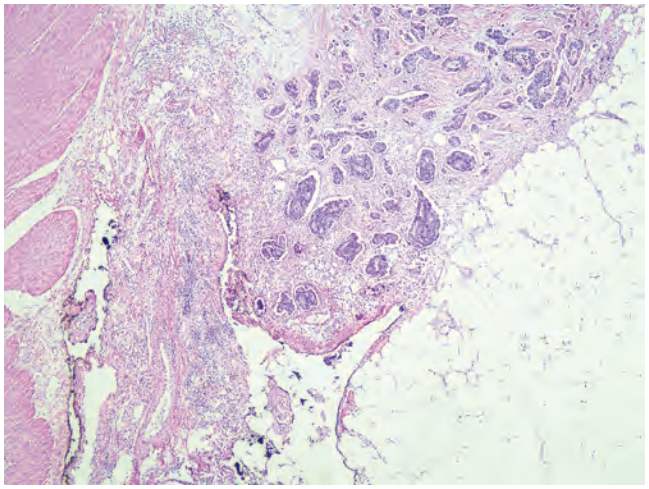

Figure 7-15 Colon carcinoma invading pericolonic adipose tissue. (Courtesy Dr. Shuji Ogino, Dana Farber Cancer Institute, Boston, Mass.)

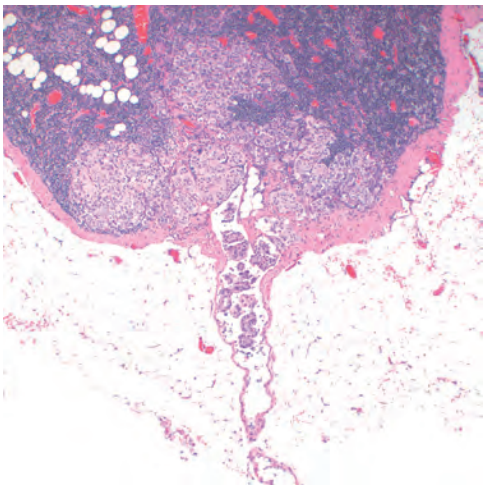

Figure 7-16 Axillary lymph node with metastatic breast carcinoma. Note the aggregates of tumor cells within the substance of the node and the dilated lymphatic channel. (Courtesy Dr. Susan Lester, Brigham and Women's Hospital, Boston, Mass.)

liquid tumors) are derived from blood-forming cells that normally have the capacity to enter the bloodstream and travel to distant sites; as a result, leukemias and lymphomas are often disseminated at diagnosis and are always taken to be malignant.

Pathways of Spread

Dissemination of cancers may occur through one of three pathways: (1) direct seeding of body cavities or surfaces, (2) lymphatic spread, and (3) hematogenous spread. Although iatrogenic spread of tumor cells on surgical instruments may occur—it is the reason, for example, why biopsies of testicular masses are never done—it is generally rare and not discussed further.

Seeding of Body Cavities and Surfaces. Seeding of body cavities and surfaces may occur whenever a malignant neoplasm penetrates into a natural "open field" lacking physical barriers. Most often involved is the peritoneal cavity (Fig. 7-15), but any other cavity—pleural, pericardial, subarachnoid, and joint spaces—may be affected. Such seeding is particularly characteristic of carcinomas arising in the ovaries, which, not infrequently, spread to peritoneal surfaces, which become coated with a heavy cancerous glaze. Remarkably, the tumor cells may remain confined to the surface of the abdominal viscera without penetrating into the substance. Sometimes mucus-secreting appendiceal carcinomas or ovarian carcinomas fill the peritoneal cavity with a gelatinous neoplastic mass referred to as *pseudomyxoma peritonei.*

Lymphatic Spread. **Transport through lymphatics is the most common pathway for the initial dissemination of carcinomas** (Fig. 7-16). Sarcomas may also use this route. Tumors do not contain functional lymphatics, but lymphatic vessels located at the tumor margins are apparently sufficient for the lymphatic spread of tumor cells. The emphasis on lymphatic spread for carcinomas and hematogenous spread for sarcomas is misleading, because ultimately there are numerous interconnections between the vascular and the lymphatic systems. The pattern of lymph node involvement follows the natural routes of lymphatic drainage. Because carcinomas of the breast usually arise in the upper outer quadrants, they generally disseminate first to the axillary lymph nodes. Cancers of the inner quadrants drain to the nodes along the internal mammary arteries. Thereafter, the infraclavicular and supraclavicular nodes may become involved. Carcinomas of the lung arising in the major respiratory passages metastasize first to the perihilar tracheobronchial and mediastinal nodes. Local lymph nodes, however, may be bypassed—so-called skip metastasis—because of venous-lymphatic anastomoses or because inflammation or radiation has obliterated lymphatic channels.

In breast cancer, determining the involvement of axillary lymph nodes is important for assessing the future course of the disease and for selecting suitable therapeutic strategies. To avoid the considerable surgical morbidity associated with a full axillary lymph node dissection, *biopsy of sentinel nodes* is often used to assess the presence or absence of metastatic lesions in the lymph nodes. **A sentinel lymph node is defined as "the first node in a regional lymphatic basin that receives lymph flow from the primary tumor."** Sentinel node mapping can be done by injection of radiolabeled tracers or colored dyes, and examination of frozen sections of the sentinel lymph node performed during surgery can guide the surgeon to the appropriate therapy. Sentinel node examination has also been used for detecting the spread of melanomas, colon cancers, and other tumors.

In many cases, the regional nodes serve as effective barriers to further dissemination of the tumor, at least for a while. Conceivably, after arrest within the node the cells may be destroyed by a tumor-specific immune response. Drainage of tumor cell debris or tumor antigens, or both, also induces reactive changes within nodes. Thus, enlargement of nodes may be caused by the spread and growth of cancer cells or reactive hyperplasia (Chapter 13). Therefore, nodal enlargement in proximity to a cancer, while it must arouse suspicion, does not necessarily equate with dissemination of the primary lesion.