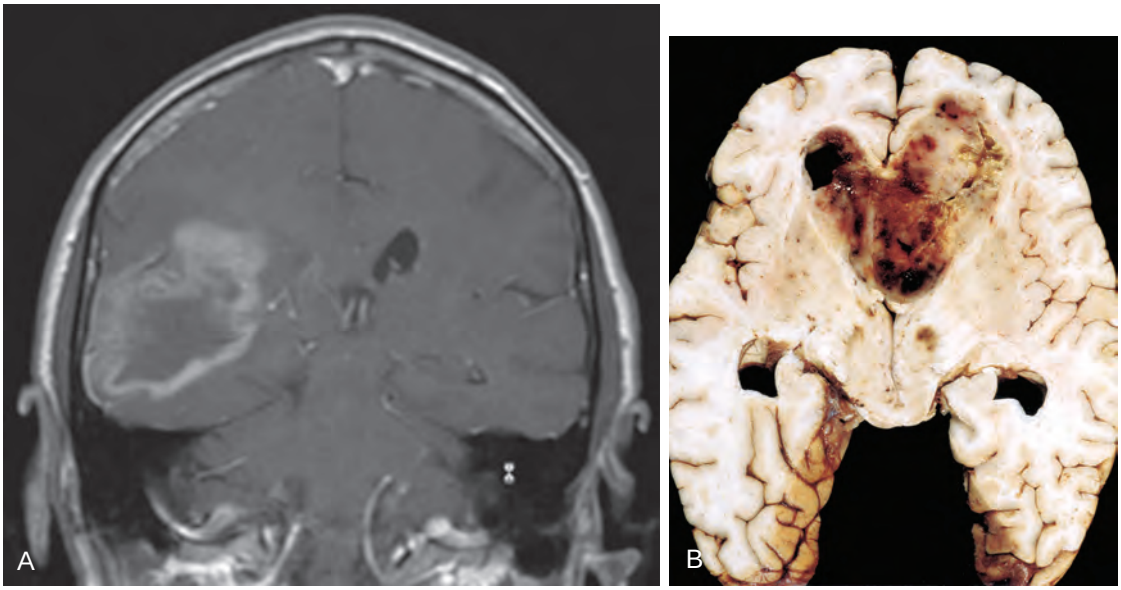

Figure 28-47 A, Contrast T1-weighted coronal magnetic resonance image shows a large mass in the right parietal lobe with "ring" enhancement. **B,** Glioblastoma appearing as a necrotic, hemorrhagic, infiltrating mass.

tumors in which the predominant neoplastic astrocyte shows a brightly eosinophilic cell body from which emanate abundant, stout processes.

In **glioblastoma** (previously called *glioblastoma multiforme*, and sometimes still abbreviated GBM), variation in the appearance of the tumor from region to region is characteristic (Fig. 28-47). Some areas are firm and white, others are soft and yellow due to necrosis, and yet others show cystic degeneration and hemorrhage. The histologic appearance of glioblastoma is similar to anaplastic astrocytoma with the additional features of **necrosis** and **vascular/endothelial cell proliferation**. Necrosis in glioblastoma often occurs in a serpentine pattern in areas of hypercellularity. Tumor cells collect along the edges of the necrotic regions, producing a histologic pattern referred to as **pseudo-palisading** (Fig. 28-48). The vascular cell proliferation produces tufts of cells that pile up and bulge into the lumen; the minimal criterion for this feature is a double layer of endothelial cells. With marked vascular cell proliferation the tuft forms a ball-like structure, the glomeruloid body. VEGF, produced by malignant astrocytes in response to hypoxia, contributes to this distinctive vascular change. Since histologic features can be extremely variable from one region to another, small biopsy specimens may not be representative of the entire tumor.

Gliomatosis cerebri is a diffuse glioma with extensive infiltration of multiple regions of the brain, in some cases the entire brain. Because of the widespread infiltration, this process follows an aggressive course and is considered to be a grade III/IV lesion.

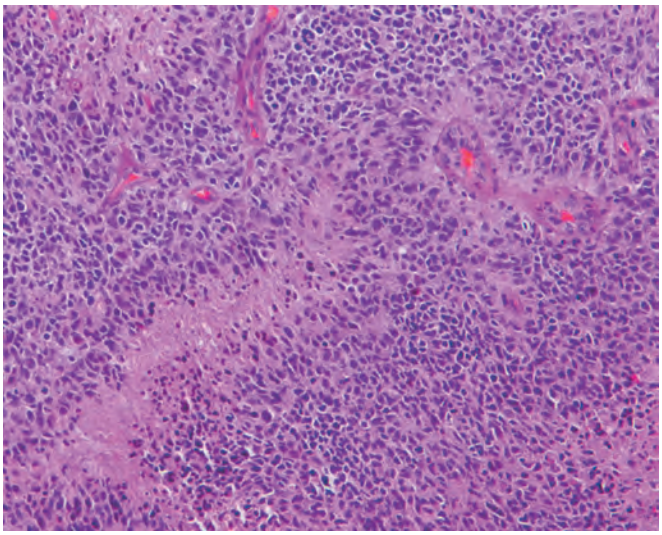

Figure 28-48 Glioblastoma. Foci of necrosis with pseudo-palisading of malignant nuclei and endothelial cell proliferation.

Clinical Features. The presenting symptoms of infiltrating astrocytomas depend, in part, on the location and growth rate of the tumor. Well-differentiated diffuse astrocytomas may remain stable or progress only slowly over a number of years; the mean survival is more than 5 years. Eventually, however, clinical deterioration invariably occurs and is usually due to the emergence of a more rapidly growing tumor of higher histologic grade. Radiologic studies show mass effect as well as changes in the brain adjacent to the tumor, such as edema. High-grade astrocytomas have abnormal vessels that are "leaky," with an abnormally permeable blood-brain barrier, and therefore, demonstrate contrast enhancement on imaging studies. The prognosis for individuals with glioblastoma is very poor, although the use of newer chemotherapeutic agents has provided some benefit. Epigenetic silencing of the promoter for the gene encoding the DNA repair enzyme MGMT predicts responsiveness to DNA alkylating drugs—as would be expected since MGMT is critical for the repair of the chemotherapeutically induced DNA modification. With current treatment, consisting of resection followed by radiation therapy and chemotherapy, the mean length of survival after diagnosis has increased to 15 months; 25%