiting and jaundice. Acute illness may last up to 3 months.
• The case fatality rate is 1% to 2%, and increases with age.
• The CDC has some useful information to use in patient consultations on the consequences if we discontinued the hepatitis B vaccine. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#hepb

Human Papillomavirus (HPV)

Causal Agent

Human papillomaviruses are small, double-stranded DNA viruses that infect the epithelium. More than 100 HPV types have been identified. Infection with low-risk, or nononcogenic types, such as types 6 and 11, can cause benign or low-grade cervical cell abnormalities, genital warts and laryngeal papillomas. High-risk HPV types are detected in 99% of cervical cancers. Type 16 is the cause of approximately 50% of cervical cancers worldwide, and types 16 and 18 together account for about 70% of cervical cancers. Infection with a high-risk HPV type is considered necessary for the development of cervical cancer.

Pathogenesis

HPV infection occurs at the basal epithelium. Although the incidence of infection is high, most infections resolve spontaneously. A small proportion of infected persons become persistently infected; persistent infection is the most important risk factor for the development of cervical cancer precursor lesions. Infection with one type of HPV does not prevent infection with another type. Of persons infected with mucosal HPV, 5% to 30% are infected with multiple types of the virus.

<table>
<thead>
<tr>
<th>Clinical Note:</th>
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<tr>
<td>Risk factors for HPV infection are related to sexual activity, including the number of sex partners, lifetime history of sex partners, and the partners’ sexual history.</td>
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Transmission

HPV is transmitted by direct contact, usually sexual, with an infected person. Transmission occurs most frequently with sexual intercourse but can occur following non-penetrative sexual activity. Studies of newly acquired HPV infection demonstrate that infection occurs soon after onset of sexual activity. It is highly contagious as sexual contact with an infected person will result in the spreading of HPV to two-thirds of sexual partners. People in their late teens and early twenties have the highest rates of infection.

Signs and Symptoms
Most HPV infections are asymptomatic and result in no clinical disease. Most infections with cancer-associated HPV are transient and do not lead to any illness. If the infection persists then there is a high risk of progression to cancer of the genital tract. Anogenital warts (anus/vagina/penis) are the most common disorder caused by genital HPV infection. Approximately 1% of sexually active men and women between 18 and 49 years of age have genital warts.

Each year, cervical cancer is diagnosed in 1350 Canadian women and approximately 400 die from the infection.

Key Points:

- Human papillomavirus (HPV) is the most common sexually transmitted infection, with 3 out of 4 sexually active Canadians infected at some point in their life.
- HPV is spread by direct physical contact between an infected and an uninfected person. It is highly contagious as sexual contact with an infected person will result in the spreading of HPV to two-thirds of sexual partners.
- Immunization introduced in 2006
- Most HPV infections are asymptomatic and result in no clinical disease. Clinical manifestations of HPV infection include anogenital warts, recurrent respiratory papillomatosis, cervical cancer precursors (cervical intraepithelial neoplasia), and cancers, including cervical, anal, vaginal, vulvar, penile, and some head and neck cancer.
- Genital warts are present in approximately 1% of sexually active men and women between 18 and 49 years of age.
- Each year, cervical cancer is diagnosed in 1350 Canadian women and approximately 400 die each year from this condition.

Influenza

Causal Agent

Influenza is a single-stranded, helically shaped, RNA virus. The three types are influenza type A, type B and type C based on their nuclear material.

Influenza type A has subtypes that are determined by the surface antigens hemagglutinin (H) and neuraminidase (N). Hemagglutinin has a role in virus attachment to cells. Neuraminidase has a role in virus penetration into cells. Influenza A causes moderate to severe illness and affects all age groups. The virus infects humans and other animals. Influenza A viruses are perpetuated in nature by wild birds, predominantly waterfowl. Most of these viruses are not pathogenic to their natural hosts and do not change or evolve.

Influenza type B generally causes milder disease than type A and primarily affects children. Influenza B outbreaks are also common in long-term care facilities and can cause severe disease in the frail elderly. Influenza B is more stable than influenza A, with less antigenic drift and consequent more immunologic stability. Outbreaks of influenza B are generally more localized and in any one year may be restricted to one region of the country.

Influenza type C is rarely reported as a cause of human illness, probably because most cases are subclinical. It has not been associated with epidemic disease.
Pathogenesis

The respiratory tract is the major target of influenza virus. The virus attaches to and penetrates respiratory epithelial cells in the trachea and bronchi. Viral replication occurs, which results in the destruction of the host cell.

Influenza A vaccine is continually evolving. Hemagglutinin and neuraminidase periodically change, apparently due to sequential evolution within immune or partially immune populations. Antigenic mutants emerge and are selected as the predominant virus to the extent that they differ from the antecedent virus, which is suppressed by specific antibody arising in the population as a result of infection. This cycle repeats continuously. In interpandemic periods, mutants arise by serial point mutations in the RNA coding for hemagglutinin. This minor change in surface antigens is referred to antigen drift.

Antigenic shift is a major change in one or both surface antigens (H or N) that occurs at varying intervals. Antigenic shifts are probably due to genetic recombination (an exchange of a gene segment) between influenza A viruses, usually those that affect humans and birds. An antigenic shift may result in a worldwide pandemic if the virus is efficiently transmitted from person to person.

Major epidemics in history include:
- 1889-90 – (“Russian Flu”) 250,000 deaths in Europe and a death total worldwide of 2 to 3 times that number
- 1918 – (“Spanish Flu”) 20 million deaths worldwide, 450,000 deaths in the United States, 30,000-50,000 deaths in Canada
- 1957 – (“Asian Flu”) 2 million deaths worldwide
- 1968 – (“Hong Kong Flu”) 1 million deaths worldwide

Transmission

Infected patients will shed the influenza virus in respiratory secretions for 5-10 days. The virus is transmitted to other people by the inhalation of infected droplets or through reaching the tissues of the nasopharynx via contaminated fingers.

Signs and Symptoms

Influenza infection results in a wide range of illness:
- Cold-like illness with or without fever
- Common flu symptoms such as sudden onset of fever, headache and myalgia (muscle pain)
- High fever (> 40°C/104°F) without other symptoms, especially in infants and young children
- Fever is often absent in the frail elderly but cough, headache and myalgias are often prominent
- Croup in children < 2 years of age

The symptoms are the result in damage to the respiratory tract caused by the influenza virus. The generalized symptoms of fever, headache, myalgia, and exhaustion are not the result of spread of the virus through the body, but rather the result of the intense response of body defences to the infection. Influenza is unique in the rapidity of onset of symptoms. Individuals feel well and then are suddenly (over a few hours) overcome by headache, myalgia and intense fatigue.
Clinical Note:
The 2009-2010 Influenza pandemic affected a different group of individuals, namely infants, young adults and pregnant women.

Key Points:
- Influenza is caused by influenza A and B viruses and occurs in Canada every year, generally during late fall and the winter months.
- There are a large variety of influenza viruses based on the hemagglutinin (H) and neuraminidase (N) surface antigens on the influenza A virus. A patient with antibodies to these antigens, particularly to H antigen, can only protect an individual against a virus carrying the same antigen. For this reason immunity to influenza is very specific and requires an annual immunization based on the most common strains circulating throughout the region.
- During inter-pandemic periods, minor H antigen change (referred to as drifts) is common. The greater the change in antigens the less the cross-immunity to the previously circulating virus.
- Pandemic influenza is usually associated with a major antigenic change (referred to as a shift) and the rapid global spread of influenza A virus with a different H and possibly a different N antigen from strains circulating previously.
- “Classic” influenza disease is characterized by the abrupt onset of fever, myalgia, sore throat, non-productive cough, and headache. Symptoms may present atypically in young children and frail elderly.
- Each year, influenza infects 10-20% of the population of North America. In Canada there have been 4000-8000 deaths caused by influenza annually.

Measles

Causal Agent
The measles virus is a paramyxovirus, genus Morbillivirus. It is 100–200 nm in diameter, with a core of single-stranded RNA. Although there has been some change in the antigen component of the measles, they are not viewed as significant changes as there has been no change in vaccine efficacy.

Pathogenesis
Measles is a systemic infection. The primary site of infection is the respiratory epithelium of the nasopharynx. Two to three days after invasion and replication in the respiratory epithelium and regional lymph nodes the virus enters the blood and is carried throughout the body to other lymph glands, the liver, spleen and bone marrow where it continues to multiply for 3 to 5 days. The virus then reinvades the blood and spreads to the skin, eyes, respiratory tract and other organs. The virus reaches a peak level at 11 to 14 days after exposure then declines rapidly over a few days.

The measles virus is shed from the nasopharynx beginning with the prodrome until 3–4 days after rash onset.
Clinical Note:

Measles is so contagious that over 90% of susceptible person exposed at home to a child with measles will develop it.4

Transmission

Measles transmission is primarily person to person via large respiratory droplets. Airborne transmission via aerosolized droplet nuclei has been documented in closed areas (e.g. office examination room) for up to 2 hours after a person with measles occupied the area.3 The measles virus is rapidly inactivated by heat, light, acidic pH, ether, and trypsin. It has a short survival time (less than 2 hours) in the air or on objects and surfaces.3

Signs and Symptoms

The incubation period for measles from exposure to prodrome averages 10-12 days.3 The prodrome phase lasts for 2-4 days (range 1-7 days) and is characterized by fever, which increases in a stepwise fashion reaching 39.4-40°C (103-104°F).5 This is followed by the onset of cough, runny nose and conjunctivitis.3 During this phase blue-white spots (Koplik spots) can be seen on buccal mucosa.5 These spots appear 1-2 days before the rash and up to 1-2 days after the rash and are characteristic of measles infection.3

Rash from Measles
Photo Courtesy: Center of Disease Control and Prevention

Koplik Spots
Photo Courtesy: Center of Disease Control and Prevention
The measles rash consists of large red spots that first appear on the face and head and spread down over the body to the arms and legs.\(^5\) The spots can become so large that there may be no unaffected skin in between, especially on the face and upper body.\(^5\) The average rash begins to fade after about a week and the total illness is 7 to 14 days.\(^5\)

Complications from measles are very common due to spread of the virus throughout the body and the extensive damage to the respiratory tract.\(^5\)

Complications include:\(^5\)

- Otitis media: 7-9\% of children
- Bacterial pneumonia: 1-6\%
- Diarrhea: 6\%
- Encephalitis: 1 in 1000 cases
- Subacute sclerosing panencephalitis (SSPE): 1 in 100,000 cases

Hospitalization occurs in 1\% of children with measles.\(^5\) Death occurs in approximately 1 in 1000 cases of measles.\(^5\) The most common causes of death from measles is by pneumonia and encephalitis.\(^5\) Measles during pregnancy results in a higher risk of premature labour, spontaneous abortion and low birth weight infants. Subacute sclerosing panencephalitis (SSPE) is a rare but fatal complication of measles. It is caused by a chronic infection of brain cells by the measles virus.\(^5\)

Clinical Note:

In March 2010 a measles outbreak occurred in lower mainland of British Columbia. As of May 17, 2010 there were 87 confirmed cases mostly in unimmunized young people and partially immunized adults.

Key Points:\(^2\)

- The measles virus is a paramyxovirus, genus Morbillivirus, with a core of single-stranded RNA\(^3\)
- Measles transmission is primarily person to person via large respiratory droplets.\(^3\)
- Vaccine introduced in 1954
- Peak incidence: Before the vaccine measles epidemics were common every 2 to 3 years and an estimated 300,000 to 400,000 cases occurred annually; down to a yearly average of 14 between 2001 and 2005.
- First symptoms of a measles infection are fever, aches and pains, runny nose, cough and red and inflamed eyes. A rash will develop starting on the face and head and spreading down over the body to arms and legs. The total illness is 7-14 days.\(^5\)
- Approximately 30\% of reported measles cases have one or more complications.\(^3\) Complications include measles encephalitis (1 in 1000 cases) resulting in permanent brain damage. Measles during pregnancy results in a higher risk of premature labour, spontaneous abortion and low birth weight infants. Otitis media, pneumonia are common complications.\(^3\) Deaths occur in about 1 in every 1000 measles cases.\(^5\)
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the measles vaccination. To find out more visit: [http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#measles](http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#measles)
Meningococcal

Causal Agent

N. meningitidis, or meningococcus, is an aerobic, gram-negative diplococcus, closely related to N. gonorrhoeae.\(^3\) The outer membrane contains several protein structures that enable the bacteria to interact with the host cells as well as perform other functions.\(^3\) The outer membrane is surrounded by a polysaccharide capsule that is necessary for pathogenicity because it helps the bacteria resist phagocytosis and complement-mediated lysis. The outer membrane proteins and the capsular polysaccharide make up the main surface antigens of the organism.\(^3\)

Meningococci are classified by using serologic methods based on the structure of the polysaccharide capsule.\(^3\) Almost all invasive disease is caused by one of five serogroups: A, B, C, Y, and W-135. The relative importance of each serogroup depends on geographic location, as well as other factors, such as age.\(^3\)

Pathogenesis

Meningococci are transmitted by droplet aerosol or secretions from the nasopharynx of colonized persons. The bacteria attach to and multiply on the mucosal cells of the nasopharynx. In a small portion (less than 1%) of colonized persons, the organism penetrates the mucosal cells and enters the bloodstream.\(^3\) The bacteria spread by way of the blood to many organs. In about 50% of bacteremic persons, the organism crosses the blood–brain barrier into the cerebrospinal fluid and causes purulent meningitis. An antecedent upper respiratory infection may be a contributing factor.\(^3\)

A majority of individuals with meningococcal disease are asymptomatic carrier. The bacteria resides and multiplies in the nasopharynx but causes no symptoms or illness.\(^5\) The frequency of carrier state is very low in infants and young children and is highest (10-30%) in adolescents and young adults.\(^5\)

<table>
<thead>
<tr>
<th>Clinical Notes from the Canadian Immunization Guide:</th>
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<tbody>
<tr>
<td>An average of 298 cases of meningococcal disease has been reported annually. Disease occurs year round, but there is seasonal variation with the majority of cases occurring in the winter months.</td>
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</table>

Transmission

The bacteria are extremely fragile outside the body and are not highly contagious.\(^5\) Meningococci are transmitted by droplet aerosol or secretions from the nasopharynx of colonized persons.\(^3\) The bacteria attach to and multiply on the mucosal cells of the nasopharynx. In a small proportion (less than 1%) of colonized persons, the organism penetrates the mucosal cells and enters the bloodstream.\(^3\)

Signs and Symptoms

The incubation period of meningococcal disease is 3 to 4 days, with a range of 2 to 10 days.\(^3\) Approximately 40% of patients with clinical manifestations of meningococcal disease have meningitis alone, 40% have both meningitis and septicemia, 10-15% have septicemia alone and 5% have other forms (pneumonia, arthritis or otitis media).\(^5\)
Meningitis is the most common presentation of invasive meningococcal disease and results from the spread of the organism through the blood. Patients will present with a sudden onset of fever, headache, and stiff neck, often accompanied by other symptoms, such as nausea, vomiting, photophobia (eye sensitivity to light), and altered mental status. Meningococci can be isolated from the blood in up to 75% of persons with meningitis. The case-fatality rate of invasive meningococcal disease is 9% to 12%, even with appropriate antibiotic therapy.

Septicemia is the most severe form of meningococcal disease. While in the blood the bacteria releases endotoxin that causes an intense reaction of the body’s defence system. This condition is characterized by abrupt onset of fever and a petechial or purpuric, non-blanching rash, often associated with hypotension, shock, acute adrenal hemorrhage and multi-organ failure. The fatality rate of meningococcemia is up to 40%. As many as 20% of survivors have permanent sequelae, such as hearing loss, neurologic damage, or loss of a limb. Septicemia can kill very rapidly, with the total of first symptoms to death being as short as 6 to 12 hours.

Key Points:

• Meningococcal disease is the result of a systemic bacterial infection by the gram-negative diplococccic Neisseria meningitidis.
• Invasive meningococcal disease (IMD) is endemic in Canada, showing periods of increased activity roughly every 10 to 15 years with no consistent pattern.
• Meningococcal disease is primarily transmitted by respiratory droplet spread or by direct contact. Approximately 10-20% of adults and adolescents are healthy carriers of meningococcal disease and act as reservoirs for the disease.
• Vaccine introduced in 1960’s and 1970’s against groups A and C meningococci. This vaccine was not effective in children less than 2 years of age. In 2006, a new conjugate vaccine against strains A, C, Y and W135 became available.
• Peak incidence: Large epidemics occurred every 7-10 years. Between 1940 and 1943 there were more than 2600 cases per year. Since, 1985, the overall incidence of IMD has remained at or below 2 per 100,000 per year.
• Meningitis is the most common presentation of invasive meningococcal disease. Septicemia, pneumonia, arthritis, otitis media and epiglottis can also occur.
• The case-fatality rate of IMD is 9% to 12%, even with appropriate antibiotic therapy. The fatality rate of meningococcemia is up to 40%. As many as 20% of survivors have permanent sequelae, such as hearing loss, neurologic damage or the loss of a limb.

Mumps

Causal Agent

Mumps virus is a paramyxovirus in the same group as parainfluenza and Newcastle disease virus. The virus has a single-stranded RNA genome. It has been recovered from the saliva, cerebrospinal fluid, urine, blood, milk, and infected tissues of patients with mumps.

Pathogenesis
The virus enters the body and starts to replicate in the nasopharynx and regional lymph nodes. After 12 to 25 days a viremia occurs, which lasts from 3 to 5 days. During the viremia, the virus spreads to multiple tissues, including the meninges, and glands such as the salivary, pancreas, testes, and ovaries. Inflammation in infected tissues leads to characteristic symptoms of parotitis and aseptic meningitis.3

Clinical Notes from the Canadian Immunization Guide:

A single dose of mumps vaccine produces an antibody response in over 95% of susceptible individuals. However, field studies have demonstrated lower estimates of vaccine efficacy, usually around 80% with single-dose regimens. For this reason each province has implemented a 2 dose regimen.

Transmission

The virus is acquired by respiratory droplets.3 The spread of the mumps virus requires close and direct contact with an infected patient.3 Mumps is less contagious than measles or chicken pox with only 1/3 of susceptible family members getting mumps following an illness in a child.5

Signs and Symptoms

Approximately 20% of mumps infections are asymptomatic. An additional 40% to 50% may have only nonspecific or primarily respiratory symptoms.3 Mumps has a long incubation period of 14-18 days (range 14 to 25 days).3 This is followed by a prodromal stage where patients will have nonspecific symptoms such as myalgia, malaise, headache and low-grade fever.3

Inflammation of the parotid salivary gland is a common complication.3 The parotid gland is the largest salivary gland and is located in front of the ear behind the angle of the jaw. Parotitis may be unilateral or bilateral, and any combination of single or multiple salivary glands may be affected. Parotitis tends to occur within the first 2 days and may first be noted as earache and tenderness.3 Symptoms tend to decrease after 1 week and usually resolve after 10 days.3

Meningitis commonly occurs with mumps.5 Symptomatic meningitis (headache, stiff neck) occurs in up to 15% of patients.3 Most cases are very mild and symptoms such as headache, neck stiffness and drowsiness last only a few days.5 It resolves without sequelae in 3 to 10 days.5

Orchitis (testicular inflammation) is the most common complication in postpubertal males.3 It occurs in as many as 20-40% of postpubertal males, usually after parotitis.3 It is bilateral in approximately 30% of affected males. There is usually abrupt onset of testicular swelling, tenderness, nausea, vomiting, and fever.3 Pain and swelling may subside in 1 week, but tenderness may last for weeks. Approximately 50% of patients with orchitis have some degree of testicular atrophy, but sterility is rare even when both testicles are infected.3

Oophoritis (ovarian inflammation) occurs in 5% of postpubertal females. It may mimic appendicitis. Patients may have painful swelling of the breasts and abdominal or pelvic pain.5 There is no relationship to impaired fertility.

Pancreatitis is infrequent, but occasionally occurs without parotitis; the hyperglycemia is transient and is reversible.3 A causal relationship with mumps virus infection and has yet to be conclusively demonstrated.3
Deafness caused by mumps virus occurs in approximately 1 per 20,000 reported cases. Hearing loss is unilateral in approximately 80% of cases. Onset is usually sudden and results in permanent hearing impairment.³

**Key Points:**²

- The mumps is a paramyxovirus in the same group as parainfluenza and Newcastle disease virus.³
- Mumps is spread through airborne transmission or by direct contact with infected droplet nuclei or saliva.³ The spread of the mumps virus requires close and direct contact with an infected patient.⁵
- Immunization introduced in 1969
- The number of mumps cases has dropped from 34,000 per year in the early 1950’s to an average of 87 cases reported annually between 2000 and 2004.²
- Mumps will normally start with a prodromal phase. The prodromal symptoms are nonspecific, and include myalgia, anorexia, malaise, headache, and low-grade fever.³ After 1 or more days, the salivary glands become swollen and painful.⁵ The parotid gland (the largest salivary gland, located in front of the ear behind the angle of the jaw) is commonly affected.⁵ Parotitis is the most common manifestation and occurs in 30% to 40% of infected persons.
- Symptomatic meningitis occurs in up to 15% of patients and resolves without long-term sequelae in 3 to 10 days.³ Orchitis (testicular inflammation) is the most common complication in postpubertal males.³ Deafness caused by mumps virus occurs in approximately 1 per 20,000 reported cases.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the mumps vaccination. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#mumps

**Pertussis**

**Causal Agent**

Pertussis or whooping cough is caused by acute infection with *Bordetella pertussis*. It is a small aerobic gram-negative bacteria.³ *B. pertussis* produces multiple antigenic and biologically active products, including pertussis toxin, filamentous hemagglutinin, agglutinogens, adenylate cyclase, pertactin, and tracheal cytotoxin. These products are responsible for the clinical features of pertussis disease and an immune response to one or more produces immunity following infection.³

**Pathogenesis**

The bacteria attaches to the cells of the respiratory tract that have cilia.⁵ Pertussis toxin and adenylate cyclase and other toxins damage the cilia, cause inflammation of the respiratory tract and interfere with their normal function of transferring mucus from the airways to the mouth.⁵ Pertussis antigens appear to allow the organism to evade host defences and interfere with the normal function of white blood cells.⁵

**Transmission**

Transmission most commonly occurs by the respiratory route through contact with respiratory droplets, or by contact with airborne droplets of respiratory secretions.³ Pertussis is highly contagious with an estimated 9 out of 10 susceptible close contacts developing pertussis if in contact with an infected person in the home.⁵ Transmission occurs less frequently by contact with freshly contaminated articles of an infected person.⁵
Pertussis is most contagious during the first 2 weeks, when the patient is presenting with mild “cold-like” symptoms. This declines rapidly but may last for up to 3 weeks. Five days after antibiotics are initiated the patient is no longer contagious.

**Signs and Symptoms**

The incubation period of pertussis is commonly 7–10 days, with a range of 4–21 days. The first stage of a pertussis infection is characterized by the insidious onset of runny nose, sneezing, low-grade fever, and a mild, occasional cough, similar to the common cold. The cough gradually becomes more severe, and after 1–2 weeks, the second or paroxysmal stage begins. Fever is generally minimal throughout the course of the illness.

The next stage involves the patient developing bursts, or paroxysms, of numerous, rapid coughs, apparently due to difficulty expelling thick mucus from the tracheobronchial tree. At the end of the paroxysm, a long inspiratory effort is usually accompanied by a characteristic high-pitched whoop. During such an attack, the patient may become cyanotic (turn blue) and vomiting and exhaustion commonly follow the episode. This stage will usually last for 1 to 6 weeks but can persist for 10 weeks. The recovery is gradual with the cough becoming less common and disappearing in 2 to 3 weeks. However, paroxysms often recur with subsequent respiratory infections for many months after the onset of pertussis.

The most common complication, and the cause of most pertussis-related deaths, is secondary bacterial pneumonia. Neurologic complications such as seizures and encephalopathy may occur as a result of hypoxia (reduction of oxygen supply) from coughing, or possibly from toxin. Young infants are at highest risk for acquiring pertussis-associated complications. Other less serious complications of pertussis include otitis media, anorexia, and dehydration.

Approximately 20-30% of infants with pertussis require hospitalization and 1 in 400 hospitalized infants with pertussis die.

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**Practice Tool:**

Immunize BC has a great sound clip that can be used when counselling patients. It can be downloaded by clicking [here](#).

**Key Points:**

- Pertussis, or whooping cough, is an acute infectious disease caused by the bacterium *Bordetella pertussis*.
- Transmission most commonly occurs by the respiratory route through contact with respiratory droplets, or by contact with airborne droplets of respiratory secretions.
- Vaccine with whole cell pertussis began in the 1940’s and has been replaced by acellular vaccine in 1997-98.
- Prior to immunization, between 30,000 and 50,000 cases of pertussis occurred every year in Canada with 50-100 deaths. Most deaths occurred in infants less than 12 months of age.
- Symptoms of pertussis infection will normally start with a runny nose, sneezing, low-grade fever and a mild and occasional cough similar to a common cold. The next phase is the paroxysmal stage where the patient has bursts, or paroxysms, of numerous, rapid coughs, apparently due to difficulty expelling...
thick mucus from the tracheobronchial tree. At the end of the paroxysm, a long inspiratory effort is usually accompanied by a characteristic high-pitched whoop.³

- The most common complication, and the cause of most pertussis-related deaths, is secondary bacterial pneumonia.³ Approximately 20-30% of infants with pertussis require hospitalization and 1 in 400 hospitalized infants with pertussis die.⁵
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the pertussis vaccination. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#pertussis

**Pneumococcal Disease**

**Causal Agent**

*Streptococcus pneumoniae* bacteria are lancet-shaped, gram-positive, facultative anaerobic organisms.³ Capsular polysaccharides are the primary basis for the pathogenicity of the organism. They are antigenic and form the basis for classifying pneumococci by serotypes. Ninety serotypes have been identified.³

Most *S. pneumoniae* serotypes have been shown to cause serious disease, but only a few serotypes produce the majority of pneumococcal infections. The 10 most common serotypes are estimated to account for about 62% of invasive disease worldwide.³

**Pathogenesis**

Up to 40% of people of all ages are healthy carriers of pneumococci in their nasopharynx.⁵ There are 2 types of infection caused by pneumococci. These include local infections of the respiratory tract which remain on the surface and surrounding tissues and invasive infections that spread through the bloodstream to other organs.⁵ In young children illness occurs in 15% within 1 month of being infected with a new pneumococcus serotype.⁵

The spread of the bacteria from the nasopharynx to the blood results in bacteremia.⁵ Bacteremia without a known site of infection is the most common invasive clinical presentation of pneumococcal infection among children 2 years of age and younger, accounting for approximately 70% of invasive disease in this age group.³ In some patients, the bacteria overwhelms the defence system and leads to septicemia.⁵

**Transmission**

Transmission of *S. pneumoniae* occurs as the result of direct person-to-person contact via respiratory droplets and by autoinoculation in persons carrying the bacteria in their upper respiratory tract.³ The pneumococcal serotypes most often responsible for causing infection are those most frequently found in asymptomatic carriers.³ Fortunately pneumococcal infections are not highly contagious.⁵

Spread from an infected person to another requires close direct contact, through activities such as kissing, coughing and sneezing.⁵ The bacteria can also be spread through vectors with contaminated saliva such as cigarettes, lipstick and drinks.⁵

**Signs and Symptoms**
The major clinical syndromes of pneumococcal disease are pneumonia, bacteremia and meningitis.\(^3\)

Pneumococcal pneumonia is the most common clinical presentation among adults, although pneumonia alone is not considered to be “invasive” disease.\(^3\) The incubation period is short, about 1 to 3 days.\(^3\) Symptoms generally include an abrupt onset of fever and chills.\(^3\) Other common symptoms include chest pain, productive cough, rusty sputum, dyspnea, tachypnea, hypoxia, tachycardia, malaise and weakness.\(^3\) Pneumococci account for up to 36% of adult community-acquired pneumonia and 50% of hospital-acquired pneumonia.\(^3\) The case-fatality rate is 5%–7% and may be much higher among elderly persons.\(^3\)

Bacteremia occurs in about 25%–30% of patients with pneumococcal pneumonia. The overall case-fatality rate for bacteremia is about 20% but may be as high as 60% among elderly patients.\(^3\)

One-fourth of patients with pneumococcal meningitis also have pneumonia.\(^3\) Symptoms may include headache, lethargy, vomiting, irritability, fever, neck rigidity, cranial nerve signs, seizures and coma. The case-fatality rate of pneumococcal meningitis is about 30% but may be as high as 80% among elderly persons.\(^3\) Neurologic sequelae are common among survivors.\(^3\)

Among children 2 years of age and younger, bacteremia without a known site of infection is the most common invasive clinical presentation of pneumococcal infection, accounting for approximately 70% of cases.\(^3\) Among the same age group, bacteremic pneumonia accounts for 12%–16% of invasive pneumococcal disease.\(^3\)

Pneumococci are a common cause of acute otitis media, and are detected in 28%–55% of middle ear aspirates.\(^3\) By age 12 months, more than 60% of children have had at least one episode of acute otitis media.\(^3\) Complications of pneumococcal otitis media may include mastoiditis and meningitis.\(^3\)

**Key Points:**\(^2\)
- *Streptococcus pneumoniae* bacteria are lancet-shaped, gram-positive, facultative anaerobic organisms.
- Transmission of *S. pneumoniae* occurs as the result of direct person-to-person contact via respiratory droplets and by autoinoculation in persons carrying the bacteria in their upper respiratory tract.\(^3\) Up to 40% of people of all ages are healthy carriers of pneumococcus.\(^5\)
- *Streptococcus pneumoniae* (pneumococcus) is the leading cause of bacteremia, meningitis, bacterial pneumonia and acute otitis media in children. There are over 90 strains of pneumococci based on surface polysaccharides.
- Immunization began in the 1940’s had only 4 strains of pneumococcus. In 1983 a polysaccharide vaccine was launched containing polysaccharides from 23 strains of pneumococcus that cause 90% of serious infection in adults. This vaccine was not effective for children under 2 years of age (the group at highest risk of pneumococcal disease) but is routinely used adults, particularly the elderly and immunocompromised patients.\(^5\) In 2001 a conjugated vaccine was approved for use in children less than 2 years of age.
- Before the conjugated vaccine in 2001, approximately 500,000 cases of pneumococcal disease occurred every year in Canada. Of these, 65 children less than 2 years suffered meningitis, 700 had bacteremia, 2,200 had pneumonia and 200,000 had otitis media.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the pneumococcal vaccination. To find out more visit: [http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#pneumo](http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#pneumo)
Polio

Causal Agent

Poliovirus is a member of the enterovirus subgroup, family Picornaviridae. Enteroviruses are transient inhabitants of the gastrointestinal tract and are stable at acid pH.

Pathogenesis

The virus enters through the mouth and primary multiplication of the virus occurs at the site of implantation in the pharynx and the gastrointestinal tract. The virus is usually present in the throat and in the stool before the onset of illness. The virus invades local lymphoid tissue, enters the bloodstream, and then may infect cells of the central nervous system. Within the central nervous system the virus moves along nerve fibres and can lead to the destruction of motor neurons. Depending on the level of destruction, weakness or paralysis of muscles can occur.

Practice Tool:

- In 2007, Canada and Australia each had an imported paralytic poliomyelitis case occurring: a vaccine-associated one and a wild-type one, respectively.
- In 2009, poliomyelitis was still endemic in four countries: Nigeria, India, Pakistan and Afghanistan. Ongoing risk of importation of wild poliovirus remains.

Transmission

Poliovirus is highly infectious, with seroconversion rates among susceptible household contacts of children nearly 100%, and greater than 90% among susceptible household contacts of adults. The most important route of transmission is fecal-oral transmission through poor hygiene and inadequate sanitation. Oral to oral transmission through infected droplets is another route of transmission.

All infected persons will shed the virus regardless of symptoms. The virus sheds from the throat for 1 to 2 weeks after infection and from the intestinal tract for 4 to 8 weeks afterwards.

Signs and Symptoms

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

Approximately 90-95% of infected persons will have no symptoms. In 4-8% of patients they have mild non-specific symptoms such as fever, sore throat, myalgia, drowsiness, headache, anorexia, nausea, vomiting, abdominal pain and constipation.

Non-paralytic aseptic meningitis can occur in 1-2% of polio infections. Patients will report neck stiffness, severe headache, vomiting and lethargy or drowsiness. The illness lasts for 2 to 10 days and is followed by a
rapid and complete recovery.\textsuperscript{5}

Approximately 1 in every 100 persons infected will develop paralytic polio.\textsuperscript{5} Paralytic symptoms generally begin 1 to 10 days after prodromal symptoms and progress for 2 to 3 days.\textsuperscript{3} Prodromal Signs and Symptoms can include a loss of superficial reflexes, initially increased deep tendon reflexes and spasms in the limbs or back.\textsuperscript{3} The illness progresses to flaccid paralysis with diminished deep tendon reflexes, reaches a plateau without change for days to weeks, and is usually asymmetrical.\textsuperscript{3} Patients do not experience sensory losses or changes in cognition.\textsuperscript{3} Many persons with paralytic poliomyelitis recover completely and in most, muscle function returns to some degree. Weakness or paralysis still present 12 months after onset is usually permanent.\textsuperscript{3}

The death-to-case ratio for paralytic polio is generally 2%–5% among children and up to 15%–30% for adults (depending on age).\textsuperscript{3}

After an interval of 30–40 years, 25%–40% of persons who contracted paralytic poliomyelitis in childhood experience new muscle pain and exacerbation of existing weakness, or develop new weakness or paralysis.\textsuperscript{3} This is referred to as postpolio syndrome.\textsuperscript{3} Factors that increase the risk of postpolio syndrome include increasing length of time since acute poliovirus infection, presence of permanent residual impairment after recovery from the acute illness, and female sex.\textsuperscript{3} Postpolio syndrome is not an infectious process, and persons experiencing the syndrome do not shed poliovirus.\textsuperscript{3}

Key Points:\textsuperscript{2}
- Poliovirus is a member of the enterovirus subgroup, family Picornaviridae. Enteroviruses are transient inhabitants of the gastrointestinal tract, and are stable at acid pH.\textsuperscript{3}
- Person-to-person spread of poliovirus via the fecal-oral route is the most important route of transmission, although the oral-oral route may account for some cases.\textsuperscript{3}
- Vaccine introduced in 1955
- The last major epidemic of polio occurred in 1959 with nearly 2000 cases of paralytic polio.\textsuperscript{5} With effective immunization, Canada was certified polio-free in 1994.\textsuperscript{2}
- Up to 95% of all polio infections are asymptomatic.\textsuperscript{3} These patients will shed the virus in the stool and are able to transmit the virus to others.\textsuperscript{3} Approximately 4%–8% of polio infections consist of a minor and nonspecific illness.\textsuperscript{3} Nonparalytic aseptic meningitis (symptoms of stiffness of the neck, back, and/or legs), occurs in 1%–2% of polio infections. Less than 1% of all polio infections result in flaccid paralysis.\textsuperscript{3}
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the polio vaccine. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#polio

**Rotavirus**

**Causal Agent**

Rotavirus is a double-stranded RNA virus of the family *Reoviridae*.\textsuperscript{3} The outermost shell contains two important proteins—VP7, or G-protein, and VP4, or P-protein that allow for classification.\textsuperscript{3} Almost 90% of all infections are caused by four G types (G1,G2, G3,G4) in conjunction with P[4] or P[8].\textsuperscript{5}
Pathogenesis

The virus enters through the oral route and replicates in the villous epithelium of the small intestine. Infection may result in decreased intestinal absorption of sodium, glucose, and water, and decreased levels of intestinal lactase, alkaline phosphatase, and sucrase activity and may lead to isotonic diarrhea.

Recovery from a first rotavirus infection usually does not lead to permanent immunity. After a single natural infection, 38% of children are protected against any subsequent rotavirus infection, 77% are protected against rotavirus diarrhea, and 87% are protected against severe diarrhea. Reinfection can occur at any age. Subsequent infections confer progressively greater protection and are generally less severe than the first.

Rotavirus is a common cause of severe diarrhea. Infection with rotavirus in children in the United States found that 1 in 73 children had been hospitalized and 1 in 19 required medical care by 5 years of age because of rotavirus infection. In less developed countries, rotavirus diarrhea kills 1 in 100 children before their 5th birthday.

Transmission

Rotavirus is a very stable virus and can survive for prolonged periods on surfaces for weeks or months and resist being destroyed by many disinfectants. Rotaviruses are shed in high concentration in the stool of infected persons. Transmission is by fecal-oral spread, both through close person-to-person contact and through contaminated objects such as toys.

Clinical Note:

To prevent the spread of germs that cause diarrhea, proper hand washing and safe food handling remain most important.

Signs and Symptoms

The incubation period for rotavirus diarrhea is short, usually less than 48 hours. Rotavirus infection causes severe, watery diarrhea and high fever. Up to 20 bouts of vomiting and 20 episodes of diarrhea can occur in a single day. The gastrointestinal symptoms generally resolve in 3 to 7 days.

Rotavirus infection in infants and young children can lead to severe diarrhea, dehydration, electrolyte imbalance, and metabolic acidosis. If dehydration is severe and not adequately addressed death can occur.

Key Points:

- Rotavirus is a double-stranded RNA virus of the family Reoviridae.
- Transmission is by the fecal-oral route, both through close person-to-person contact and through contaminated objects such as toys.
- Rotavirus is a common cause of severe diarrhea. Infection with rotavirus in children in the United States found that 1 in 73 children had been hospitalized and 1 in 19 required medical care by 5 years of age because of rotavirus infection. From April 1993 to March 1995, the Alberta Children's Hospital saw 91 cases of rotavirus-associated diarrhea. In less developed countries, rotavirus diarrhea kills 1 in 100 children before their 5th birthday.
- Vaccine introduced in 2006.
Infection may be asymptomatic. It may cause self-limiting watery diarrhea, or may result in severe dehydrating diarrhea with fever and vomiting. Up to one-third of infected children may have a temperature greater than 102°F (39°C). The gastrointestinal symptoms generally resolve in 3 to 7 days.

Rotavirus infection in infants and young children can lead to severe diarrhea, dehydration, electrolyte imbalance, and metabolic acidosis. If dehydration is severe and not adequately addressed death can occur.

Rubella (German Measles)

Causal Agent

Rubella virus is classified as a togavirus, genus Rubivirus. It is an enveloped RNA virus, with a single antigenic type that does not cross-react with other members of the togavirus group. Rubella virus is relatively unstable and requires close contact to spread.

Pathogenesis

Once the infected droplets with the virus are inhaled, the virus first infects the cells lining the nasopharynx and travels to regional lymphnodes. Viremia occurs 5 to 7 days after exposure and the virus spreads to the skin, eyes, respiratory tract and other organs. Onset of the rash indicates the peak of infection and the beginning of recovery.

Transmission

Rubella is spread from person to person via airborne transmission or droplets shed from the respiratory secretions of infected persons. Spread of the virus requires close, direct contact between people. The virus is present in the nasopharynx of the infected person from about 7 days before the start of the rash to 2 weeks after. Rubella is not as contagious as other childhood infections such as measles and varicella.

Signs and Symptoms

The incubation period of rubella is 14 days, with a range of 12 to 23 days. Symptoms are often mild, and up to 50% of infections may be subclinical. In older children and adults, there is often a 1 to 5 day prodrome with low-grade fever, malaise, lymphadenopathy, and upper respiratory symptoms preceding the rash. The rash of rubella is maculopapular and occurs 14 to 17 days after exposure. The rash usually occurs initially on the face and then progresses from head to foot. It lasts about 3 days and is occasionally pruritic. Arthralgia and arthritis occur so frequently in adults that they are considered by many to be an integral part of the illness rather than a complication.

Complications of rubella are not common, but they generally occur more often in adults than in children. Arthralgia or arthritis may occur in up to 70% of adult women who contract rubella. Fingers, wrists, and knees are often affected. Joint symptoms tend to occur about the same time or shortly after appearance of the rash and may last for up to 1 month.

Encephalitis occurs in one in 6,000 cases, more frequently in adults (especially in females) than in children. Mortality estimates vary from 0 to 50%. Thrombocytopenia (reduction in platelet levels) occurs in
approximately 1 in 3000 cases and is more common in children. Increased bruising and hemorrhage risk is possible. This can last for days to months and most patients will recover.

Congenital rubella syndrome occurs when a pregnant women is infected with rubella sometime during the first 20 weeks of pregnancy. Infection may lead to fetal death, spontaneous abortion, or premature delivery. The virus may affect all organs and cause a variety of congenital defects. Approximately 85% of infants infected in the first trimester will be found to be affected if followed after birth.

The stage of pregnancy determines the degree of damage done by rubella infection. If the infection occurs in the first 12 weeks of pregnancy, the baby is born with multiple congenital anomalies, infection at 16-20 weeks deafness is the only major complication. Infection after 20 weeks does not affect the fetus. One in five fetal infections leads to severe malformations and fetal death. 1 in 10 babies dies of complications in the first 12 months of life.

Practice Tip:
In 2005 there was an outbreak of rubella, and close to 300 people were infected. This outbreak started at a religious school where only 40% of the children are immunized.

Key Points:
- Rubella virus is classified as a togavirus, genus *Rubivirus*.
- The rubella virus is spread through airborne transmission of infected droplets from the respiratory secretions of infected persons.
- Vaccine introduced in 1983
- The average number of cases dropped from an average of 5300 cases per year (between 1971-1982) to fewer than 30 cases per year (between 1998-2004)
- Symptoms are often mild, and up to 50% of infections may be subclinical. The rash of rubella is maculopapular and occurs 14 to 17 days after exposure. The rash usually occurs initially on the face and then progresses from head to foot. It lasts about 3 days and is occasionally pruritic.
- Arthralgia or arthritis may occur in up to 70% of adult women who contract rubella, but it is rare in children and adult males. Encephalitis occurs in 1 in 6,000 cases, more frequently in adults (especially in females) than in children. Mortality estimates vary from 0 to 50%.
- The most significant concern with rubella is infection in a pregnant patient. The virus may affect all organs and cause a variety of congenital defects. Infection may lead to fetal death, spontaneous abortion or premature delivery.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the rubella vaccination. To find out more visit: [http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#rubella](http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#rubella)

Tetanus

**Causal Agent**

Tetanus is an acute, often fatal, disease caused by an exotoxin produced by the bacterium *Clostridium tetani*. *C. tetani* is a slender, gram-positive, anaerobic rod that may develop a terminal spore, giving it a drumstick appearance. The organism is very sensitive to heat and oxygen but the spore is very resistant to
heat and can remain active for years in dust and soil.\textsuperscript{5} \textit{C. tetani} produces an exotoxin called tetanospasmin.\textsuperscript{3} On the basis of weight, tetanospasmin is one of the most potent toxins known.\textsuperscript{3}

**Pathogenesis**

\textit{C. tetani} spores usually enter the body through a wound. In the presence of anaerobic (low oxygen) conditions, the spores germinate.\textsuperscript{3} Toxins are produced and disseminated via the blood and lymphatic systems.\textsuperscript{3} Toxins act at several sites within the central nervous system.\textsuperscript{3} The typical clinical manifestations of tetanus are caused when tetanus toxin interferes with release of neurotransmitters, blocking inhibitor impulses. This leads to unopposed muscle contraction and spasm.\textsuperscript{3}

<table>
<thead>
<tr>
<th>Practice Tip:</th>
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<td><strong>Tetanus bacteria are present in soil, increasing the risk of serious disease in adults not immunized.</strong></td>
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**Transmission**

The spores of \textit{C. tetani} are widely distributed in soil and in the intestines and feces of horses, sheep, cattle, dogs, cats, rats, guinea pigs and chickens.\textsuperscript{3} The spores enter the body through a wound and it is not spread from person to person.\textsuperscript{3} It should be noted that most cases of tetanus are related to minor injuries and almost one-third of tetanus cases in North America occur in people who don’t report an injury.\textsuperscript{2}

**Signs and Symptoms**

Most cases of tetanus begin within 1 to 7 days of injury.\textsuperscript{5} The shorter the incubation period, the higher the chance of death.\textsuperscript{3} The most common form is generalized tetanus.\textsuperscript{3} The first sign is trismus or lockjaw, followed by stiffness of the neck, difficulty in swallowing, and rigidity of abdominal muscles. Other symptoms include elevated temperature, sweating, elevated blood pressure, and episodic rapid heart rate. Spasms may occur frequently and last for several minutes. Spasms continue for 3–4 weeks. Complete recovery may take months.\textsuperscript{3}

Even with treatment in an intensive care unit, the death rate from generalized tetanus is 10-20%.\textsuperscript{5} Cases most likely to be fatal are those occurring in persons 60 years of age and older and unvaccinated persons.\textsuperscript{3} It is important to recognize that immunization is still necessary for those individuals who have recovered from tetanus, as infection does not confer protective immunity.

**Key Points:**\textsuperscript{2}

- Tetanus is caused by an exotoxin produced by the bacterium \textit{Clostridium tetani}.\textsuperscript{3}
- The spores of \textit{C. tetani} are widely distributed in soil and in the intestines and feces of horses, sheep, cattle, dogs, cats, rats, guinea pigs and chickens.\textsuperscript{3} The spores enter the body through a wound and it is not spread from person to person.\textsuperscript{3}
- Vaccine introduced in 1940
- Tetanus is rare in Canada. Prior to immunization the number of deaths were 40 to 50 annually in Canada.\textsuperscript{2} After vaccine introduction, the cases dropped to 1 to 10 per year.\textsuperscript{2}
- The most common form is generalized tetanus. The condition presents with a descending pattern. The first sign is trismus or lockjaw, followed by stiffness of the neck, difficulty in swallowing, and rigidity of abdominal muscles. Other symptoms include elevated temperature, sweating, elevated blood
pressure and episodic rapid heart rate. Spasms may occur frequently and last for several minutes. Spasms continue for 3–4 weeks. Complete recovery may take months.3

- Tetanus is fatal in approximately 11% of reported cases. Cases most likely to be fatal are those occurring in persons 60 years of age and older (18%) and unvaccinated persons (22%).3
- The vaccine provides antitoxic protection and is not antibacterial. Therefore a patient can still be infected by the bacteria but is protected from the potentially lethal systemic effects.
- Active immunization is still required for persons who recover from tetanus as infection does not confer protective immunity.
- The CDC has some useful information to use in patient consultations on the consequences if we discontinued the tetanus vaccination. To find out more visit: http://www.cdc.gov/vaccines/vacc-gen/whatifstop.htm#tetanus

Varicella

Causal Agent

Varicella zoster virus is a DNA virus and is a member of the herpesvirus group.3 It has the capacity to persist in the body after the primary (first) infection as a latent infection.3 Varicella zoster virus persists in sensory nerve ganglia. Primary infection with varicella zoster virus results in chickenpox. Herpes zoster (shingles) is the result of recurrent infection.

Pathogenesis

Varicella zoster virus is highly contagious.5 It enters through the respiratory tract and conjunctiva. The virus is believed to replicate at the site of entry in the nasopharynx and in regional lymph nodes. A primary viremia occurs 4 to 6 days after infection and disseminates the virus to other organs, such as the liver, spleen, and sensory ganglia.3

Transmission

Varicella is highly contagious and spreads very easily through exposure of contaminated respiratory droplets containing the virus.5 The virus is shed in the highest amount 1 day before the appearance of the rash and decreases dramatically after the onset of the rash.5 In a household, chickenpox will spread to 60-85% of susceptible individuals.5

Patients with shingles (zoster) are also contagious only to persons who have never had chickenpox.5 This occurs through direct contact with the rash and very rarely through the air.3

Signs and Symptoms

In chickenpox, a mild prodrome may precede the onset of a rash.3 The rash is generalized and pruritic and progresses rapidly from macules to papules to vesicular lesions before crusting.3 Lesions are usually 1 to 4 mm in diameter. The vesicles are superficial and delicate and contain clear fluid on an erythematous base. Vesicles may rupture or become purulent before they dry and crust. Successive crops appear over several days, with lesions present in several stages of development on the body. The rash will normally start on the face and
scalp and progress down the body onto the arms and legs. The rash is more intense on the face and trunk than on the extremities. Healthy children usually have 200 to 500 lesions in 2 to 4 successive crops. Fever is more common in adults and they tend to have a more extensive rash.

Chicken pox causes an average of 5.8 deaths per year in Canada. In the paediatric age group, complications occur in 5-10% of healthy children with chicken pox. Secondary bacterial infections of skin lesions with *Staphylococcus* or *Streptococcus* are the most common cause of hospitalization and outpatient medical visits. Secondary infection with invasive group A streptococci may cause serious illness and lead to hospitalization or death.

Pneumonia and encephalitis (1 in 5000 cases) are complications with chicken pox. Cerebellar ataxia occurs in 1 in 4000 cases and is caused by inflammation in the cerebellum. It manifests as loss of muscle coordination so the child has a difficult time walking and performing voluntary movements.

The death rate from chickenpox is approximately 25 times higher in adults than children. Only 5% of all chickenpox cases occur in adults but 55-60% of all deaths from chickenpox are in this group. The main causes of death are from encephalitis and pneumonia.

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**Practice Tip:**

Risk of necrotising fasciitis (flesh-eating disease) is increased with varicella.

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Chickenpox during the first 20 weeks of pregnancy carries the risk that the virus will spread to the fetus. This may lead to congenital varicella syndrome. The risk is 0.4% if chickenpox occurs in the first trimester and 2.0% if in the second trimester. If the mother develops chickenpox around the time of birth (from 5 days before to 2 days after delivery), up to 30% of newborn infants will develop very severe chickenpox with multiple organ involvement. Approximately 1 in 5 infected newborns will die if not treated.

Reactivation of the varicella zoster virus that has remained latent in the nerve ganglia causes shingles. Over time aging, immunosuppression causes a reactivation of the latent varicella zoster virus. The virus will travel down the sensory nerve and causes a unilateral eruption on the skin. Most often, this involves the trunk or the fifth cranial nerve. Two to four days prior to the eruption, there may be pain and paresthesia in the involved area. When the rash appears it is followed by the formation of blisters. These blisters will break, forming small ulcers that begin to dry and form crusts. The crusts fall off in 2 to 3 weeks. Scarring is rare. There are few systemic symptoms.

Zoster can lead to postherpetic neuralgia, or pain in the area of the occurrence that persists after the lesions have resolved. Postherpetic neuralgia may last a year or longer after the episode of zoster. Ocular nerve involvement can often lead to severe sequelae such as blindness.

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**Key Points:**

- Varicella zoster virus is a DNA virus and is a member of the herpesvirus group.
- Varicella is highly contagious and spreads very easily through exposure of contaminated respiratory droplets containing the virus.
- Vaccine introduced in 1998
• Prior to immunization close to 95% of Canadians would be infected with the varicella zoster virus during their lifetime.³
• Most cases of varicella zoster infection are mild with a generalized rash appearing.³ The rash is pruritic and progresses rapidly from macules to papules to vesicular lesions before crusting.³
• Secondary bacterial infections of skin lesions with *Staphylococcus* or *Streptococcus* are the most common cause of hospitalization and outpatient medical visits.³ Secondary infection with invasive group A streptococci may cause serious illness and lead to hospitalization or death.³ Pneumonia and encephalitis are rare complications.
• Reactivation of varicella zoster virus in later life causes shingles
• The CDC has some useful information to use in patient consultations on the consequences if we discontinued the varicella vaccination. To find out more visit: http://www.cdc.gov/vaccines/vac-gen/whatifstop.htm#varicella

Please note that this is not an exhaustive list of all vaccine-preventable diseases. Participants should visit the Public Health Agency of Canada website for more information.

**Post Test**

*Mrs. Milette is in with her daughter Deborah to discuss the HPV vaccine. She mentions that she has been reading a fair amount about it and she has heard that this vaccine is really risky and the condition is not that serious anyway. She wants to know why we should immunize and actually promote our children to have sex. She has been your patient for a long time and she has always trusted your opinion. She would like you to discuss a bit more about this controversial disease.*

1. You start by discussing the cause of this infection. Which of the following statements is the MOST appropriate to use?
   a. This condition is caused by a virus and like you have read, it is normally transmitted by sexual intercourse
   b. The virus that causes the condition does not usually cause any serious problems but we immunize to protect our children
   c. The virus has 100 different types but it is only about 50 of them that tend to cause problems
   d. All of the above statements are appropriate to use

2. Mrs. Milette wonders if it would just be easier to wait to see if Deborah developed an infection and then treat it then. Which of the following is the MOST appropriate response?
   a. I think you are right, the vaccine is very expensive and I doubt that Deborah is at serious risk
   b. I think you are right, we usually can see the symptoms of this infection early and the treatment is highly effective
   c. I would encourage you to give her the immunization as the cancer it protects against kills 400 Canadian women every year
   d. I would encourage you to immunize, the experts say it is the right thing to do so you should do it

3. Mrs. Millette asks about the symptoms of the infection. Which of the following is the MOST appropriate response?
   a. Infection with this virus does not normally cause any symptoms
b. For most people, even infection with some of the cancer causing strains of this virus does not lead to problems
c. In some people the virus leads to chronic infection. This places the women at very high risk of cervical cancer
d. All of the above are appropriate responses

4. Mrs. Millette mentioned she never heard of this vaccine before the last couple of years. She wonders if it is just the pharmaceutical industry trying to make more money. She wants to know how common this condition really is. Which of the following statements is the most appropriate to use?
   a. About 20% of sexually active Canadians will be infected with HPV at some point in their life
   b. About 40% of sexually active Canadians will be infected with HPV at some point in their life
   c. About 60% of sexually active Canadians will be infected with HPV at some point in their life
   d. About 75% of sexually active Canadians will be infected with HPV at some point in their life

5. Approximately what percentage of sexual partners can become infected by a person with HPV?
   a. 10%
   b. 25%
   c. 65%
   d. 90%

6. Mrs. Millette never read that it caused cancer, only that it caused genital warts. What percentages of sexually active adults between the ages of 18 and 49 years have genital warts?
   a. 1%
   b. 5%
   c. 10%
   d. 20%

Mrs. Chang is in to see you for her son David’s second dose of tetanus-diphtheria-acellular pertussis IPV and Hib vaccine. She is questioning if this is really necessary. She has heard that Canada was classified as polio free and that diphtheria was only a concern back in the 20’s. Why should we immunize our children and place them at risk of adverse effects of vaccines when most of these conditions are minor or are essentially gone from Canada. You decide the best approach is to provide Mrs. Chang with a tidbit of information on each of the conditions in the vaccine you wish to administer.

7. You start with tetanus. Which of the following statements is the MOST appropriate to use when discussing tetanus with Mrs. Chang?
   a. Tetanus is real concern as it is easily transmittable through airborne transmission
   b. Even with treatment about 10% of tetanus cases are fatal
   c. The vaccine we give will help to protect your son from being infected with tetanus
   d. Most cases will have mild lock-jaw symptoms and we will see improvement in symptoms within a week

8. The next is diphtheria. Which of the following statements is the MOST appropriate to use when discussing diphtheria with Mrs. Chang?
   a. This bacteria is transmitted through contaminated clothing and through inhaling infected droplets in the air
   b. Close to 20% of Canadians do not have sufficient antibodies to protect against diphtheria
c. We have seen a resurgence in diphtheria in other parts of the world that when we stop immunizing
d. All of the above are appropriate to include

9. The next is pertussis. Which of the following statements is the MOST appropriate to use when discussing pertussis with Mrs. Chang?
   a. Prior to immunization there was 50-100 deaths per year in Canada from pertussis infection
   b. The patient will start with severe coughing then will develop a mild fever, runny nose and sneezing
   c. Approximately 5% of infants with pertussis require hospitalization
   d. 5 days of antibiotics will normally make the patient asymptomatic

10. The next is polio. Which of the following statements is the MOST appropriate to use when discussing polio with Mrs. Chang?
   a. There are still parts of the world where polio occurs regularly and with international travel it can resurge in this country if we stop using the vaccine
   b. Polio is not overly contagious but normally causes complications
   c. Only people showing symptoms can transmit the virus
   d. Polio is transmitted from inhaling infecting droplets in the air

11. The last is Hib. Which of the following statements is the MOST appropriate to use when discussing Hib with Mrs. Chang?
   a. Hib commonly causes otitis media and respiratory tract disorders
   b. Hib was the leading cause of meningitis prior to immunization
   c. About 1% of children with Hib meningitis die from the condition
   d. Hib is an age dependent condition and rarely affects any patient under the age of 10

References:


