object and tearing of tissue. As with any other organ, a blow to the surface of the brain, transmitted through the skull, leads to rapid tissue displacement, disruption of vascular channels, and subsequent hemorrhage, tissue injury, and edema (Fig. 28-9). Hemorrhage can extend into the subarachnoid space from these lesions. The crests of gyri are most susceptible, since this is where the direct force is greatest. The most common locations for contusions correspond to the most frequent sites of direct impact and to regions of the brain that overlie a rough and irregular inner skull surface, such as the frontal lobes along the orbital ridges and the temporal lobes. Contusions are less frequent over the occipital lobes, brainstem, and cerebellum unless these sites are adjacent to a skull fracture *(fracture contusions)*.

A person who suffers a blow to the head may develop a contusion at the point of contact (a *coup* injury) or a contusion on the brain surface diametrically opposite to it (a *contrecoup* injury). Since their macroscopic and microscopic appearance is indistinguishable, the distinction between them is based on identification of the point of impact. In general, if the head is immobile at the time of trauma, only a coup injury is found. If the head is mobile, both coup and contrecoup lesions may be found. Whereas the coup lesion is caused by the contact between brain and skull at the site of impact, the contrecoup contusion is thought to develop when the brain strikes the opposite inner surface of the skull after sudden deceleration.

Sudden impacts that result in violent posterior or lateral hyperextension of the neck (as occurs when a pedestrian is struck from the rear by a vehicle) may avulse the pons from the medulla or the medulla from the cervical cord, causing instant death.

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When seen on cross-section, contusions are wedge shaped, with the broad base lying along the surface at the point of impact (Fig. 28-9*B*). The appearance of contusions is similar regardless of the source of the trauma. In the earliest stages, there is edema and hemorrhage, which is often pericapillary. During the next few hours, the extravasation of blood extends throughout the involved tissue, across the width of the cerebral cortex, and into the white matter and subarachnoid space. Morphologic evidence of neuronal injury (pyknosis of the nucleus, eosinophilia of the cytoplasm, and disintegration of the cell) takes about 24 hours to appear, although functional deficits may occur earlier. Axonal swellings develop in the vicinity of damaged neurons or at great distances away. The inflammatory response to the injured tissue follows its usual course, with the appearance of neutrophils followed by macrophages. Old traumatic lesions on the surface of the brain have a characteristic gross appearance. They are depressed, retracted, yellowish brown patches involving the crests of gyri, most commonly those that are located at the sites of contrecoup injuries (inferior frontal cortex, temporal and occipital poles). The term **plaque jaune** is applied to these lesions (Fig. 28-9*C*), which can become epileptic foci. More extensive hemorrhagic regions of brain trauma give rise to larger cavitated lesions, which can resemble remote infarcts. In old contusions, gliosis and residual hemosiderin-laden macrophages predominate.

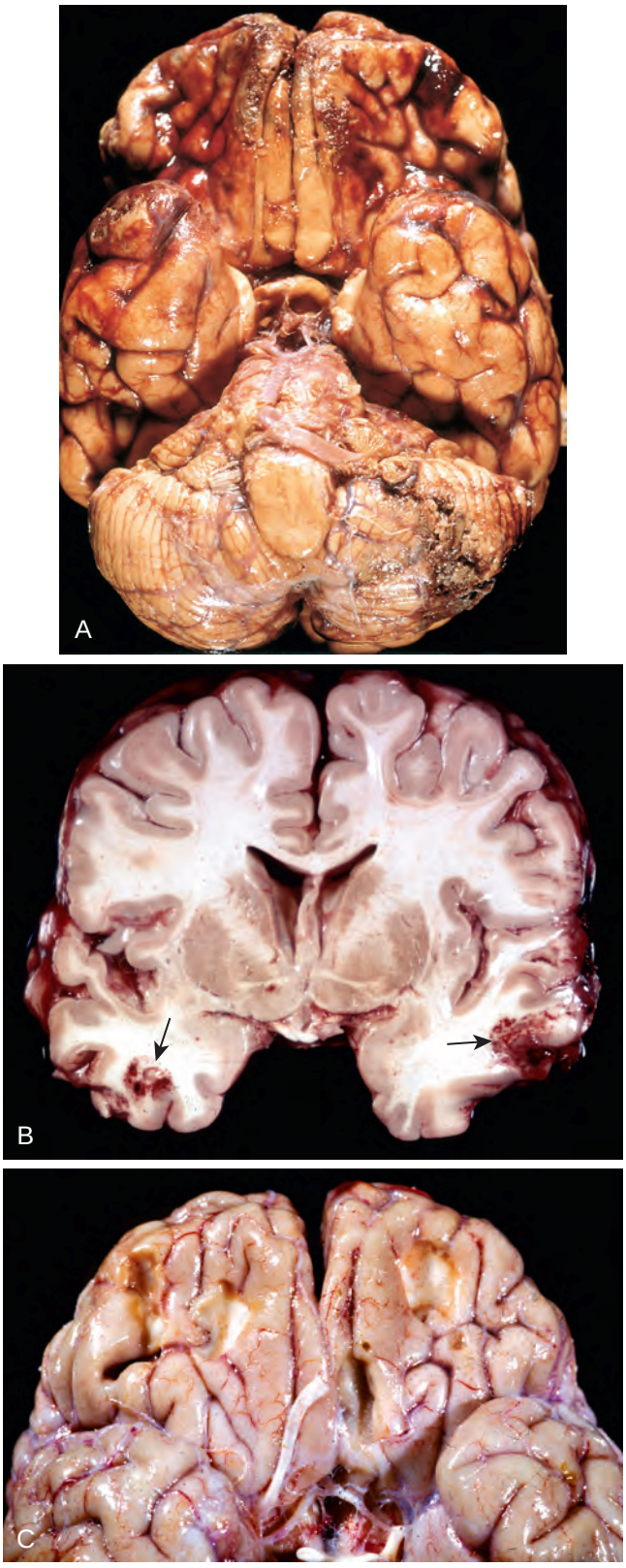

Figure 28-9 **A,** Multiple contusions involving the inferior surfaces of frontal lobes, anterior temporal lobes, and cerebellum. **B,** Acute contusions are present in both temporal lobes, with areas of hemorrhage and tissue disruption (*arrows*). **C,** Remote contusions are present on the inferior frontal surface of this brain, with a yellow color (associated with the term *plaque jaune*).