forms of vasculitis, skin biopsy specimens of the associated nodules will show the changes characteristic of a necrotizing vasculitis and/or granulomatous inflammation.

RED-BROWN LESIONS

The cutaneous lesions in *sarcoidosis* (Chap. 390) are classically red to red-brown in color, and with diascopy (pressure with a glass slide), a yellow-brown residual color is observed that is secondary to the granulomatous infiltrate. The waxy papules and plaques may be found anywhere on the skin, but the face is the most common location. Usually there are no surface changes, but occasionally the lesions will have scale. Biopsy specimens of the papules show "naked" granulomas in the dermis, i.e., granulomas surrounded by a minimal number of lymphocytes. Other cutaneous findings in sarcoidosis include annular lesions with an atrophic or scaly center, papules within scars, hypopigmented papules and patches, alopecia, acquired ichthyosis, erythema nodosum, and lupus pernio (see below).

The differential diagnosis of sarcoidosis includes foreign-body granulomas produced by chemicals such as beryllium and zirconium, late secondary syphilis, and *lupus vulgaris*. Lupus vulgaris is a form of cutaneous tuberculosis that is seen in previously infected and sensitized individuals. There is often underlying active tuberculosis elsewhere, usually in the lungs or lymph nodes. Lesions occur primarily in the head and neck region and are red-brown plaques with a yellow-brown color on diascopy. Secondary scarring and squamous cell carcinomas can develop within the plaques. Cultures or PCR analysis of the lesions should be performed, along with an interferon γ release assay of peripheral blood, because it is rare for the acid-fast stain to show bacilli within the dermal granulomas.

A generalized distribution of red-brown macules and papules is seen in the form of mastocytosis known as *urticaria pigmentosa* (Chap. 376). Each lesion represents a collection of mast cells in the dermis, with hyperpigmentation of the overlying epidermis. Stimuli such as rubbing cause these mast cells to degranulate, and this leads to the formation of localized urticaria (Darier's sign). Additional symptoms can result from mast cell degranulation and include headache, flushing, diarrhea, and pruritus. Mast cells also infiltrate various organs such as the liver, spleen, and gastrointestinal tract, and accumulations of mast cells in the bones may produce either osteosclerotic or osteolytic lesions on radiographs. In the majority of these patients, however, the internal involvement remains indolent. A subtype of chronic cutaneous small-vessel vasculitis, *erythema elevatum diutinum* (EED), also presents with papules that are red-brown in color. The papules coalesce into plaques on the extensor surfaces of knees, elbows, and the small joints of the hand. Flares of EED have been associated with streptococcal infections.

BLUE LESIONS

Lesions that are blue in color are the result of vascular ectasias, hyperplasias and tumors or melanin pigment within the dermis. *Venous lakes* (ectasias) are compressible dark-blue lesions that are found commonly in the head and neck region. *Venous malformations* are also compressible blue papulonodules and plaques that can occur anywhere on the body, including the oral mucosa. When there are multiple rather than single congenital lesions, the patient may have the blue rubber bleb syndrome or Maffucci's syndrome. Patients with the blue rubber bleb syndrome also have vascular anomalies of the gastrointestinal tract that may bleed, whereas patients with Maffucci's syndrome have associated osteochondromas. *Blue nevi* (moles) are seen when there are collections of pigment-producing nevus cells in the dermis. These benign papular lesions are dome-shaped and occur most commonly on the dorsum of the hand or foot or in the head and neck region.

VIOLACEOUS LESIONS

Violaceous papules and plaques are seen in *lupus pernio*, *lymphoma cutis*, and *cutaneous lupus*. Lupus pernio is a particular type of sarcoidosis that involves the tip and alar rim of the nose as well as the earlobes, with lesions that are violaceous in color rather than red-brown. This form of sarcoidosis is associated with involvement of the upper respiratory tract. The plaques of lymphoma cutis and cutaneous lupus may be red or violaceous in color and were discussed above.

PURPLE LESIONS

Purple-colored papules and plaques are seen in vascular tumors, such as *Kaposi's sarcoma* (Chap. 226) and *angiosarcoma*, and when there is extravasation of red blood cells into the skin in association with inflammation, as in *palpable purpura* (see "Purpura," below). Patients with congenital or acquired AV fistulas and venous hypertension can develop purple papules on the lower extremities that can resemble Kaposi's sarcoma clinically and histologically; this condition is referred to as pseudo-Kaposi's sarcoma (acral angiodermatitis). Angiosarcoma is found most commonly on the scalp and face of elderly patients or within areas of chronic lymphedema and presents as purple papules and plaques. In the head and neck region, the tumor often extends beyond the clinically defined borders and may be accompanied by facial edema.

BROWN AND BLACK LESIONS

Brown- and black-colored papules are reviewed in "Hyperpigmentation," above.

CUTANEOUS METASTASES

These are discussed last because they can have a wide range of colors. Most commonly, they present as either firm, skin-colored subcutaneous nodules or firm, red to red-brown papulonodules. The lesions of lymphoma cutis range from pink-red to plum in color, whereas metastatic melanoma can be pink, blue, or black in color. Cutaneous metastases develop from hematogenous or lymphatic spread and are most often due to the following primary carcinomas: in men, melanoma, oropharynx, lung, and colon; and in women, breast, melanoma, and ovary. These metastatic lesions may be the initial presentation of the carcinoma, especially when the primary site is the lung.

PURPURA

(Table 72-16) *Purpura* are seen when there is an extravasation of red blood cells into the dermis and, as a result, the lesions do not blanch with pressure. This is in contrast to those erythematous or violet-colored lesions that are due to localized vasodilatation—they do blanch with pressure. Purpura (≥3 mm) and petechiae (≤2 mm) are divided into two major groups: palpable and nonpalpable. The most frequent causes of *nonpalpable* petechiae and purpura are primary cutaneous disorders such as *trauma*, *solar* (*actinic*) *purpura*, and *capillaritis*. Less common causes are *steroid purpura* and *livedoid vasculopathy* (see "Ulcers," below). Solar purpura are seen primarily on the extensor forearms, whereas steroid purpura secondary to potent topical glucocorticoids or endogenous or exogenous Cushing's syndrome can be more widespread. In both cases, there is alteration of the supporting connective tissue that surrounds the dermal blood vessels. In contrast, the petechiae that result from capillaritis are found primarily on the lower extremities. In capillaritis, there is an extravasation of erythrocytes as a result of perivascular lymphocytic inflammation. The petechiae are bright red, 1–2 mm in size, and scattered within yellow-brown patches. The yellow-brown color is caused by hemosiderin deposits within the dermis.

Systemic causes of nonpalpable purpura fall into several categories, and those secondary to clotting disturbances and vascular fragility will be discussed first. The former group includes *thrombocytopenia* (Chap. 140), *abnormal platelet function* as is seen in uremia, and *clotting factor defects*. The initial site of presentation for thrombocytopenia-induced petechiae is the distal lower extremity. Capillary fragility leads to nonpalpable purpura in patients with systemic *amyloidosis* (see "Papulonodular Skin Lesions," above), disorders of collagen production such as *Ehlers-Danlos syndrome*, and *scurvy*. In scurvy, there are flattened corkscrew hairs with surrounding hemorrhage on the lower extremities, in addition to gingivitis. Vitamin C is a cofactor for lysyl hydroxylase, an enzyme involved in the posttranslational modification of procollagen that is necessary for cross-link formation.