The most common complication of pertussis is pneumonia caused by a superinfecting organism such as *Streptococcus pneumoniae*. Atelectasis is also common but may be recognized only by radiologic examination. Other complications, including convulsions and subconjunctival or cerebral bleeding, are related to the venous pressure effects of the paroxysmal coughing and the anoxia produced by inadequate ventilation and apneic spells.

**DIAGNOSIS**

A clinical diagnosis of pertussis is best confirmed by isolation of *B pertussis* from nasopharyngeal secretions or swabs. Throat swabs are not suitable, because the cilia to which the organism attaches are not found there. Specimens collected early in the course of disease (during the catarrhal or early paroxysmal stage) provide the greatest chance of successful isolation. Unfortunately, the diagnosis is frequently not considered until paroxysmal coughing has been present for some time, and the number of organisms has decreased significantly. The nasopharyngeal specimens are plated onto a special charcoal blood agar medium made selective by the addition of a cephalosporin. This allows the slow-growing *B pertussis* to be isolated in the presence of more rapidly growing members of the normal upper respiratory flora. The characteristic colonies appear after 3 to 7 days of incubation and look like tiny drops of mercury. Immunologic methods (agglutination, immunofluorescence) are required for specific identification.

A direct immunofluorescent antibody (DFA) technique has been successfully applied to nasopharyngeal smear for rapid diagnosis of pertussis. DFA is particularly helpful in pertussis because of the many days required for culture results. Because the sensitivity and specificity of DFA can vary with the quality of the reagents, these results should always be confirmed by culture, if possible. Nucleic acid amplification tests have been developed, which show potential for being highly sensitive but are not yet practical for most laboratories. Serologic tests are widely used for epidemiologic studies but not for diagnosis of individual clinical cases. :: DFA, p. 66

**TREATMENT**

Once the paroxysmal coughing stage has been reached, the treatment of pertussis is primarily supportive. Antimicrobial therapy is useful at earlier stages and for limiting spread to other susceptible individuals. Of a number of antimicrobics active in vitro against *B pertussis*, the macrolides are preferred for treatment. Of these, erythromycin has the greatest experience demonstrating its clinical effectiveness and relative lack of toxicity.

**PREVENTION**

Active immunization is the primary method of preventing pertussis. The original vaccine, which produced a dramatic reduction in disease, was prepared from inactivated whole cell suspensions and given together with diphtheria and tetanus toxoids as DTP. The undoubted efficacy of this vaccine was colored by a high rate of side effects due to the crude nature of the whole cell preparation. These included local inflammation, fever and, rarely, febrile seizures. Although permanent neurologic sequelae were never convincingly linked to pertussis immunization, there were those who argued that the vaccine was worse than the disease. This led to the development of acellular vaccines, guided by knowledge of the pathogenesis of pertussis. These vaccines contain virulence factors purified from whole cell preparations inactivated where appropriate. Another vaccine strategy is the production of recombinant components, genetically engineered to be immunogenic but nontoxic.

The multiple acellular vaccines have different combinations of virulence factors. All contain PT and FHA, and some add pertactin or pili (vaccine manufacturers use the term fimbriae). The efficacy of these vaccines has now been established, and all have dramatically less frequent side effects compared with the whole cell preparations. In combination with diphtheria and tetanus toxoids, they have now replaced the whole cell DTP as DTaP (“a” for acellular). This vaccine is now recommended for the full primary immunization (at 2, 4,