cells that are diagnostic of this disease. Widespread acute eruptions of erythematous papules are discussed in the section on exanthems.

There are several infectious diseases that present as erythematous papules or nodules in a lymphocutaneous or sporotrichoid pattern, i.e., in a linear arrangement along the lymphatic channels. The two most common etiologies are *Sporothrix schenckii* (sporotrichosis) and the atypical mycobacterium *Mycobacterium marinum*. The organisms are introduced as a result of trauma, and a primary inoculation site is often seen in addition to the lymphatic nodules. Additional causes include *Nocardia*, *Leishmania*, and other atypical mycobacteria and dimorphic fungi; culture of lesional tissue will aid in the diagnosis.

The diseases that are characterized by erythematous plaques with scale are reviewed in the papulosquamous section, and the various forms of dermatitis are discussed in the section on erythroderma. Additional disorders in the differential diagnosis of red papules/plaques include *cellulitis*, *polymorphous light eruption* (PMLE), *cutaneous lymphoid hyperplasia* (lymphocytoma cutis), *cutaneous lupus*, *lymphoma cutis*, and *leukemia cutis*. The first three diseases represent primary cutaneous disorders, although cellulitis may be accompanied by a bacteremia. PMLE is characterized by erythematous papules and plaques in a primarily sun-exposed distribution—dorsum of the hand, extensor forearm, and upper trunk. Lesions follow exposure to UV-B and/or UV-A, and in higher latitudes, PMLE is most severe in the late spring and early summer. A process referred to as "hardening" occurs with continued UV exposure, and the eruption fades, but in temperate climates, it will recur in the spring. PMLE must be differentiated from cutaneous lupus, and this is accomplished by observation of the natural history, histologic examination, and direct immunofluorescence of the lesions. Cutaneous lymphoid hyperplasia (pseudolymphoma) is a *benign* polyclonal proliferation of lymphocytes in the skin that presents as infiltrated pink-red to red-purple papules and plaques; it must be distinguished from lymphoma cutis.

Several types of red plaques are seen in patients with systemic *lupus*, including (1) erythematous urticarial plaques across the cheeks and nose in the classic butterfly rash; (2) erythematous discoid lesions with fine or "carpet-tack" scale, telangiectasias, central hypopigmentation, peripheral hyperpigmentation, follicular plugging, and atrophy located on the face, scalp, external ears, arms, and upper trunk; and (3) psoriasiform or annular lesions of subacute cutaneous lupus with hypopigmented centers located primarily on the extensor arms and upper trunk. Additional mucocutaneous findings include (1) a violaceous flush on the face and V of the neck; (2) photosensitivity; (3) urticarial vasculitis (see "Urticaria," above); (4) lupus panniculitis (see below); (5) diffuse alopecia; (6) alopecia secondary to discoid lesions; (7) periungual telangiectasias and erythema; (8) EM-like lesions that may become bullous; (9) oral ulcers; and (10) distal ulcerations secondary to Raynaud's phenomenon, vasculitis, or livedoid vasculopathy. Patients with only discoid lesions usually have the form of lupus that is limited to the skin. However, up to 10% of these patients eventually develop systemic lupus. Direct immunofluorescence of involved skin, in particular discoid lesions, shows deposits of IgG or IgM and C3 in a granular distribution along the dermal-epidermal junction.

In *lymphoma cutis*, there is a proliferation of malignant lymphocytes in the skin, and the clinical appearance resembles that of cutaneous lymphoid hyperplasia—infiltrated pink-red to red-purple papules and plaques. Lymphoma cutis can occur anywhere on the surface of the skin, whereas the sites of predilection for lymphocytomas include the malar ridge, tip of the nose, and earlobes. Patients with non-Hodgkin's lymphomas have specific cutaneous lesions more often than those with Hodgkin's disease, and, occasionally, the skin nodules precede the development of extracutaneous non-Hodgkin's lymphoma or represent the only site of involvement (e.g., primary cutaneous B cell lymphoma). Arcuate lesions are sometimes seen in lymphoma and lymphocytoma cutis as well as in CTCL. *Adult T cell leukemia/lymphoma* that develops in association with HTLV-1 infection is characterized by cutaneous plaques, hypercalcemia, and circulating CD25+ lymphocytes. *Leukemia cutis* has the same appearance as lymphoma cutis, and specific lesions are seen more commonly in monocytic leukemias than in lymphocytic or granulocytic leukemias. Cutaneous chloromas (granulocytic sarcomas) may precede the appearance of circulating blasts in acute myelogenous leukemia and, as such, represent a form of aleukemic leukemia cutis.

Sweet syndrome is characterized by pink-red to red-brown edematous plaques that are frequently painful and occur primarily on the head, neck, and upper (and, less often, lower) extremities. The patients also have fever, neutrophilia, and a dense dermal infiltrate of neutrophils in the lesions. In ~10% of the patients, there is an associated malignancy, most commonly acute myelogenous leukemia. Sweet syndrome has also been reported with inflammatory bowel disease, systemic lupus erythematosus, and solid tumors (primarily of the genitourinary tract) as well as drugs (e.g., all-*trans*-retinoic acid, granulocyte colony-stimulating factor [G-CSF]). The differential diagnosis includes neutrophilic eccrine hidradenitis; bullous forms of pyoderma gangrenosum; and, occasionally, cellulitis. Extracutaneous sites of involvement include joints, muscles, eye, kidney (proteinuria, occasionally glomerulonephritis), and lung (neutrophilic infiltrates). The idiopathic form of Sweet syndrome is seen more often in women, following a respiratory tract infection.

Common causes of erythematous subcutaneous nodules include inflamed epidermoid inclusion cysts, acne cysts, and furuncles. *Panniculitis*, an inflammation of the fat, also presents as subcutaneous nodules and is frequently a sign of systemic disease. There are several forms of panniculitis, including erythema nodosum, erythema induratum/nodular vasculitis, lupus panniculitis, lipodermatosclerosis, α_1-antitrypsin deficiency, factitial, and fat necrosis secondary to pancreatic disease. Except for erythema nodosum, these lesions may break down and ulcerate or heal with a scar. The shin is the most common location for the nodules of erythema nodosum, whereas the calf is the most common location for lesions of erythema induratum. In erythema nodosum, the nodules are initially red but then develop a blue color as they resolve. Patients with erythema nodosum but no underlying systemic illness can still have fever, malaise, leukocytosis, arthralgias, and/or arthritis. However, the possibility of an underlying illness should be excluded, and the most common associations are streptococcal infections, upper respiratory viral infections, sarcoidosis, and inflammatory bowel disease, in addition to drugs (oral contraceptives, sulfonamides, penicillins, bromides, iodides). Less common associations include bacterial gastroenteritis (*Yersinia*, *Salmonella*) and coccidioidomycosis followed by tuberculosis, histoplasmosis, brucellosis, and infections with *Chlamydophila pneumoniae* or *Chlamydia trachomatis, Mycoplasma pneumoniae,* or hepatitis B virus.

Erythema induratum and nodular vasculitis have overlapping features clinically and histologically, and whether they represent two separate entities or the ends of a single disease spectrum is a point of debate; in general, the latter is usually idiopathic and the former is associated with the presence of *Mycobacterium tuberculosis* DNA by PCR within skin lesions. The lesions of lupus panniculitis are found primarily on the cheeks, upper arms, and buttocks (sites of abundant fat) and are seen in both the cutaneous and systemic forms of lupus. The overlying skin may be normal, erythematous, or have the changes of discoid lupus. The subcutaneous fat necrosis that is associated with pancreatic disease is presumably secondary to circulating lipases and is seen in patients with pancreatic carcinoma as well as in patients with acute and chronic pancreatitis. In this disorder, there may be an associated arthritis, fever, and inflammation of visceral fat. Histologic examination of deep incisional biopsy specimens will aid in the diagnosis of the particular type of panniculitis.

Subcutaneous erythematous nodules are also seen in cutaneous polyarteritis nodosa and as a manifestation of *systemic vasculitis* when there is involvement of medium-sized vessels, e.g., systemic polyarteritis nodosa, allergic granulomatosis, or granulomatosis with polyangiitis (Wegener's) (Chap. 385). Cutaneous polyarteritis nodosa presents with painful subcutaneous nodules and ulcers within a red-purple, netlike pattern of livedo reticularis. The latter is due to slowed blood flow through the superficial horizontal venous plexus. The majority of lesions are found on the lower extremities, and while arthralgias and myalgias may accompany cutaneous polyarteritis nodosa, there is no evidence of systemic involvement. In both the cutaneous and systemic