Alternative agents are erythromycin and azithromycin. Azithromycin is more expensive but offers the advantages of better gastrointestinal tolerability, once-daily dosing, and a 5-day treatment course. Resistance to erythromycin and other macrolides is common among isolates from several countries, including Spain, Italy, Finland, Japan, and Korea. Macrolide resistance may be becoming more prevalent elsewhere with the increasing use of this class of antibiotics. In areas with resistance rates exceeding 5–10%, macrolides should be avoided unless results of susceptibility testing are known.

Follow-up culture after treatment is no longer routinely recommended but may be warranted in selected cases, such as those involving patients or families with frequent streptococcal infections or those occurring in situations in which the risk of ARF is thought to be high (e.g., when cases of ARF have recently been reported in the community).

COMPLICATIONS Suppurative complications of streptococcal pharyngitis have become uncommon with the widespread use of antibiotics for most symptomatic cases. These complications result from the spread of infection from the pharyngeal mucosa to deeper tissues by direct extension or by the hematogenous or lymphatic route and may include cervical lymphadenitis, peritonsillar or retropharyngeal abscess, sinusitis, otitis media, meningitis, bacteremia, endocarditis, and pneumonia. Local complications, such as peritonsillar or parapharyngeal abscess formation, should be considered in a patient with unusually severe or prolonged symptoms or localized pain associated with high fever and a toxic appearance. Nonsuppurative complications include ARF (Chap. 381) and PSGN (Chap. 338), both of which are thought to result from immune responses to streptococcal infection. Penicillin treatment of streptococcal pharyngitis has been shown to reduce the likelihood of ARF but not that of PSGN.

BACTERIOLOGIC TREATMENT FAILURE AND THE ASYMPTOMATIC CARRIER STATE Surveillance cultures have shown that up to 20% of individuals in certain populations may have asymptomatic pharyngeal colonization with GAS. There are no definitive guidelines for management of these asymptomatic carriers or of asymptomatic patients who still have a positive throat culture after a full course of treatment for symptomatic pharyngitis. A reasonable course of action is to give a single 10-day course of penicillin for symptomatic pharyngitis and, if positive cultures persist, not to re-treat unless symptoms recur. Studies of the natural history of streptococcal carriage and infection have shown that the risk both of developing ARF and of transmitting infection to others is substantially lower among asymptomatic carriers than among individuals with symptomatic pharyngitis. Therefore, overly aggressive attempts to eradicate carriage probably are not justified under most circumstances. An exception is the situation in which an asymptomatic carrier is a potential source of infection to others. Outbreaks of food-borne infection and nosocomial puerperal infection have been traced to asymptomatic carriers who may harbor the organisms in the throat, vagina, or anus or on the skin.

TREATMENT ASYMPTOMATIC PHARYNGEAL COLONIZATION WITH GAS

When a carrier is transmitting infection to others, attempts to eradicate carriage are warranted. Data are limited on the best regimen to clear GAS after penicillin alone has failed. Regimens reported to have efficacy superior to that of penicillin alone for eradication of carriage include (1) oral clindamycin (7 mg/kg; 300 mg maximum) three times daily for 10 days or (2) penicillin (as recommended for treatment of pharyngitis in Table 173-3) plus oral rifampin (10 mg/kg; 300 mg maximum) twice daily for the first 4 days of treatment. A 10-day course of oral vancomycin (250 mg four times daily) and rifampin (600 mg twice daily) has eradicated rectal colonization.

Scarlet Fever Scarlet fever consists of streptococcal infection, usually pharyngitis, accompanied by a characteristic rash (Fig. 173-2).

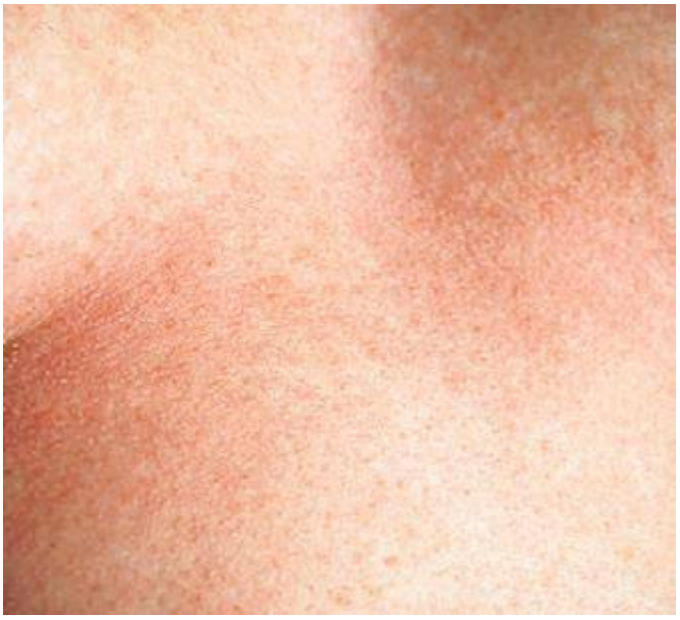

FIGURE 173-2 Scarlet fever exanthem. Finely punctate erythema has become confluent (scarlatiniform); petechiae can occur and have a linear configuration within the exanthem in body folds (Pastia's lines). *(From Fitzpatrick, Johnson, Wolff: Color Atlas and Synopsis of Clinical Dermatology, 4th ed, New York, McGraw-Hill, 2001, with permission.)*

The rash arises from the effects of one of several toxins, currently designated *streptococcal pyrogenic exotoxins* and previously known as *erythrogenic* or *scarlet fever toxins*. In the past, scarlet fever was thought to reflect infection of an individual lacking toxin-specific immunity with a toxin-producing strain of GAS. Susceptibility to scarlet fever was correlated with results of the Dick test, in which a small amount of erythrogenic toxin injected intradermally produced local erythema in susceptible individuals but elicited no reaction in those with specific immunity. Subsequent studies have suggested that development of the scarlet fever rash may reflect a hypersensitivity reaction requiring prior exposure to the toxin. For reasons that are not clear, scarlet fever has become less common in recent years, although strains of GAS that produce pyrogenic exotoxins continue to be prevalent in the population. The symptoms of scarlet fever are the same as those of pharyngitis alone. The rash typically begins on the first or second day of illness over the upper trunk, spreading to involve the extremities but sparing the palms and soles. The rash is made up of minute papules, giving a characteristic "sandpaper" feel to the skin. Associated findings include circumoral pallor, "strawberry tongue" (enlarged papillae on a coated tongue, which later may become denuded), and accentuation of the rash in skinfolds (*Pastia's lines*). Subsidence of the rash in 6–9 days is followed after several days by desquamation of the palms and soles. The differential diagnosis of scarlet fever includes other causes of fever and generalized rash, such as measles and other viral exanthems, Kawasaki disease, TSS, and systemic allergic reactions (e.g., drug eruptions).

Skin and Soft Tissue Infections GAS—and occasionally other streptococcal species—can cause a variety of infections involving the skin, subcutaneous tissues, muscles, and fascia. While several clinical syndromes offer a useful means for classification of these infections, not all cases fit exactly into one category. The classic syndromes are general guides to predicting the level of tissue involvement in a particular patient, the probable clinical course, and the likelihood that surgical intervention or aggressive life support will be required.

IMPETIGO (PYODERMA) Impetigo, a superficial infection of the skin, is caused primarily by GAS and occasionally by other streptococci or *Staphylococcus aureus*. Impetigo is seen most often in young children, tends to occur during warmer months, and is more common in semitropical or tropical climates than in cooler regions. Infection is more