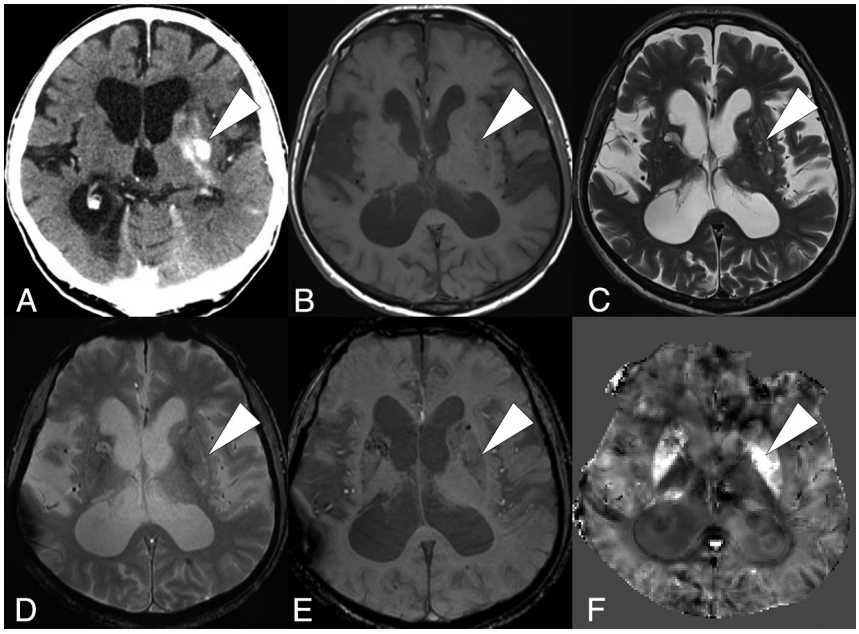
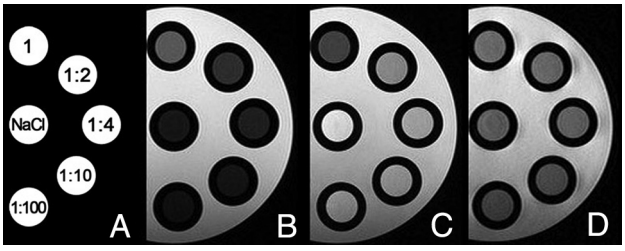


ON-LINE FIG 1. A 78-year-old patient with an acute stroke caused by occlusion of the left middle cerebral artery. Postinterventional axial CT (*A*) and MR imaging (*B–F*) after mechanical revascularization. On postinterventional CT obtained 1 hour after revascularization, there are hyperattenuated areas in the left lentiform nucleus (*A*, *arrowhead*). On MR imaging obtained 2 hours after revascularization, there is no T1 and T2 shortening but prolongation of T1 (*B*, *arrowhead*) and T2 (*C*, *arrowhead*), corresponding to the beginning of ischemic edema. The affected areas appear normal on T2*-weighted gradient-echo (*D*, *arrowhead*) and susceptibility-weighted imaging (*E*, *arrowhead*). On the susceptibility map, there is no susceptibility shift compared with the nonischemic contralateral lentiform nucleus ($P = .351$, Student *t* test) (*F*, *arrowhead*). For the quantitative susceptibility map, the gradient-echo phase data were unwrapped within a brain mask and the field was estimated by applying a regression fit to the realigned phase in the time domain. Background contributions were removed by our in-house software, and the susceptibility distribution was finally estimated by using an algorithm based on Tikhonov and gradient regularization ($\lambda = 0.01$, $\mu = 0.015$, 80 iterations).[9,10]

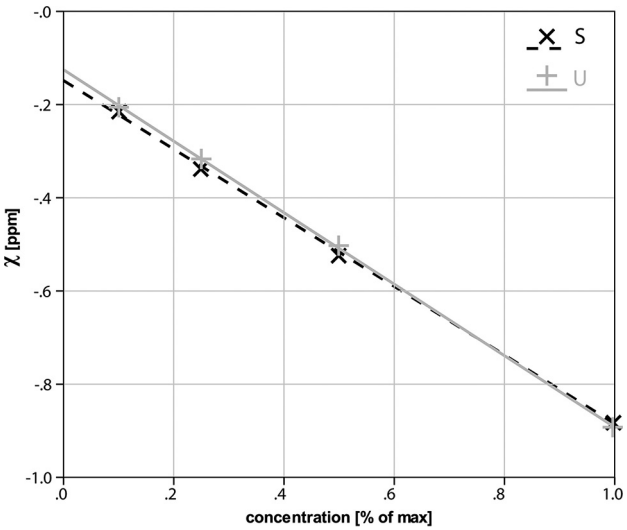


ON-LINE-FIG 2. Phantom model with iopamidol. Schematic illustration of the phantom (*A*) and actual phantom with T1-weighted SE (*B*), T2-weighted TSE (*C*), and $T2^*$-weighted GRE (*D*) MR images on a 3T scanner. Clockwise from upper to lower: pure iopamidol and iopamidol with physiologic saline solution in dilutions of 1:2, 1:4, 1:10, and 1:100. Center: physiologic saline solution. The phantom is made of acrylic glass and filled with a mixture of H_2O and gadolinium-based contrast agent.

On-line Table: Relaxation times of different concentrations of iopamidol and iopromide solutions and physiologic saline[a]

	T1 (ms)		T2 (ms)		T2* (ms)	
	1.5T	3T	1.5T	3T	1.5T	3T
Iopamidol						
1:1	317 ± 101	941 ± 30	59 ± 5	98 ± 6	53 ± 24	33 ± 2
1:2	1750 ± 151	1801 ± 109	120 ± 8	105 ± 6	80 ± 71	72 ± 6
1:4	2342 ± 223	2412 ± 215	230 ± 22	185 ± 10	110 ± 52	160 ± 31
1:10	2790 ± 288	2719 ± 237	578 ± 75	487 ± 31	188 ± 188	756 ± 730
1:100	3150 ± 431	3357 ± 281	2399 ± 1169	1463 ± 174	183 ± 284	2344 ± 1919
Physiologic saline	3012 ± 431	3264 ± 244	2550 ± 1182	2093 ± 444	206 ± 267	2652 ± 1920
Iopromide						
1:1	322 ± 139	1261 ± 78	93 ± 6	96 ± 6	80 ± 57	35 ± 2
1:2	1961 ± 166	2161 ± 135	185 ± 15	107 ± 5	161 ± 132	75 ± 7
1:4	2565 ± 273	2805 ± 290	349 ± 117	182 ± 9	222 ± 79	162 ± 34
1:10	2924 ± 315	2876 ± 257	568 ± 70	352 ± 20	404 ± 362	454 ± 590
1:100	3181 ± 432	3277 ± 257	2473 ± 1126	1444 ± 210	667 ± 874	2369 ± 1970

[a] T2* mapping at 3T proved difficult for the lower concentrations of the relaxation agent and for the physiologic saline because background field gradients (imperfect shimming) become important before the signal decays substantially by T2* decay. The T2* values for pure saline and the 1:100 dilution should, therefore, be regarded as tentative.



ON-LINE-FIG 3. Estimated susceptibility for different concentrations of iopamidol (S, *dotted line*) and iopromide (U, *solid line*). The estimated susceptibility values (in parts per million) are plotted with respect to the concentration for both contrast agents. The fitted slopes (−0.737 for iopamidol and −0.764 for iopromide) indicate the true susceptibility of the compounds.