9. Extraaxial and Subarachnoid Hemorrhage

Subdural hematomas (SDH) are most frequently seen in the setting of trauma, particularly in older individuals, and result from injury to the veins bridging the subdural space. Depending upon the age of the lesion, these may be isointense to adjacent brain parenchyma (Fig. 9.1 A, T2WI), thus difficult to detect on MR using some pulse sequences. SDH (Fig. 9.1 B, arrows, T1WI) typically exhibit a crescentic shape and do not cross dural attachments at the venous sinuses. The evolution of SDH SI follows in general that of intraparenchymal hemorrhages (see Ch. 8). In a child, the presence of multiple SDH of various temporal stages (Fig. 9.2 A – T2WI, B 1-4 - T1WI) is pathognomonic of abuse. Unlike intraparenchymal bleeds, SDH (except in recurrent bleeds) do not demonstrate a hemosiderin phase: the lack of a dural BBB allows the macrophages that scavenge hemosiderin to return into the bloodstream. The appearance of chronic SDH, therefore, correlates with methemoglobin resorption which leads to a progressively lower SI on T1WI. The appearance of SDH may be confused for brain atrophy in these chronic or even subacute phases, and the presence of contrast enhancement (of the lining membrane) may help to confirm the presence of SDH. Chronic SDH may further degrade into a subdural hygroma, although these may also arise from a CSF leak (typically from an arachnoid membrane tear). Hygromas contain little if any methemoglobin, and thus appear isointense to CSF on T1 and T2WI. Hygromas with greater protein concentrations may demonstrate higher SI on FLAIR scans, while areas of residual hemorrhage correlate with low SI on GRE. In distinction, arachnoid cysts are isointense to CSF on all sequences. Epidural hematomas are lens-shaped and most commonly associated with fractures of the temporal bone. Figure 9.3 A (white arrow), a T1WI, demonstrates an epidural hematoma with two distinct signal components that crosses the tentorium. While epidural hematomas may cross the falx or tentorium, in general they do not cross the suture lines connecting the dura to the inner bone table. A band of low SI dura compressed against brain parenchyma also suggests an epidural location (Fig. 9.3 A, white arrow).

Unlike the entities above, subarachnoid hemorrhage (SAH) usually results from rupture of
berry aneurysms or arteriovenous malformations rather than from trauma. Hyperacute and acute SAH are not well-seen on most MR sequences, as the high oxygen content of CSF prevents reduction of oxyhemoglobin to deoxyhemoglobin. The presence of CSF also tends to dilute the hemoglobin, decreasing the effect of its byproducts on SI. The presence of additional protein within a hemorrhagic region, however, decreases T1, and results in hyperintensity to the normal low SI of CSF on FLAIR scans. Thus, FLAIR imaging is exquisitely sensitive to the detection of SAH (likely even more so than CT), and demonstrates SAH as areas of abnormally high SI either within the basal cisterns or cortical sulci (Fig. 9.3 B, arrows). Leading differential considerations include meningitis and meningeal carcinomatosis, although oxygen administration at the time of MRI and pulsation artifacts, particularly in the basal cisterns, may have a similar appearance. GRE (and susceptibility weighted imaging, or SWI), identifies the paramagnetic by-products of hemorrhage, and may be of equal sensitivity to FLAIR (with the two sequences complementary to each other). Chronic or recurrent hemorrhage, as seen in patients with vascular abnormalities, may result in superficial hemosiderosis, often seen as a thin rim of hypointensity lining the parenchymal surface on T2WI. Intraventricular hemorrhage, seen in trauma and pre-term infants, is similar to SAH in its SI and delayed temporal evolution. FLAIR scans demonstrate hyperintensity against attenuated (suppressed) CSF SI, most often in the dependent portions (i.e. atria and occipital horns) of the lateral ventricle. Layering of blood products and CSF is commonly seen in interventricular hemorrhage, regardless of patient age.
Fig. 9.3