manifested by an increase in head circumference. Hydrocephalus developing after this period, in contrast, is associated with expansion of the ventricles and increased intracranial pressure, without a change in head circumference. If the ventricular system is obstructed and does not communicate with the subarachnoid space, as may occur because of a mass in the third ventricle, it is called *noncommunicating, or obstructive, hydrocephalus*. In *communicating hydrocephalus,* the ventricular system is in communication with the subarachnoid space, and there is enlargement of the entire ventricular system. The term *hydrocephalus ex vacuo* refers to a compensatory increase in ventricular volume secondary to a loss of brain parenchyma.

Raised Intracranial Pressure and Herniation

Herniation is the displacement of brain tissue past rigid dural folds (the falx and tentorium) or through openings in the skull because of increased intracranial pressure. As the volume of the brain increases, CSF is displaced and the vasculature is compressed, leading to increasing pressure within the cranial cavity. When the increase is beyond the limit permitted by compression of veins and displacement of CSF, tissue herniates between compartments across the pressure gradient. Herniation is mostly associated with mass effect, either diffuse (generalized brain edema) or focal (tumors, abscesses, or hemorrhages). Elevated intracranial pressure may also reduce perfusion of the brain, further exacerbating cerebral edema. If the expansion is sufficiently severe, herniation may occur in multiple anatomic locations (Fig. 28-3).

- *Subfalcine (cingulate) herniation* occurs when unilateral or asymmetric expansion of a cerebral hemisphere displaces the cingulate gyrus under the falx. This may lead to compression of the anterior cerebral artery and its branches.
- *Transtentorial (uncinate, mesial temporal) herniation* occurs when the medial aspect of the temporal lobe is compressed against the free margin of the tentorium. With increasing displacement of the temporal lobe, the third cranial nerve is compromised, resulting in pupillary dilation and impairment of ocular movements on the side of the lesion. The posterior cerebral artery may also be compressed, resulting in ischemic injury to the territory supplied by that vessel, including the primary visual cortex. When the extent of herniation is large enough the contralateral cerebral peduncle may be compressed, resulting in hemiparesis ipsilateral to the side of the herniation; the compression in the peduncle in this setting is known as the *Kernohan notch*. Progression of transtentorial herniation is often accompanied by secondary hemorrhagic lesions in the midbrain and pons, termed *Duret hemorrhages* (Fig. 28-4). These linear or flame-shaped lesions usually occur in the midline and paramedian regions and are believed to be due to distortion or tearing of penetrating veins and arteries supplying the upper brainstem.
- *Tonsillar herniation* refers to displacement of the cerebellar tonsils through the foramen magnum. This pattern of herniation is life-threatening because it causes brainstem compression and compromises vital respiratory and cardiac centers in the medulla.

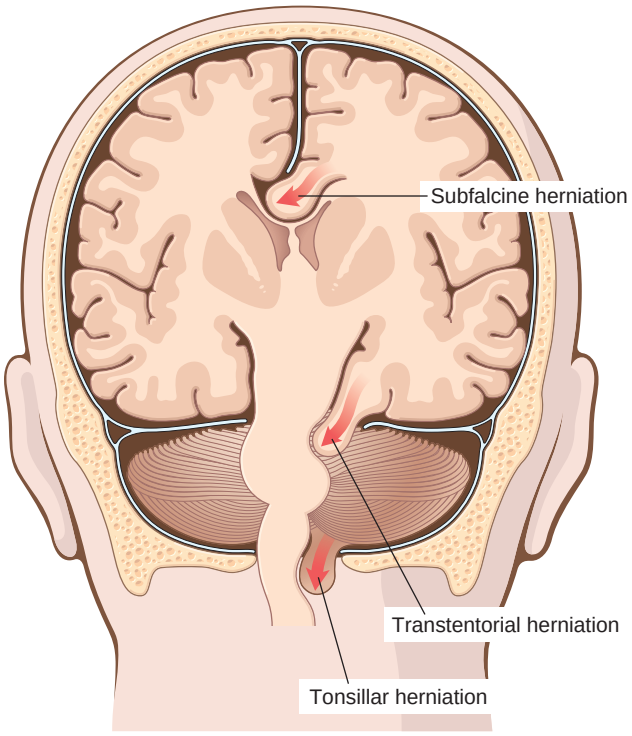

Figure 28-3 Major herniation syndromes of the brain: subfalcine, transtentorial, and tonsillar.

Figure 28-4 Duret hemorrhage involving the brainstem at the junction of the pons and midbrain.

KEY CONCEPTS

Cerebral Edema, Hydrocephalus, and Raised Intracranial Pressure and Herniation

- Cerebral edema is the accumulation of excess fluid within the brain parenchyma. Hydrocephalus is an increase in CSF volume within all or part of the ventricular system.