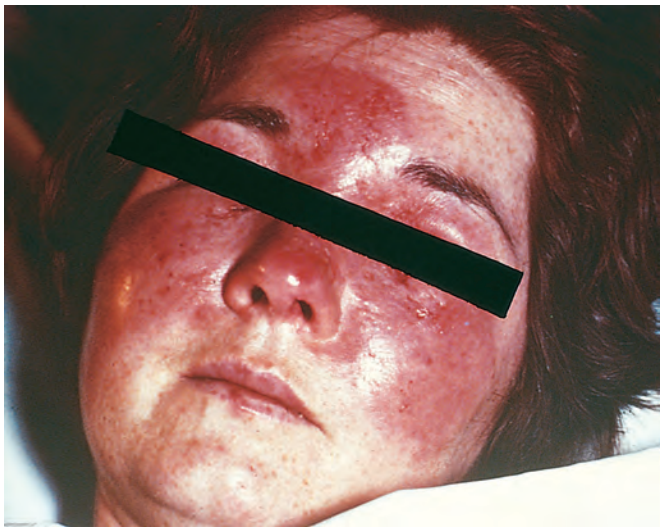

Figure 8-17 Streptococcal erysipelas.

injure host tissues. The emergence of enterococci as pathogens is primarily due to their resistance to antibiotics.

MORPHOLOGY

Streptococcal infections are characterized by diffuse interstitial neutrophilic infiltrates with minimal destruction of host tissues. The skin lesions caused by streptococci (furuncles, carbuncles, and impetigo) resemble those of staphylococci.

Erysipelas is caused by exotoxins from superficial infection with *S. pyogenes*. It is characterized by rapidly spreading erythematous cutaneous swelling that may begin on the face or, less frequently, on the body or an extremity. The rash has a sharp, well-demarcated, serpiginous border and may form a "butterfly" distribution on the face (Fig. 8-17). On histologic examination there is a diffuse, edematous, neutrophilic inflammatory reaction in the dermis and epidermis extending into the subcutaneous tissues. Microabscesses may be formed, but tissue necrosis is usually minor.

Streptococcal pharyngitis, which is the major antecedent of poststreptococcal glomerulonephritis (Chapter 20), is marked by edema, epiglottic swelling, and punctate abscesses of the tonsillar crypts, sometimes accompanied by cervical lymphadenopathy. Swelling associated with severe pharyngeal infection may encroach on the airways, especially if there is peritonsillar or retropharyngeal abscess formation.

Scarlet fever, associated with pharyngitis caused by *S. pyogenes*, is most common between the ages of 3 and 15 years. It is manifested by a punctate erythematous rash that is most prominent over the trunk and inner aspects of the arms and legs. The face is also involved, but usually a small area about the mouth remains relatively unaffected, producing circumoral pallor. The skin usually becomes hyperkeratotic and scaly during defervescence.

S. pneumoniae is an important cause of lobar pneumonia (described in Chapter 15 and pictured in Fig. 8-4).

Diphtheria

Diphtheria is caused by *Corynebacterium diphtheriae*, a slender gram-positive rod with clubbed ends that spreads from person to person in respiratory droplets or skin exudate. Respiratory diphtheria causes pharyngeal or, less often, nasal or laryngeal infection. There is toxin-mediated formation of a gray pharyngeal membrane, and damage to the heart, nerves, and other organs. Cutaneous diphtheria causes chronic ulcers with a dirty gray membrane, but does not cause systemic damage. *C. diphtheriae* produces a phage-encoded A-B toxin that blocks host cell protein synthesis. The A fragment does this by catalyzing the covalent transfer of adenosine diphosphate (ADP)-ribose to elongation factor-2 (EF-2). This inhibits EF-2 function, which is required for the translation of mRNA into protein. A single molecule of diphtheria toxin can kill a cell by ADP-ribosylating, and thereby inactivating, more than a million EF-2 molecules. Immunization with diphtheria toxoid (formalin-fixed toxin) stimulates production of toxin-neutralizing antibodies that protect people from the lethal effects of the toxin.

MORPHOLOGY

Inhaled *C. diphtheriae* carried in respiratory droplets proliferate at the site of attachment on the mucosa of the nasopharynx, oropharynx, larynx, or trachea. The bacteria also form satellite lesions in the esophagus or lower airways. Release of exotoxin causes necrosis of the epithelium, accompanied by an outpouring of a dense fibrinosuppurative exudate. The coagulation of this exudate on the ulcerated necrotic surface creates a tough, dirty gray to black, superficial membrane, sometimes called **pseudo-membrane** because it is not formed by viable tissue (Fig. 8-18). There is an intense neutrophilic infiltration in the underlying tissues with marked vascular congestion, interstitial edema, and fibrin exudation. When the membrane sloughs off its inflamed and vascularized bed, bleeding and asphyxiation may occur. With control of the infection, the membrane is coughed up or removed by enzymatic digestion, and the inflammatory reaction subsides.

Although the bacterial invasion remains localized, with entry of exotoxin into the blood and its systemic distribution, there may be fatty change in the myocardium with isolated myofiber necrosis, polyneuritis with degeneration of the myelin sheaths and axis cylinders, and (less commonly) fatty change and focal necroses of parenchymal cells in the liver, kidneys, and adrenals.

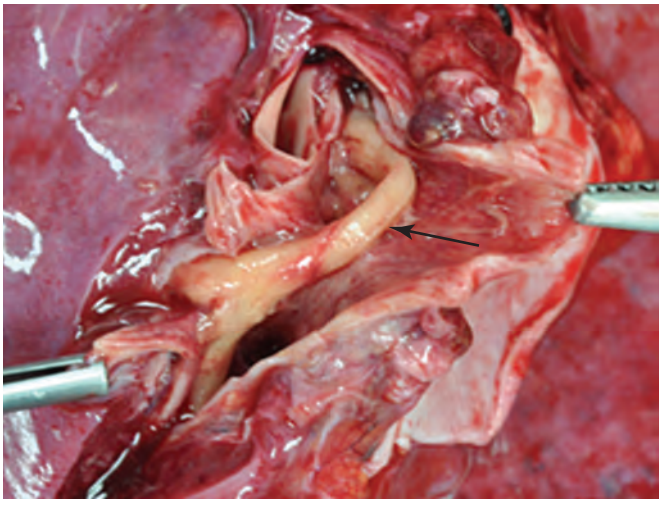

Figure 8-18 Membrane of diphtheria *(arrow)* lying within a transverse bronchus. (Courtesy Dr. Robin A. Cooke, Department of Anatomical Pathology, Princess Alexandria Hospital, Brisbane, Australia.)