Table 13-3 Causes of Leukocytosis

Type of Leukocytosis	Causes
Neutrophilic leukocytosis	Acute bacterial infections, especially those caused by pyogenic organisms; sterile inflammation caused by, for example, tissue necrosis (myocardial infarction, burns)
Eosinophilic leukocytosis (eosinophilia)	Allergic disorders such as asthma, hay fever, parasitic infestations; drug reactions; certain malignancies (e.g., Hodgkin and some non-Hodgkin lymphomas); automimmune disorders (e.g., pemphigus, dermatitis herpetiformis) and some vasculitides; atheroembolic disease (transient)
Basophilic leukocytosis (basophilia)	Rare, often indicative of a myeloproliferative disease (e.g., chronic myelogenous leukemia)
Monocytosis	Chronic infections (e.g., tuberculosis), bacterial endocarditis, rickettsiosis, and malaria; autoimmune disorders (e.g., systemic lupus erythematosus); inflammatory bowel diseases (e.g., ulcerative colitis)
Lymphocytosis	Accompanies monocytosis in many disorders associated with chronic immunologic stimulation (e.g., tuberculosis, brucellosis); viral infections (e.g., hepatitis A, cytomegalovirus, Epstein-Barr virus); *Bordetella pertussis* infection

children, can cause the appearance of large numbers of activated lymphocytes that resemble neoplastic lymphoid cells. At other times, particularly in severe infections, many immature granulocytes appear in the blood, mimicking a myeloid leukemia *(leukemoid reaction)*. Special laboratory studies (discussed later) are helpful in distinguishing reactive and neoplastic leukocytoses.

Lymphadenitis

Following their initial development from precursors in the central (also called primary) lymphoid organs,—the bone marrow for B cells and the thymus for T cells—, lymphocytes circulate through the blood and, under the influence of specific cytokines and chemokines, home to lymph nodes, spleen, tonsils, adenoids, and Peyer's patches, which constitute the peripheral (secondary) lymphoid tissues. Lymph nodes, the most widely distributed and easily accessible lymphoid tissue, are frequently examined for diagnostic purposes. They are discrete encapsulated structures that contain well-organized B-cell and T-cell zones, which are richly invested with phagocytes and antigen-presenting cells (see Fig. 6-8, Chapter 6).

The activation of resident immune cells leads to morphologic changes in lymph nodes. Within several days of antigenic stimulation, the primary follicles enlarge and develop pale-staining *germinal centers*, highly dynamic structures in which B cells acquire the capacity to make high-affinity antibodies against specific antigens. Paracortical T-cell zones may also undergo hyperplasia. The degree and pattern of the morphologic changes are dependent on the inciting stimulus and the intensity of the response. Trivial injuries and infections induce subtle changes, while more significant infections inevitably produce nodal enlargement and sometimes leave residual scarring. For this reason, lymph nodes in adults are almost never "normal" or "resting," and it is often necessary to distinguish morphologic changes secondary to past experience from those related to present disease. Infections and inflammatory stimuli often elicit regional or systemic immune reactions within lymph nodes. Some that produce distinctive morphologic patterns are described in other chapters. Most, however, cause stereotypical patterns of lymph node reaction designated acute and chronic nonspecific lymphadenitis.

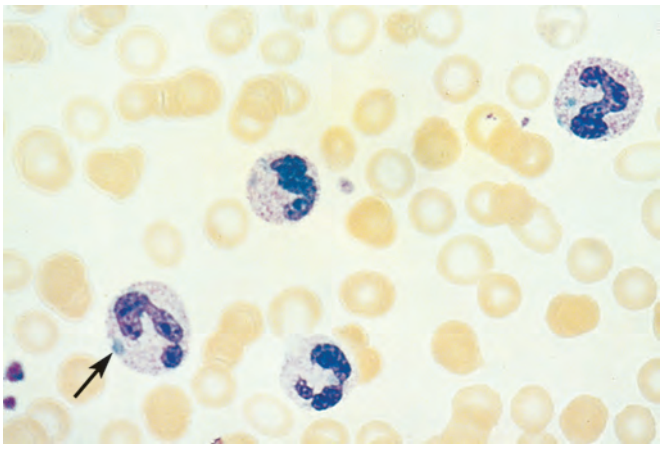

Figure 13-2 Reactive changes in neutrophils. Neutrophils containing coarse purple cytoplasmic granules (toxic granulations) and blue cytoplasmic patches of dilated endoplasmic reticulum (Döhle bodies, *arrow*) are observed in this peripheral blood smear prepared from a patient with bacterial sepsis.

Acute Nonspecific Lymphadenitis

Acute lymphadenitis in the cervical region is most often due to drainage of microbes or microbial products from infections of the teeth or tonsils, while in the axillary or inguinal regions it is most often caused by infections in the extremities. Acute lymphadenitis also occurs in mesenteric lymph nodes draining acute appendicitis. Other self-limited infections may also cause acute mesenteric adenitis and induce symptoms mimicking acute appendicitis, a differential diagnosis that plagues the surgeon. Systemic viral infections (particularly in children) and bacteremia often produce acute generalized lymphadenopathy.

MORPHOLOGY

The nodes are swollen, gray-red, and engorged. Microscopically, there is prominence of large reactive germinal centers containing numerous mitotic figures. Macrophages often contain particulate debris derived from dead bacteria or necrotic cells. When pyogenic organisms are the cause, neutrophils are prominent and the centers of the follicles may undergo necrosis; sometimes the entire node is converted to a bag of pus. With less severe reactions, scattered neutrophils infiltrate about the follicles and accumulate within the lymphoid sinuses. The endothelial cells lining the sinuses undergo hyperplasia.

Nodes involved by acute lymphadenitis are enlarged and painful. When abscess formation is extensive the nodes become fluctuant. The overlying skin is red. Sometimes, suppurative infections penetrate through the capsule of the

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