Pertussis was first recognized as an epidemic disease in the 16th century. The classic illness is a three-stage illness (catarrhal, spasmodic, and convalescent), with a distinctive cough, and its characteristics today are similar to those in the prevaccine era. In the prevaccine era, the calculated attack rate was 872/100,000 population, and the majority of cases occurred in children <5 years of age. On average, there were 7,300 deaths/year; the death rate began to decline before antimicrobial therapy and vaccination. Reported pertussis in adults was rare, but numerous investigators noted that atypical cases of pertussis were common in adults.

Pertussis (whooping cough) is an infectious illness of the respiratory tract, caused by *Bordetella pertussis* and less commonly by *Bordetella parapertussis* [1]. From 1922 through 1931 in the preantibiotic, prevaccine era, there were ~1.7 million reported cases and 73,000 deaths due to pertussis in the United States [2]. In comparison, during a recent 10-year period (1983–1992), there were 34,325 reported pertussis cases and 56 deaths [3].

In 1940, Holmes [4] commented on five peculiar features of pertussis, as follows: the disease is unlike other severe, classic contagious diseases (e.g., plague, smallpox) in that it has no ancient history; pathological examination reveals no characteristic histological changes; the paroxysmal cough of the illness is distinctive, but its mechanism is unknown; in contrast with other infectious illnesses, the death rate is higher in girls than in boys; and although it is an infectious disease, there is no fever or other physical findings during its spasmodic stage.

In this review, I examine clinical and epidemiological aspects of pertussis and look specifically at adult pertussis in the preantibiotic, prevaccine era.

**Etiology**

*B. pertussis* is the cause of endemic and epidemic pertussis throughout the world. Outbreaks of pertussis are also caused by *B. parapertussis*. Illnesses due to *B. pertussis* and *B. parapertussis* are similar, but those due to *B. parapertussis* tend to have a shorter duration [12]. Many other infectious agents cause illnesses with cough, and it is frequently stated that some of these can cause clinical pertussis. In particular, *Chlamydia trachomatis*, *Mycoplasma pneumoniae*, and certain adenoviruses have been implicated. However, the characteristics of the cough caused by these agents, as well as other clinical findings, are sufficiently different from those caused by *B. pertussis* during primary infections in children that clinical confusion should not occur; an exception is in mild and atypical *B. pertussis* illnesses occurring in previously infected or vaccinated older children and adults.

**Clinical Illness**

The classic description of pertussis was that of Guillaume de Baillou (Ballonius) in 1578 [4, 5]. There are several versions...
of this description because of the inaccuracies resulting from the translations from Latin to French to English. One version is as follows: "The lung is so irritated by every attempt to expel that which is causing the trouble it neither admits the air nor again expels it. The patient is seen to swell up and as if strangled, holds his breath tightly in the middle of his throat."

Classic pertussis today is a three-stage illness (catarrhal, spasmodic, and convalescent) with a duration of 4–8 weeks or longer [1]. The catarrhal stage is characterized by the onset of coryza, slight temperature elevation, and the onset of cough. It is similar to the typical common cold. However, by the second week, the cough becomes paroxysmal. During the paroxysmal stage, there is no fever or other systemic symptoms or signs. Absolute lymphocytosis is common, and the paroxysmal cough is frequently associated with posttussive whoop and vomiting [13]. In recent studies, we have found that 17%–25% of unvaccinated children have illnesses due to B. pertussis that are of relatively short duration (≤3 weeks) [13, 14]. The most important complications today are pneumonia, seizures, apnea, and cyanosis, otitis media, poor feeding, and encephalopathy [1, 13].

In the prevaccine era, clinical pertussis was remarkably similar to the illness seen today. However, some findings were more pronounced, and complications were more frequent [15].

The symptoms of pertussis are usually divided into three stages—the catarrhal, the spasmodic, and the stage of decline.

The catarrhal stage continues on the average for about ten days, although cases show considerable variation on this point. Some children whoop almost from the very beginning of the disease, while others may cough for three to four weeks before a typical whoop is noticed. The symptoms in the beginning are indistinguishable from those of an ordinary attack of subacute tracheo-bronchitis, and unless there has been an exposure to pertussis no suspicion is excited. After five or six days, however, the cough, instead of abating as in an ordinary cold, gradually increases in severity and occurs in paroxysms. At first these are mild, and there are only two or three a day, but they gradually increase in frequency and severity until the typical whoop is heard which marks the beginning of the spasmodic stage. During the first stage there may be symptoms of a mild grade of catarrhal inflammation of the nose, pharynx, and larynx, and often there is a slight elevation of temperature.

The spasmodic stage.—In a typical paroxysm of average severity the child, who can usually foretell it, will often run for support to the lap of the mother or nurse, or seize a chair with both hands. There now occurs a series of explosive coughs, from ten to twenty in number, coming in such rapid succession that the child cannot get its breath between them; the face becomes of a deep red or purple colour, sometimes almost black; the veins of the face and scalp stand out prominently; the eyes are suffused, and seem almost to start from their sockets; there follows a long-drawn inspiration through the narrowed glottis, producing the cowing sound known as the whoop; and then another succession of rapid coughs follows and another whoop. In a single severe paroxysm, which lasts two or three minutes, the child may whoop half a dozen times; with the final paroxysm a mass of tenacious mucus is usually brought up. In a young child vomiting is almost certain to follow, if food has been recently taken. Epistaxis sometimes occurs with nearly every paroxysm, but in most cases the bleeding is slight. After a severe attack the child is at times so exhausted as to be hardly able to stand; there is profuse perspiration; his mind is confused, and he may be completely dazed. In infants the attack may result in a degree of asphyxia requiring artificial respiration. Those old enough to describe their sensations tell of a sense of impending suffocation, the suffering from which is almost indescribable.

The number of severe paroxysms or "kinks" in twenty-four hours varies, according to the severity of the case, from half a dozen to forty or fifty. There are always many more of a milder form. Paroxysms are often excited by eating or drinking anything cold, by a draught of air, or by imitation; they are usually more frequent during the night than during the day, and in a close room than in open air.

In less severe cases no paroxysms of the grade above described may occur, and no typical whoop may be heard throughout the attack; but the paroxysmal nature of the cough which continues until the plug of mucus is expelled, the watery eyes, and the vomiting which follows a paroxysm, stamp the disease as pertussis. In young infants the whoop is frequently not marked. The child sometimes coughs until it is asphyxiated, and yet no whoop occurs. The paroxysms are also modified by intercurrent disease, especially by attacks of pneumonia or severe bronchitis. At such times they usually become less frequent and less typical, and may be absent for several days, returning as the complication subsides.

The average duration of the spasmodic stage is about one month. It increases in intensity for the first two weeks, remains stationary for about one week, and then gradually diminishes in severity. The course and duration of this stage are, however, subject to wide variations. In mild cases it may last only a week; in severe cases, especially in the winter season, it may continue for three months, at times almost subsiding, but lighting up again with all its previous severity with every fresh attack of cold. After it has entirely ceased the whoop may return with an attack of bronchitis, and continue for a month or more. This is not to be regarded as a true relapse of pertussis. The habit of paroxysmal cough once established, it tends to recur with every slight bronchitis, often for months afterward.

The stage of decline.—Gradually the severity of the paroxysms abates, the whoop ceases, and the cough resembles more and more that of ordinary bronchitis. This stage usually continues about three weeks, but may be prolonged indefinitely in the winter months.

Holt [15] discussed complications under four categories: hemorrhages, respiratory system, digestive system, and nervous system. Hemorrhagic complications included epistaxis, conjunctival hemorrhages, and those involving the CNS. It appears that hemorrhagic complications are much less common today than in the prevaccine era. Holt noted that the most serious complications were respiratory, with pneumonia being the leading cause of death. Holt also noted that during the summer,
infants with pertussis nearly always had accompanying diarrhea. This is not a problem today. Infants with severe posttussive vomiting and poor food intake often developed malnutrition. Nervous system complications included "convulsions, coma, paralysis, aphasia, disturbances of sight or hearing, and in rare cases even of the mental condition."

**Epidemiology: Prevacine Era**

The attack and death rates of reported pertussis cases/100,000 population from 1922 to 1945 in the United States are presented in figure 1. The average attack rate (1932–1941) was 157/100,000 population, and data from Massachusetts in the 1940s and from Maryland in the 1920s suggested that only ~18% of cases were reported [9, 16]. Therefore, the calculated attack rate was 872/100,000 population. Epidemic peaks occurred about every 3 years.

The age distribution of pertussis in selected areas of the United States in the prevaccine era is presented in table 1. In New York City, Luttinger [17] noted that 80% of cases were observed in children <5 years of age. In Maryland and Massachusetts, 56% of the cases were observed in children <5 years of age. In all three geographic areas, ~3% of the reported cases were observed in persons ≥15 years of age.

During the period from 1922 through 1931, there were on average 7,300 deaths/year due to pertussis [2]. The vast majority of deaths occurred in infants, and it was noted by Mortimer and Jones [19] that the yearly death rate was declining in the prevaccine era. In the period 1900–1904, the death rate in infants was 4.34 per 1,000, and it fell to 0.86 per 1,000 in the period 1940–1944. The age distributions of pertussis deaths in Manhattan and the Bronx from 1866 to 1915 [17] and in whites in the United States from 1917 to 1923 [20] are presented in table 2. As can be seen, >95% of the deaths occurred in children <5 years of age. Interestingly, a small number of deaths were reported in adolescents and adults.

A remarkable feature of pertussis is that the attack rate in unvaccinated populations is higher in females than males [16]. In the period 1945–1949 in England and Wales, the percentage of cases of pertussis in females by age was as follows: <1 year, 51%; 1–4 years, 52%; 5–9 years, 53%; 10–14 years, 55%; and ≥15 years, 74%.

In 1929, Collins [20] reported the percentage of persons by age who had had an attack of pertussis. For this survey, ~43,000 subjects (or their parents) of different ages were questioned as to whether they had had pertussis. The percentage of positivity increased linearly (9%/year) until 7 years. The curve then flattened out and was level at age 13, with 77% having a history of an attack of pertussis. At all ages from 13 to 37 years, ~23% of those surveyed did not have a history of pertussis.

### Table 1. Age distribution of pertussis in selected areas of the United States in the prevaccine era.

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>%</th>
<th>Age (y)</th>
<th>%</th>
<th>Age (y)</th>
<th>%</th>
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</thead>
<tbody>
<tr>
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<td>19.4</td>
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<td>. .</td>
<td>.</td>
<td>10.3</td>
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<td>. .</td>
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<tr>
<td>2–4</td>
<td>40.1</td>
<td>0–4</td>
<td>56</td>
<td>1–4</td>
<td>44.6</td>
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<tr>
<td>5–14</td>
<td>17.9</td>
<td>5–9</td>
<td>35</td>
<td>5–9</td>
<td>38.5</td>
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<tr>
<td>≥15</td>
<td>2.3</td>
<td>10–14</td>
<td>6</td>
<td>10–14</td>
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<td>. . . .</td>
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<tr>
<td>. . .</td>
<td>.</td>
<td>≥20</td>
<td>2</td>
<td>. . .</td>
<td>.</td>
</tr>
</tbody>
</table>

### Table 2. Age distribution of deaths due to pertussis in Manhattan and the Bronx (1866–1915) and in whites in the United States (1917–1923).

<table>
<thead>
<tr>
<th>Age</th>
<th>No.</th>
<th>%</th>
<th>Age</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
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<td>1</td>
<td>4,659</td>
<td>27.3</td>
<td>. . . .</td>
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<tr>
<td>2–4</td>
<td>3,213</td>
<td>18.8</td>
<td>&lt;5</td>
<td>46,065</td>
<td>95.1</td>
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<tr>
<td>5–14</td>
<td>471</td>
<td>2.7</td>
<td>5–9</td>
<td>1,834</td>
<td>3.8</td>
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<td>≥15</td>
<td>35</td>
<td>0.3</td>
<td>10–14</td>
<td>233</td>
<td>0.5</td>
</tr>
<tr>
<td>. . .</td>
<td>.</td>
<td>.</td>
<td>15–19</td>
<td>85</td>
<td>0.2</td>
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<td>. . .</td>
<td>.</td>
<td>.</td>
<td>≥20</td>
<td>246</td>
<td>0.5</td>
</tr>
</tbody>
</table>

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**Figure 1.** Reported cases of pertussis (solid line) and deaths (dashed line) due to pertussis per 100,000 population by year (1922 to 1945) in the United States (modified from [9]).
Pertussis in Adults: Prevaccine Era

As noted in table 1, reported pertussis in persons >14 years of age was rare. In a study of childhood contagious diseases in American soldiers during World War I, Holmes [4] noted only 109 cases of pertussis, whereas there were 221,060 cases of mumps and 93,629 cases of measles. In 1931, Stallybrass [21] investigated second attacks of pertussis. He noted in a 1902 study by Laing and Hay that only 0.26% of 20,405 cases were second attacks. He also noted that in an island population in which pertussis occurred after a 29-year absence, no cases occurred in persons >29 years of age.

Although survey data in the prevaccine era showed little evidence of adult pertussis, many writers of the time suggested that atypical cases in adults were not uncommon. Luttinger [17] in 1916 described an adult who spread pertussis in three different family settings (table 3). In a 1925 report, Madsen [22] discussed pertussis in the Faroe Islands in 1914 and 1915: “It is worthy of note that many of the substantiated cases of whooping cough were second attacks so called ‘grandmothers whooping cough’; however, these were always light and shorter in duration than the first attacks.”

In his 1943 book, Lapin [5] discussed adult pertussis on several occasions. Several vignettes are listed below:

Whooping cough is so ubiquitous that often, when the source of infection remains unknown, it may be assumed that the disease was transmitted through uncharacteristic unrecognized cases. Huehennmann, Kristensen (loc cit.) and others found H. pertussis in patients with abortive whooping cough. Clinical experience shows abortive whooping cough, unrecognized except by cough plate, as a frequent finding. It has been observed in older children, or adults with a recurrence of the disease, or children who had been vaccinated against pertussis. Mild or missed cases in adults, the so-called “formes frustes,” have been demonstrated by many investigators. Rosenau (loc cit.) estimates these cases as constituting three to five per cent of the total cases of whooping cough, and they may be important epidemiologic problem.

Zischinsky on the other hand, reported one infant with a second and a third attack of whooping cough, which were severe, and resulted in pulmonary complications.

Kramer considers the immunity resulting from one attack of whooping cough similar to that gained by immunization, and the milder characters of the second attacks similar in both cases.

Luttinger [17] in 1916 discussed adult pertussis and second attacks. His notations were as follows: “Eight patients undoubtedly were infected by the kiss of adults whose children had the whooping cough but had never been in contact with these patients, as they lived in different parts of the city. All these adults had a little cough, which lasted for 5 or 6 weeks. . . . Of 811 atypical cases of pertussis 69 (9%) had had pertussis before. . . . Of 9400 typical cases of pertussis with whoop only 18 were second attacks.”

In 1934, Mannerstedt [23] wrote about adult pertussis, as follows: “Often older children and adults develop persistent coughs without any definite paroxysms and whoops, which are probably mild grades of whooping cough. . . . Hennes states that his 4 children contracted whooping cough from an adult and he contracted it in an atypical form. . . . Schwenkenbecher describes an epidemic of pertussis which ran through a connecting group of 5 offices. Most had atypical illness but carried disease to their families.”

Mannerstedt’s conclusions were as follows: adult pertussis occurs more frequently than generally assumed; second attacks are more frequent than commonly believed; illness starts with insidious cough 1–3 weeks after exposure, lasts 5–6 weeks or longer, and is worse at night; gagging and choking are common and thick, white, tenacious phlegm is raised; a blood cell count does not reveal characteristic findings.

According to Friedlander [24]: “The disease may occur at all ages however, and adults may contract the disease. It even occurs in old age.” In addition, “adults who have been exposed may show such mild paroxysms, without definite whoop, that the diagnosis is difficult.”

Kristenson [25], in 1933, wrote: “In all, among 202 children (up to 15 years) whooping cough occurred in 116, and in 40 of these (i.e., about 35 per cent) it took an abortive course. In 183 adults there were 5 cases of whooping 4 of these being abortive. Thus, it is reasonable to assume that abortive cases of whooping cough, often not recognized, are very frequent. As a rule, they occur in older children or adults, in persons who have had whooping cough before, or in patients who have been vaccinated against this disease.”

Kramer [26], in 1939, had the following conclusions: “Pertussis vaccine (Sauer) confers a relative immunity within a period of two years from the time of administration. Immunity resulting from the use of pertussis vaccine (Sauer) is as effective as a previous attack of the disease in conferring immunity upon children. An attack of pertussis does not confer complete immunity from a mild recurrence of the disease.”

From a vaccine efficacy study in a children’s home, Kramer [27] observed that “there was exposure in one unit with 29 children with 12 nonvaccinated, 9 vaccinated, and 8 previous pertussis. All 12 nonvaccinated developed typical pertussis, 6 of 9 vaccinees had mild cough lasting 3 weeks, and 8 previous pertussis had mild coughing spells for about 3 weeks.”

Hess [28] quotes Dr. Kate C. Meade as saying “‘My 12th case was a man, aged 76, who had typical pertussis and pneu-

Table 3. Spread of pertussis in three different family settings by an adult (“Pertussis Pete”) [17].

1. Peter G. boarded with his sister in Harlem.
2. Two nieces and one nephew contracted whooping cough. Peter began to cough a few weeks later.
3. Beginning of March: Peter visited another sister in Brooklyn, and 8 days later her children developed pertussis.
4. Peter went to live with brother; a week later, the brother’s child developed pertussis.
5. Peter moved to a cousin’s house, and shortly thereafter, a neighbor’s child developed pertussis.
6. April 20: Peter sailed for Italy, having enlisted in the army.

H. pertussis
monia” to which Dr. Frank W. Pinneo responded “Dr. Meade’s case reminds me of an old lady who contracted whooping-cough last summer from a little child.”

Summary and Conclusions

Pertussis in the prevaccine era was an endemic and epidemic disease with a high mortality. Characteristics of the illness are the same today as they were in the prevaccine era. Pertussis mortality was declining before the introduction of vaccines or antimicrobial agents. Although precise quantitative data are lacking, the clinical case data available indicate that reinfec-
tions in adults in the prevaccine era were common and atypical disease frequent. Furthermore, the same clinical case data sug-
gram that the perception that infection resulted in long-lasting immunity was due to constant reexposure and unrecognized atypical illness, and immunity from disease was actually short-
lived.

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compared with illness caused 

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pertussis 


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