PURPOSE. Identification and characterization of intracranial hemorrhages (ICH) are critical for appropriate management of hemorrhagic stroke patients. As the hematoma evolves from hyperacute (<1 day), acute (<3 days), early (3-7 days) and late subacute (7-14 days), to chronic (>14 days) stage, metalloprotein in erythrocytes degrades from oxygenated hemoglobin (OxyHb), deoxygenated hemoglobin (DeoxyHb), intra- and extra-cellular methemoglobin (MetHb), and hemosiderin, which all have different appearances in corresponding T1 weighted (T1w) and T2 weighted (T2w) MR images (1). It has been hypothesized that the differences are partly attributed to the changing magnetic susceptibilities associated with these blood breakdown products. Therefore, we applied quantitative susceptibility mapping (QSM) in this study to investigate the magnetic susceptibility of hematomas at various stages.

METHODS. We retrospectively analyzed 18 patients with a total of 32 hematomas. Based on clinical history and image findings on CT and MRI (T1w, T2w and gradient echo (GRE)), hematomas were classified according to their ages as hyperacute (N=4), acute (N=7), early subacute (N=9), late subacute (N=9) and chronic (N=3). QSM images were generated using the morphology enabled dipole inversion (MEDI) (2-4) method from the same gradient echo datasets, whose imaging parameters were: TE/ΔTE/ΔTE=5ms/5ms/8; TR=45ms; FA=20°; FOV=24cm; slice thickness=2mm; acquisition matrix=256×256×50~60. On QSM, image features of the hematomas at various stages were qualitatively described by two senior neuro-radiologists, and quantitatively measured in hand-drawn regions of interests covering the hematoma.

RESULTS. Acute, early and late subacute, and chronic hematomas were seen as paramagnetic and revealed mostly homogenously hyperintense signal compared to brain parenchyma on QSM. Their size and shape on QSM correlated well with CT and MRI findings. In the hyperacute stage of ICH, QSM demonstrated heterogeneous, strong and less strong hyperintensity (Fig. 1). In the 4 hyperacute hematomas, there was no evidence of isointense, homogeneous signal intensity in the core of the hematoma, which was anticipated from pure OxyHb as described in the literature (1,5-8). In addition, the hypointense rim on GRE images was not observed on QSM. The susceptibilities of various blood products are summarized in Table 2.

DISCUSSION. Hematomas are consistently paramagnetic at all stages. The quantitative measurement showed striking agreement with literature and theoretical prediction.

Assuming the DeoxyHb is the dominating species in acute hematoma, the measured average susceptibility 1.32 ppm agreed well with expected value of 1.37 ppm calculated using Hct × ΔXDeoxy, where the hematocrit value Hct = 0.4 and fully deoxygenated hemoglobin susceptibility ΔXDeoxy = 3.43 ppm (9-10). The susceptibility increase in early subacute stage agrees with the theoretical prediction when iron in DeoxyHb changed from Fe2+ to Fe3+ in intracellular MetHb. The reduced susceptibility in late subacute stage may reflect the clearance of extracellular MetHb by modified macrophages and/or the dilution of MetHb by interstitial fluid (5). Later on, the susceptibility increased again in hemosiderin, probably due to the hemosiderin crystallization generating superparamagnetism (11). The hypointense appearance of hematomas on QSM agreed with expectation except for the hyperacute stage, which is expected to be dominated by the diamagnetic oxyhemoglobin at this stage. On conventional GRE images, hyperacute hematoma may show a thin, irregular rim of hypointensity which is explained as rapid deoxygenation at the periphery of the hematoma (1,5-8). However, there is lack of histological staining demonstrating DeoxyHb at the periphery of ICH. One hypothesis states that the dense aggregate of neutrophils results in high local concentrations of paramagnetic oxygen radicals, which may be the origin of the observed low signal on T2-weighted images as well as in GRE (7). Interestingly, none of our QSM cases showed hypointense central core in hyperacute hematomas corresponding to pure OxyHb, and there was no hypointense rim on QSM matching the hypointense rim on GRE images. This finding suggests that the degradation of OxyHb to DeoxyHb in hematomas may be faster than expected and warrants further investigation. Furthermore, the hypointense rim on GRE images is potentially a susceptibility artifact.

CONCLUSION. We demonstrated the feasibility of using QSM to quantitatively image hematomas at various stages. While the susceptibility values agreed with literature in acute, subacute and chronic stages, the elevated susceptibility in every early stage hematoma indicates the existence of DeoxyHb.