folliculitis. Other differential diagnostic considerations include the various infections causing genital ulceration, such as primary syphilis, secondary syphilis (condyloma latum), genital herpes, and donovanosis. In rare cases, chancroid lesions become secondarily infected with bacteria; the result is extensive inflammation.

DIAGNOSIS

Clinical diagnosis of chancroid is often inaccurate, and laboratory confirmation should be attempted in suspected cases. An accurate diagnosis of chancroid relies on culture of *H. ducreyi* from the lesion or from an aspirate of suppurative lymph nodes. Since the organism can be difficult to grow, the use of selective and supplemented media is necessary. No polymerase chain reaction assay for *H. ducreyi* is commercially available; such tests can be performed by Clinical Laboratory Improvement Amendment (CLIA)–certified clinical laboratories that have developed their own assays.

A probable diagnosis of chancroid can be made when the following criteria are met: (1) one or more painful genital ulcers; (2) no evidence of *Treponema pallidum* infection by dark-field examination of ulcer exudate or by a negative serologic test for syphilis performed at least 7 days after ulcer onset; (3) a typical clinical presentation for chancroid; and (4) a negative test for herpes simplex virus in the ulcer exudate.

TREATMENT *HAEMOPHILUS DUCREYI*

Treatment regimens recommended by the Centers for Disease Control and Prevention include (1) a single 1-g oral dose of azithromycin; (2) ceftriaxone (250 mg intramuscularly in a single dose); (3) ciprofloxacin (500 mg by mouth twice a day for 3 days); and (4) erythromycin base (500 mg by mouth three times a day for 7 days). Isolates from patients who do not respond promptly to treatment should be tested for antimicrobial resistance. In patients with HIV infection, healing may be slow and longer courses of treatment may be necessary. Clinical treatment failure in HIV-seropositive patients may reflect co-infection, especially with herpes simplex virus. Contacts of patients with chancroid should be identified and treated, whether or not symptoms are present, if they have had sexual contact with the patient during the 10 days preceding the patient's onset of symptoms.

MORAXELLA CATARRHALIS

MICROBIOLOGY

M. catarrhalis is an unencapsulated gram-negative diplococcus whose ecologic niche is the human respiratory tract. The organism was initially designated *Micrococcus catarrhalis*. Its name was changed to *Neisseria catarrhalis* in 1970 because of phenotypic similarities to commensal *Neisseria* species. On the basis of more rigorous analysis of genetic relatedness, *Moraxella catarrhalis* is now the widely accepted name for this species.

EPIDEMIOLOGY

Nasopharyngeal colonization by *M. catarrhalis* is common in infancy, with colonization rates ranging between 33% and 100% and depending on geographic location. Several factors probably account for this geographic variation, including living conditions, day-care attendance, hygiene, household smoking, and population genetics. The prevalence of colonization decreases steadily with age.

The widespread use of pneumococcal conjugate vaccines in some countries has resulted in alterations in patterns of nasopharyngeal colonization in resident populations. A relative increase in colonization by nonvaccine pneumococcal serotypes, nontypable *H. influenzae*, and *M. catarrhalis* has occurred. These changes in colonization patterns may be altering the distribution of pathogens of both otitis media and sinusitis in children.

PATHOGENESIS

M. catarrhalis causes mucosal infections of the respiratory tract by contiguous spread from its colonizing site in the upper airway. A preceding viral upper respiratory tract infection is a common inciting event for otitis media. In exacerbations of COPD, the acquisition of new strains is critical for pathogenesis. Strains exhibit substantial genetic diversity and differences in virulence properties.

The expression of several adhesin molecules with differing specificities for various host cell receptors reflects the importance of adherence to the respiratory epithelial surface in the pathogenesis of infection. *M. catarrhalis* invades multiple cell types. Its intracellular residence in lymphoid tissue provides a potential reservoir for persistence in the human respiratory tract. Like many gram-negative bacteria, *M. catarrhalis* sheds vesicles into the surrounding environment. The vesicles are internalized by host cells and mediate several virulence mechanisms, including induction of inflammation and delivery of β-lactamase, that can promote the survival of co-pathogens.

CLINICAL MANIFESTATIONS

In children, *M. catarrhalis* causes predominantly mucosal infections when the bacterium migrates from the nasopharynx to the middle ear or the sinuses (Chap. 44). The inciting event for both otitis media and sinusitis is often a preceding viral infection. Overall, cultures of middle-ear fluid obtained by tympanocentesis indicate that *M. catarrhalis* causes 15–20% of cases of acute otitis media. Acute otitis media caused by *M. catarrhalis* or nontypable *H. influenzae* is clinically milder than otitis media caused by *S. pneumoniae*, with less fever and a lower prevalence of a red bulging tympanic membrane. However, substantial overlap makes it impossible to predict etiology in an individual child on the basis of clinical features.

A small proportion of viral upper respiratory tract infections are complicated by bacterial sinusitis. Cultures of sinus puncture aspirates show that *M. catarrhalis* accounts for ~20% of cases of acute bacterial sinusitis in children and for a smaller proportion in adults.

M. catarrhalis is a common cause of exacerbations in adults with COPD. The bacterium has been overlooked in this clinical setting because it has long been considered to be a commensal and because it is easily mistaken for commensal *Neisseria* species in cultures of respiratory secretions (see "Diagnosis," below). Several independent lines of evidence have established *M. catarrhalis* as a pathogen in COPD. These include (1) the demonstration of *M. catarrhalis* in the lower airways during exacerbations, (2) the association of exacerbation with acquisition of new strains, (3) elevations of inflammatory markers in association with *M. catarrhalis*, and (4) the development of specific immune responses following infection. *M. catarrhalis* is the second most common bacterial cause of COPD exacerbations (after *H. influenzae*), as shown in a 10-year prospective study; the distribution of exacerbations associated with new-strain acquisitions is shown in Fig. 182-3. Not included are culture-negative cases or cases from which a pathogen had been previously isolated. With the application of rigorous clinical criteria for defining the etiology of exacerbations (both culture-positive and culture-negative), ~10% of all exacerbations in the same study were caused by *M. catarrhalis*. The clinical features of an exacerbation due to *M. catarrhalis* are similar to those of exacerbations due to other bacterial pathogens, including *H. influenzae* and *S. pneumoniae*. The cardinal symptoms are cough with increased sputum production, sputum purulence, and dyspnea in comparison with baseline symptoms.

Pneumonia due to *M. catarrhalis* occurs in the elderly, particularly in the setting of underlying cardiopulmonary disease, but is infrequent. Invasive infections, such as bacteremia, endocarditis, neonatal meningitis, and septic arthritis, are rare.

DIAGNOSIS

Tympanocentesis is required for etiologic diagnosis of otitis media, but this procedure is not performed routinely. Therefore, treatment of otitis media is generally empirical. Similarly, an etiologic diagnosis of sinusitis requires an invasive procedure and thus is usually not available to the clinician. Isolation of *M. catarrhalis* from an expectorated sputum sample from an adult experiencing clinical symptoms of an exacerbation is suggestive, but not diagnostic, of *M. catarrhalis* as the cause.