

# Pertussis In Adults

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Pertussis, or whooping cough, is an acute infectious disease of the respiratory tract, caused by the bacterium *Bordetella pertussis*, or less frequently by *Bordetella parapertussis*.

In 1679, Sydenham gave the disease the name pertussis or violent cough. It is also known as whooping cough because of the classic paroxysmal cough. While outbreaks of pertussis were first described in the 16th century, the *B. pertussis* organism was first isolated in 1906 by Bordet and Gengou.

Their ager media is still in use as a selective media for the isolation of this fastidious organism.

In the United States, during the 20th century, pertussis was one of the most common childhood diseases and a major cause of childhood mortality. In the 1940s, more than 200,000 cases of pertussis and 7,500 resulting deaths were reported annually. For reasons discussed below, clinically diagnosed pertussis was rarely recognized in adults in the pre-vaccine and pre-antibiotic era. However, since 1997, pertussis among all age groups is on an upswing, and annual reported cases number in the tens of thousands (Figure 1.)

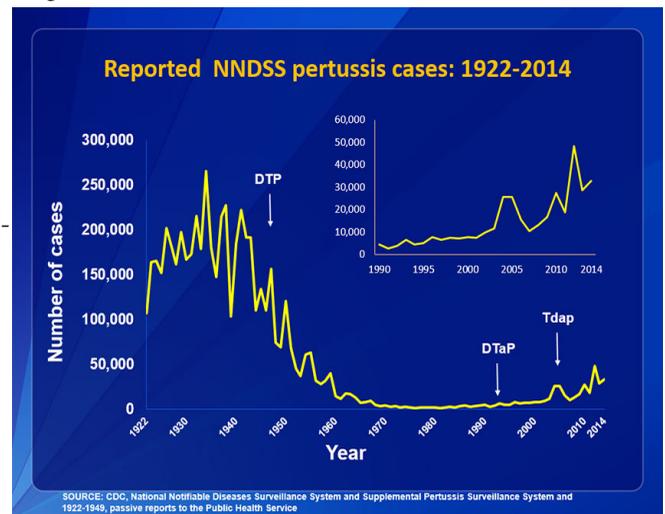
**Clinical Aspects:** Pertussis is primarily a toxin-mediated disease. The bacteria attach to the cilia of the respiratory epithelial cells, produce toxins that paralyze the cilia, and cause inflammation of the respiratory tract, which interferes with the clearing of pulmonary secretions.

The clinical course of the disease is divided into three stages: The first, the **catarrhal stage**, is characterized by the insidious onset of coryza (runny nose), sneezing, low-grade fever, and a mild, occasional cough, similar to the common cold. The cough gradually becomes more severe, and after 1-3 weeks, the second, or **paroxysmal stage**, begins. Fever is generally minimal throughout the course of the illness. During the paroxysmal stage, the diagnosis of pertussis is usually suspected. Characteristically, 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, marking a strong inspiratory effort. Respiratory secretions become thick, tenacious, and bubble out of the mouth and nose during this stage, which may last up to 4 weeks. During such an attack, the patient may become cyanotic (turn blue). Children especially, appear very ill and distressed. Vomiting and exhaustion commonly follow the episode. The person does not appear to be ill between attacks. Although symptoms in unvaccinated adults are generally milder, adults experiencing their first *B. pertussis* infection may have severe symptoms or even fatal disease.

In the third stage, the **convalescent stage**, recovery is gradual. The cough becomes less paroxysmal and disappears in 3 to 4 weeks. The total course of the disease often reaches 100 days, thus the term the “100-day cough”.

Complications associated with pertussis are important consideration in both children and adults. The respiratory complications, which may be associated with death, include pneumonia, respiratory failure, or pneumothorax. The hemorrhagic complications include severe epistaxis, intracranial bleed, and conjunctival or retinal bleeds. Neurologic complications include seizures and encephalopathy secondary to hemorrhage or hypoxia. All of these may be seen in adults as well as children with the diagnosis of pertussis, but the complication and mortality rates are much higher in infants than adults.

Figure 1



**Pre-Vaccine Era Adult Pertussis:** Pertussis disease was fairly rare in adults prior to the advent of the pertussis vaccine, in part because most individuals experienced their first and most symptomatic episode as infants or young children because of the highly contagious nature of the *B. pertussis* organism. In addition, then as now, second or third episodes were diagnosed as a “cold” or not diagnosed at all. However, because the immunity from pertussis disease was not life-long, there were reinfections and subsequent episodes of usually but not always, mild disease. There does not seem to be good data regarding the duration or degree of immunity from an episode of clinical pertussis.

Regardless of the clinical immunity provided by recovery from a previous episode of pertussis disease, those who are infected but asymptomatic or diagnosed as having a “cold” may transmit the disease to other susceptible persons, including unimmunized or incompletely immunized infants and children. Exposure of adults to the highly contagious *B. pertussis* organisms, (with an attack rate of between 80 and 100%) from infants and children, provided occult boost of their immunity to pertussis, thus in part explaining the rarity of pertussis disease in adults. It is probable that the continued low incidence in adults during the pre-vaccine era was due to herd immunity and low colonization rates in infants and young children.

**Pertussis Disease in the Whole-Cell Vaccine Era 1948-1997:** Clearly, the incidence of pertussis disease in all age groups markedly decreased after the advent of whole-cell pertussis vaccine in 1948. It should be noted that the efficacy of the whole-cell vaccine was never optimal, ranging between 40 and 80%. As was the case with immunity after natural pertussis disease, waning immunity after the whole-cell vaccine also occurred. Rough estimates of the duration of immunity suggest a range of 5 to 10 years, which is especially important in older individuals. Adult cases were recognized during the whole-cell vaccine era, perhaps at a higher incidence than during the pre-vaccine era, and transmissions from adults to infants and children were documented. In fact, there are many reports of healthcare professionals involved in the transmission of pertussis to their patients.

It should be noted that the pertussis vaccines do not protect against *B. parapertussis*. The overall incidence of pertussis disease began to increase in the mid 1980's when the immunization rate was in the 50 to 60% range. Most of the reported disease in infants and children occurred in un- or under-immunized individuals. Between 1990 and 1996, when the childhood immunization rate was above 90%, the incidence of reported pertussis disease in the adolescent and adult age groups increased almost 100%. The reason for this changing epidemiology remain unclear.

**Pertussis Disease in the Acellular Vaccine Era:** In 1997 because of the incidence of adverse reactions ascribed to the whole-cell pertussis vaccine (DPT), it was replaced in the United States by acellular pertussis vaccines (DTaP). Within 5 years, the incidence of reported pertussis disease in the United States began climbing in all age groups, but especially in infants and young children (Figure 1). This occurred in spite of immunization rates above 90%. Because of data suggesting that adolescents and young adults were experiencing more pertussis disease, and that these two age groups were a significant vector for spread to unimmunized or incompletely immunized infants, Tdap was licensed for adolescents and adults in 2005. In 2012, it was noted that case counts were increasingly elevated among children 7-10 years and among adolescents aged 13 and 14. This increase in susceptibility was seen in these two age groups who exclusively received the acellular pertussis vaccines for the primary and booster series.

The change in the epidemiology of pertussis in recent years, with an increasing burden of disease among fully-vaccinated children and adolescents, is likely being driven by the transition to acellular vaccines in 1997. Recent data suggest the efficacy of acellular vaccine of 75% during the first year decreases to 39% within three years. While symptomatic adult pertussis disease was unusual during the pre-vaccine era and remained fairly low during the whole cell pertussis vaccine era, we have yet to see what will happen during the acellular pertussis vaccine era.

## References:

1. Guris D, Strebel PM, Bardenheier B, et. al. Changing epidemiology of pertussis in the United States: increasing reported incidence among adolescents and adults, 1990-1996. *Clin Infect Dis.* 1999;28:1230-1237.
2. Cherry JD. Pertussis in the preantibiotic and prevaccine era, with emphasis on adult pertussis. *Clin Infect Dis.* 1999;28:S107-S111.
3. Centers for Disease Control and Prevention. Pertussis. In *The Pink Book* 13th Ed. CDC. Washington, DC. 2015.

## CME QUIZ

1. The reason for increasing incidence of pertussis since the 1980's is which one of the following:
  - a. Decreasing rate of immunization coverage
  - b. Increased diagnosis and reporting
  - c. Mutation of B. pertussis organisms
  - d. Waning immunity of vaccines
  - e. The reason remains unclear
2. During which era was the apparent incidence of pertussis disease in adults the lowest?
  - a. Paleolithic era
  - b. Pre-vaccine era
  - c. Whole cell vaccine era
  - d. Acellular vaccine era
  - e. It has been the same regardless of the era
3. What is the effectiveness of the current pertussis vaccine after 3 years?
  - a. >95%
  - b. 75 to 95%
  - c. 50 to 75%
  - d. 25 to 50%
  - e. <25%
4. The duration of immunity from whole cell pertussis vaccine is:
  - a. 1 year
  - b. 5 to 10 years
  - c. 10 to 15 years
  - d. 15 to 20 years
  - e. Lifelong
5. The most characteristic manifestation of pertussis disease is which one of the following?
  - a. Fever
  - b. Hemorrhagic complications
  - c. Hypoxic encephalopathy
  - d. Paroxysmal cough
  - e. Pneumonia
6. The paroxysmal stage of pertussis is characterized by all of the following except:
  - a. Coryza
  - b. High fever
  - c. Paroxysmal cough
  - d. Cough followed by a "whoop"
  - e. Copious tenacious bubbling mucous
7. The duration of a cough associated with pertussis disease may last as long as:
  - a. 10 days
  - b. 14 days
  - c. 21 days
  - d. 50 days
  - e. 100 days
8. Which one of the following adverse effects is caused by pertussis vaccine?
  - a. Autism
  - b. Autoimmune disease
  - c. Hypoxic encephalopathy
  - d. Low-grade fever
  - e. Pneumonia

Answers: 1.(E); 2.(B); 3.(D); 4.(B); 5.(D); 6.(B); 7.(E); 8.(D)



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