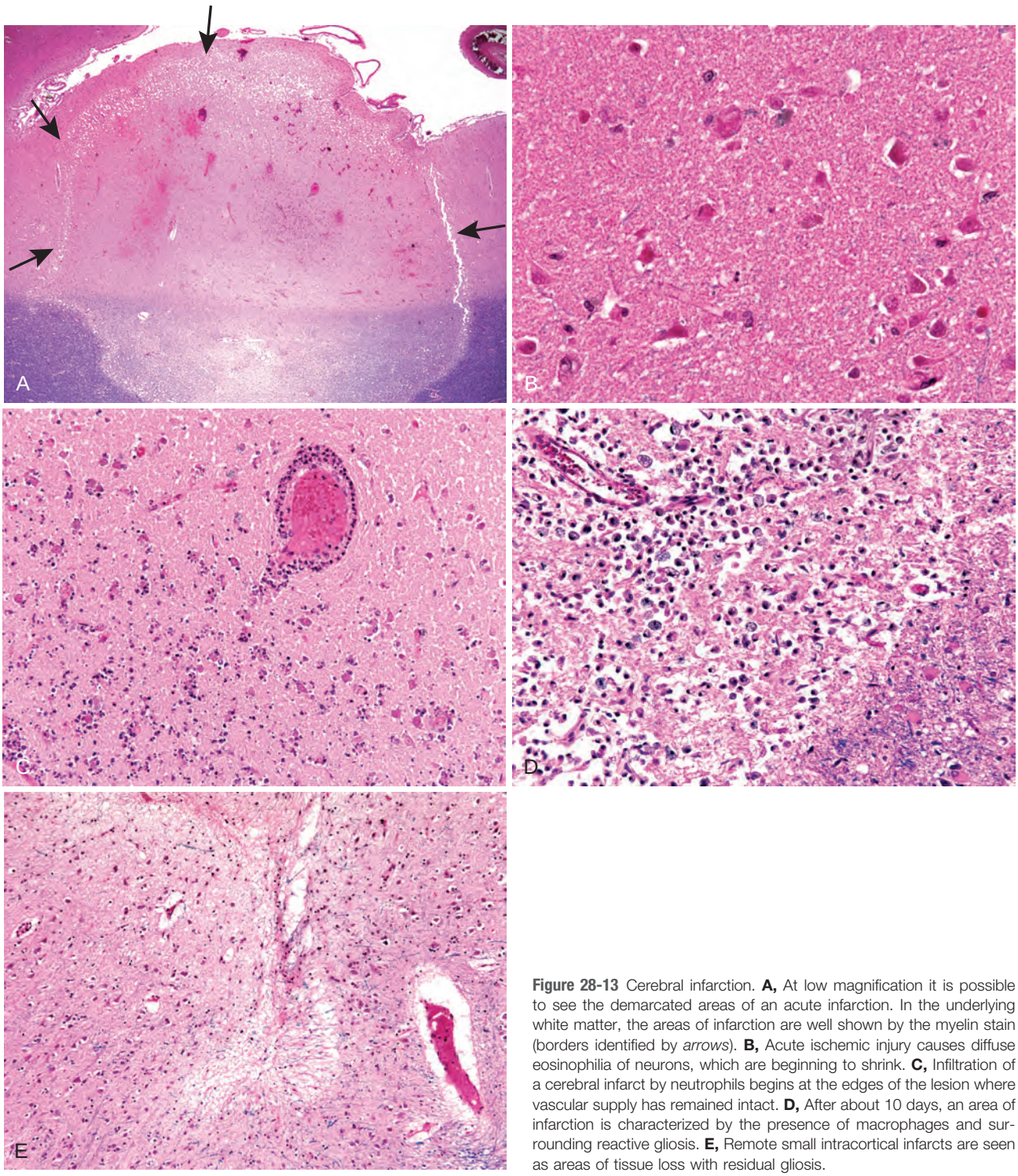


**Figure 28-13** Cerebral infarction. **A,** At low magnification it is possible to see the demarcated areas of an acute infarction. In the underlying white matter, the areas of infarction are well shown by the myelin stain (borders identified by *arrows*). **B,** Acute ischemic injury causes diffuse eosinophilia of neurons, which are beginning to shrink. **C,** Infiltration of a cerebral infarct by neutrophils begins at the edges of the lesion where vascular supply has remained intact. **D,** After about 10 days, an area of infarction is characterized by the presence of macrophages and surrounding reactive gliosis. **E,** Remote small intracortical infarcts are seen as areas of tissue loss with residual gliosis.

often originating over atheromatous plaques within the carotid arteries. Other sources of emboli include paradoxical emboli, particularly in children with cardiac anomalies; emboli associated with cardiac surgery; and emboli of other material (tumor, fat, or air). The territory of distribution of the middle cerebral artery—the direct extension of the internal carotid artery—is most frequently affected by embolic infarction; the incidence is about equal in the two hemispheres. Emboli tend to lodge where blood vessels branch or in areas of preexisting luminal stenosis. "Shower embolization," as in fat embolism, may occur after fractures; affected individuals manifest generalized cerebral dysfunction with disturbances of higher cortical function and consciousness, often without localizing signs. Widespread hemorrhagic lesions involving the white matter are characteristic of embolization of bone marrow after trauma (Fig. 28-14).