Susceptibility-weighted imaging (SWI) employs a high-resolution, velocity-compensated (for flow in all three axes), spoiled 3D GRE scan, and specifically the phase information from this scan, to emphasize susceptibility differences between tissues. With sufficiently long echo times using SWI, lesions or structures with magnetic susceptibility different from neighboring normal brain will possess a phase difference and be depicted with low signal intensity. In the SWI implementation, two types of images are reconstructed, a high pass filtered phase image and a minimum intensity projection, the latter being with phase-weighted magnitude information. SWI has high sensitivity to normal venous blood/flow (due to deoxygenation), high levels of deoxyhemoglobin (acute hemorrhage), hemosiderin (chronic hemorrhage), and iron in the form of ferritin. In common with most MR applications, this technique is further improved at 3 T, due to both increased SNR and more prominent susceptibility related effects. The latter allows TE and thus TR to be halved, markedly reducing scan time.

Primary applications for SWI include traumatic brain injury (both acute and chronic) and cavernous malformations. In regard to trauma, SWI offers improved depiction of the multiple punctate hemorrhages seen in diffuse axonal injury (DAI), relative to T2*-weighted GRE scans. The lesions in DAI, which are due to shear injury, occur anatomically at the gray–white matter junction and within the corpus callosum, deep gray nuclei, and brain stem. Also improved with SWI is the visualization of intraventricular and subarachnoid hemorrhage, both depicted with low signal intensity within otherwise normal CSF-containing spaces. Use of SWI thus complements that of FLAIR in the detection of acute subarachnoid hemorrhage. In regard to patients with cavernous malformations (Fig. 38.1, acquired at 3 T), due to the improved sensitivity to hemosiderin, these lesions will in general appear larger on SWI (arrow, B) when compared with T2*-weighted GRE scans (A). SWI offers improved detectability of very small cavernous malformations, an
important point given that 10 to 30% of patients have the familial type of disease in which multiple lesion are seen, many very small. Venous angiomas and the venous drainage within neoplastic tissue are also well depicted by SWI.

The images in Fig. 38.2 present a confusing radiologic picture, with SWI facilitating scan interpretation. On higher sections (not shown), the patient had an easily recognized, early subacute, anterior cerebral artery territory infarct. At the anatomic level illustrated, (A) FLAIR demonstrates a mass lesion with surrounding high signal intensity. No abnormal enhancement is seen (B) postcontrast on the T1-weighted image. (C) On DWI, the abnormal high signal intensity on FLAIR is demonstrated to be composed both of cytotoxic edema (high signal intensity on DWI, white arrows, due to the infarct) and vasogenic edema (intermediate signal intensity on DWI, black arrow, due to the mass). (D) SWI simplifies the diagnosis, making the large cerebral hematoma (the central mass, with marked hypointensity) readily evident. Subsequent to the onset of ischemia, the patient had suffered the complication of parenchymal hemorrhage within the area of the infarct. These scans were acquired at 3 T, with (D) the SWI scan (in common with Fig. 38.1B) being the projection image.