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Carotid MRI Detection of Intraplaque Hemorrhage at 3T and 1.5T

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Abstract

BACKGROUND AND PURPOSE—Carotid intraplaque hemorrhage leads to plaque progression and ischemic events.

Detection can be accomplished with 3T T1w sequences, but may be limited by false-positive lipid/necrosis. The purpose of this study was threefold: (1) to determine if magnetization-prepared rapid acquisition with gradient-echo (MPRAGE) detects intraplaque hemorrhage versus lipid/necrosis; (2) if 3T MPRAGE image quality is retained at 1.5T; and (3) to determine observer agreement.

METHODS—MPRAGE positive areas were compared to hemorrhage and lipid/necrosis areas from 100 carotid endarterectomy slides in 12 subjects using multivariable linear regression. Image quality was determined between 3T and 1.5T in 716 carotid arteries using *t*-tests and multivariable linear regression. Kappa analysis was used to determine agreement.

RESULTS—Intraplaque hemorrhage, not lipid/necrosis, was a significant predictor of MPRAGE positive area before and after adjusting for confounders (slope = .52 vs. .51, $P < .001$).

Image quality at 3T was slightly lower than 1.5T (mean 3.87 vs. 4.34, $P < .0001$). 3T image quality remained slightly decreased before and after adjusting for confounders (slope = $-.46$ vs. $-.41$, $P < .001$). Kappa values for inter-/intraobserver agreement were .807/.919 at 3T and .803/.871 at 1.5T.

CONCLUSIONS—Carotid MPRAGE detects intraplaque hemorrhage, not lipid/necrosis. 3T image quality was retained at 1.5T with very good observer agreement.

Keywords

Carotid; MRI; Intraplaque Hemorrhage

Background and Purpose

Based on early pathologic studies, carotid intraplaque hemorrhage was found to be more highly associated with ischemic events than any other plaque component including ulceration, calcification, and lipid and necrosis.¹ Intraplaque hemorrhage is thought to originate from plaque microvessel leakage, initiating a cycle of red blood cell breakdown, lipid and cholesterol deposition, plaque growth and increased microvessel surface area resulting in further hemorrhage risk.^{2–5} In the last few years, noninvasive carotid MRI has demonstrated that intraplaque hemorrhage is a better marker of ischemic stroke risk than stenosis alone.^{6–8}

Early studies demonstrated that lipid/necrosis demonstrate high signal on T1w images.^{9–12} Other studies have also shown that intraplaque hemorrhage exhibits high signal intensity on T1w images.^{12–15} These sequences were hampered by false positives from T1 hyperintense lipid/necrosis.¹⁶ Differentiation between these components is extremely important, since carotid intraplaque hemorrhage has a much stronger association with ipsilateral ischemic events than does lipid/necrosis.¹⁷

Improved detection of intraplaque hemorrhage has been accomplished using heavily T1 weighted magnetization-prepared rapid acquisition with gradient-echo (MPRAGE) sequence.¹⁸ In a recent 3T study, MPRAGE more accurately identified intraplaque hemorrhage than conventional T1 or TOF sequences.¹⁹ Still, no studies have yet investigated the relative contributions of lipid/necrosis and intraplaque hemorrhage to MPRAGE positive signal. Using the MPRAGE sequence at 1.5T, retrospective studies have found an increased risk of ipsilateral ischemic events in patients with carotid MPRAGE hyperintense signal.^{6,8} Despite this, studies have not compared image quality at 3T to 1.5T, though it is assumed to be higher at 3T.

The initial aim of this study was to determine the diagnostic performance of MPRAGE in detecting intraplaque hemorrhage versus lipid/necrosis. The second aim was to determine if the image quality at 3T was retained at 1.5T. The third aim was to determine inter- and intraobserver agreement of the MPRAGE sequence at 3T and 1.5T.

Methods

Patient Population

IRB approval was obtained for a 2-year retrospective review of all 362 patients undergoing carotid MRI at the University of Utah from 2009 to 2011. Images were obtained using the 3-dimensional MPRAGE sequence on Siemens 3T and 1.5T MRI scanners. Potential confounding variables were recorded including age and sex. At 1.5T, 282 patients were scanned, yielding 564 carotids, 2 of which were occluded. At 3T, 32 patients (64 carotids) underwent MRI with standard coils, and 48 patients (90 carotids, 6 occluded) underwent MRI using custom-made bilateral dual-element phased array coils.²⁰ There was no significant difference in image quality at 3T with standard coils versus custom coils ($3.84 \pm .09$ vs. $3.89 \pm .09$, $\pm$ SEM, $P=.70$). At 1.5T, all carotid MPRAGE images were obtained with standard neck coils. Of the 362 patients, 12 were recruited for an IRB-approved histologic

study on patients undergoing carotid endarterectomy. In this subset, we determined the ability of carotid MPRAGE to detect intraplaque hemorrhage.

Carotid MPRAGE Sequence at 3T and 1.5T

Three-dimensional MPRAGE parameters were optimized at 3T and were as follows: TR/TE/TI = 6.39/2.37/370 ms, flip angle = 15°, FOV = 130 × 130 × 48 mm³, matrix 256 × 256 × 48, voxel = .5 × .5 × 1.0 mm³, fat saturation, time ~5 minutes. The TI was initially optimized for 3T and transferred to 1.5T. An initial TI of approximately 500 ms was chosen based on prior computer simulations at 3T and was adjusted down to a TI of 370 ms to maximize contrast between hemorrhage and inflowing blood in human volunteers as described previously.^{18,20} Images were obtained from 20 mm below to 20 mm above the carotid bifurcation at 1.0mm slice thickness. In order to produce 3-dimensional images, a secondary phase encoding gradient was used in the slice select direction and measurements for all slice selection phase encodings were performed with rapid acquisition in each segment.

Carotid MPRAGE Interpretation

MPRAGE positive plaque was defined by at least 1 voxel with at least 2-fold higher signal intensity relative to adjacent sternocleidomastoid muscle.⁶ MPRAGE status was determined independently by two radiologists (JSM) and (HCY), blinded to patient characteristics, histology results and independent of adjacent images. In a subset of 12 patients undergoing carotid endarterectomy, the radiologists outlined areas of MPRAGE positive plaque to compare with areas of intraplaque hemorrhage and lipid/necrosis as defined by histology.

Histology Processing

In the subset of 12 patients undergoing carotid endarterectomy, each specimen was fixed in 10% neutral buffered formalin for 3 days in preparation for histology. The ratio of fixative to specimen was at least 10:1. Specimens were decalcified in 1% Enhanced Decalcification Formulation™. The sections were submitted sequentially from inferior to superior in plastic tissue cassettes. Tissue cassettes were processed on an automated Sakura Vacuum Infiltrating Processor, embedded in paraffin wax, sectioned at 3–4 mm intervals and stained with hematoxylin and eosin (H&E) and Mallory's phosphotungstic acid hematoxylin (PTAH) for fibrin.

Histology Interpretation of Intraplaque Hemorrhage and Lipid/Necrosis

A pathologist outlined recent intraplaque hemorrhage and lipid/necrosis using H&E and PTAH stains, blinded to MPRAGE results. "Recent" intraplaque hemorrhage was defined by any of the following: intact red blood cells or degenerated red blood cells on H&E with PTAH stain positive for fibrin. Prior research has demonstrated that fibrin corresponds to recent intraplaque hemorrhage, within 6 weeks of age.²¹ Lipid/necrosis was detected on H&E stain and defined as any of the following: extracellular lipid droplet accumulation, cholesterol clefts or acellular granular debris (necrosis). The carotid bifurcation and gross morphology were used as anatomic guides to pair each MPRAGE slice with the corresponding histology slide. Each carotid MPRAGE positive area was then compared with

(1) intraplaque hemorrhage area, (2) lipid/necrosis area overlapping with intraplaque hemorrhage, (3) lipid/necrosis area without intraplaque hemorrhage, and (4) total plaque area.

Image Quality and Inter- and Intraobserver Agreement at 3T Versus 1.5T

MPRAGE status was determined in each complete arterial segment in patients undergoing carotid MPRAGE at 3T versus 1.5T. MPRAGE image quality was recorded on a scale of 1–5 with 1 = *poor*, 2 = *fair*, 3 = *good*, 4 = *very good*, and 5 = *excellent*. Reasons for image degradation were also recorded (“motion” = motion degradation, “fat sat” = incomplete local fat saturation resulting in high signal in adjacent fat and sternocleidomastoid, “flow” = high intraluminal signal due to flow artifact, “signal” = borderline MPRAGE signal due to small size or low signal). Interobserver agreement was determined between observer 1 and 2. Intraobserver agreement was also determined for observer 1.

Statistical Analysis

To determine the diagnostic ability of the MPRAGE sequence in detecting hemorrhage versus lipid/necrosis, we compared MPRAGE positive area to histology-defined hemorrhage and lipid/necrosis areas (all measured as mm²). We used a random intercept linear regression model with an autoregressive correlation structure. In this repeated measures analysis of the 12 subjects, the “time” repetition variable was the slice position as measured in mm relative to the bifurcation. Using MPRAGE area as the outcome variable, our predictor variables were analyzed one at a time and included: (1) intraplaque hemorrhage area (with or without lipid/necrosis), (2) lipid/necrosis area overlapping with intraplaque hemorrhage, (3) lipid/necrosis area without intraplaque hemorrhage, and (4) total plaque area. In the final model, all of the above predictor variables were included. In addition, a secondary analysis was performed with (1) MPRAGE area on the adjacent inferior slice and (2) MPRAGE signal on the adjacent superior slice.

To determine image quality between 3T and 1.5T, mean image quality was first compared between 3T and 1.5T using a *t*-test. This was followed by linear regression analysis on the outcome (image quality) and the primary predictor (magnet strength: 3T or 1.5T). Finally, a multivariable linear regression analysis was performed with confounders of BMI, age, male sex and disease severity (MPRAGE positive plaque). Missing BMI data met the <.05 threshold proportion of missing data, and any missing values were imputed with the median nonmissing value.²²

Kappa analysis was used to calculate inter- and intraobserver agreement. PABAK was also used. Significant differences in image quality were determined by 2-tailed *t*-tests.

Results

Patient Population

A total of 362 patients (724 carotid arteries) were scanned. Since the MPRAGE sequence imaged both carotid arteries in each patient, this provided 724 carotid arteries. Eight carotid arteries were excluded due to occlusion (6 at 3T, 2 at 1.5T), leaving 716 carotid arteries for

analysis (154 at 3T and 562 at 1.5T). Mean patient age was significantly higher at 3T compared to 1.5T (3T: mean 67.7, SD 12.9; 1.5T: mean 59.5, SD 19.1; $P < .0001$). Mean body mass index was not significantly different at 3T compared to 1.5T (BMI, 3T: mean 26.5, SD .36; 1.5T: mean 27.3, SD 5.6, $P = .13$). The percentage of MPRAGE positive plaque was significantly greater at 3T compared to 1.5T (20.1% compared to 4.8%, chi-squared $P < .001$). The percentage of males was significantly greater at 3T compared to 1.5T (75.3% compared to 44.5%, chi-squared $P < .001$).

MPRAGE Positive Plaque

MPRAGE positive plaque was defined by at least 1 voxel with at least 2-fold higher signal intensity relative to adjacent sternocleidomastoid muscle as shown in Figure 1.

Carotid Plaque Histology and MPRAGE Signal

One hundred carotid MPRAGE image/histology pairs from 12 patients who underwent surgery were analyzed using a linear mixed model. In the linear regression model using subject and slice position for the group and time variables, histology-defined area of intraplaque hemorrhage significantly predicted MPRAGE positive area (slope = .52, $P < .001$, 95%CI: .41, .64, Fig 2A). Overlapping area of lipid/necrosis with intraplaque hemorrhage also significantly predicted MPRAGE positive area (slope = .15, $P = .04$, 95%CI: .01, .29, Fig 2B). Lipid/necrosis without intraplaque hemorrhage was not a significant predictor of MPRAGE positive area (slope = .01, $P = .846$, 95%CI: -.13, .16, Fig 2C). Total plaque area was a significant predictor of MPRAGE positive area (slope = .20, $P < .001$, 95%CI: .10, .30, Fig 2D). In the final model, with all of the above predictors included, only intraplaque hemorrhage remained a significant predictor (slope = .51, $P < .001$, 95%CI: .36, .65, Table 1(A)). On a secondary analysis, intraplaque hemorrhage remained a significant predictor of MPRAGE positive area even when controlling for MPRAGE area on the adjacent inferior slice and MPRAGE signal on the adjacent superior slice (Table 1(B)). Representative histology of a large central area of intraplaque hemorrhage with more peripheral areas of lipid/necrosis compared to MPRAGE area is shown in Figure 3. A comparison of MPRAGE positive and negative plaques with intraplaque hemorrhage and lipid/necrosis is shown in Figure 4. Kappa values for inter- and intraobserver agreement on MPRAGE hyperintensity in this segment of the study were .818 and .806, respectively, indicating very good agreement.

Carotid MPRAGE Interobserver Agreement

The interobserver agreement of carotid MPRAGE was determined at 3T versus 1.5T with pooled data and interobserver agreement (Kappa and PABAK) shown in Tables 2 and 3, respectively. Contingency tables for the Kappa analysis are shown in Table 2. Overall, the Kappa value for interobserver agreement was .812. After adjusting for prevalence and bias, the overall PABAK for interobserver agreement was .944. In addition, Kappa values for interobserver agreement were calculated separately at 3T and 1.5T. The Kappa value for interobserver agreement at 3T was .807 and was .803 at 1.5T. After adjusting for prevalence and bias, the PABAK at 3T was .883 and was .961 at 1.5T.

Carotid MPRAGE Intraobserver Agreement

The intraobserver agreement of carotid MPRAGE was determined at 3T versus 1.5T for observer 1 with pooled data and Kappa and PABAK values shown in Tables 4 and 5, respectively. Overall, the Kappa value for intraobserver agreement was .899. After adjusting for prevalence and bias, the overall PABAK for intraobserver agreement was .969. At 3T, the intraobserver Kappa value was .919 and at 1.5T was .871. After adjusting for prevalence and bias, the PABAK at 3T was .948 and at 1.5T was .975.

Carotid MPRAGE Image Quality

Image quality was rated on a scale of 1–5 and reasons limiting image quality were recorded and pooled data shown in Tables 6–8. Mean image quality (from observers 1 and 2) at 3T was 3.87, significantly lower compared to 4.34 at 1.5T (Table 6). For observer 1 alone, the mean image quality at 3T was 3.80, significantly lower than the image quality at 1.5T of 4.46, $P < .0001$. Similarly for observer 2, the mean image quality at 3T was 3.95, significantly lower than the image quality at 1.5T of 4.21, $P = .002$.

In the linear regression model before controlling for confounding factors, magnet strength was a significant predictor of image quality, with image quality decreasing at 3T compared to 1.5T (slope = $-.46$, $P < .001$, 95%CI: $-.60$, $-.33$, Fig 4B). That is, image quality at 3T was .46 lower than 1.5T on a scale from 1 to 5 (with corresponding means above). In the multivariable linear regression model, confounding factors (BMI, age, male sex, and disease severity [MPRAGE positive plaque]) were included and the results are shown in Figure 4(C). Magnet strength remained a significant predictor of image quality, with image quality decreasing at 3T compared to 1.5T (slope = $-.41$, $P < .001$, 95%CI: $-.54$, $-.27$). Other significant predictors of image quality included BMI (slope = $-.02$, $P < .001$, 95%CI: $-.03$, $-.01$) and age (slope = $-.01$, $P < .001$, 95%CI: $-.01$, $-.002$), indicating image quality minimally, but significantly, decreased with increasing BMI and age. Male sex and MPRAGE positive plaque were not significant predictors of image quality.

Reasons for Diminished MPRAGE Image Quality at 3T Versus 1.5T

The percentage of scans with limitations due to motion, fat saturation failure, flow artifact and borderline signal were recorded in Table 9. At 3T, 36.4% of images had image quality decreased due to motion, 39.0% due to local fat-saturation failure, and 22.7% due to intraluminal flow artifact. This was significantly different from 1.5T, with 2.3% images limited by motion, 2.8% from local fat-saturation failure, and 9.3% due to flow artifact (two-tailed $P < .002$). Image quality was also decreased due to borderline signal at both 3T (8.4% of images) and 1.5T (7.1%) but this did not satisfy the binomial requirement to test for a significant difference.

Image Quality in Cases of Interobserver Disagreement

Of the 20 arteries with interobserver disagreement, the average quality rating was 3.58, SEM = .13 compared to the overall average quality rating of 4.34, SEM = .028. In these 20 arteries, 30% had quality degraded by motion, 35% by local fat-saturation failure, 35% by flow artifact, and 10% by borderline signal. This was compared to overall limitations of 25%, 13%, 14%, and 4%, respectively. Although fewer studies were performed at 3T (154

carotids) compared to 1.5T (562 carotids), there was a higher prevalence of disagreement within the 3T cases ($9/154 = 5.8\%$) compared to 1.5T ($11/562 = 2.0\%$).

Discussion

The importance of carotid intraplaque hemorrhage in stroke has been known since pathology studies dating back to 1982.^{23–25} However, it was not until recently that carotid intraplaque hemorrhage could be imaged in a non-invasive manner. In 2003, Murphy and colleagues were the first to demonstrate complex plaque has high signal using a magnetic resonance direct thrombus imaging (MRDTI) technique.²⁶ MRI has since demonstrated unparalleled sensitivity and specificity in detecting intraplaque hemorrhage compared with ultrasound, CT angiography, and digital subtraction angiography.¹⁶

Still, controversy has existed concerning the specificity of T1 weighted sequences in detecting hemorrhage, since other plaque components such as lipid/necrosis can also be T1 hyperintense.^{12,27} In 2010, Ota used the MPRAGE sequence and demonstrated significantly higher sensitivity (80%) and specificity (97%) in detecting intraplaque hemorrhage compared to conventional T1 weighted or time-of-flight (TOF) sequences, using histology as a gold standard.¹⁹ In 2008, Bitar used a T1 weighted sequence in which 97 images were compared with histology with a similar accuracy, specificity and sensitivity (87–90%, 80–88%, and 94–100%, respectively).²⁸ Still, no prior studies have addressed the ability of the MPRAGE sequence to discriminate between intraplaque hemorrhage and lipid/necrosis.

In this study, the area of intraplaque hemorrhage was found to be highly predictive of MPRAGE positive area outlined by radiologists using a 2-fold signal threshold over adjacent sternocleidomastoid. This is in contrast to lipid/necrosis, which was not predictive of MPRAGE signal. The choice of TI time in the MPRAGE sequence may result in decreased signal of lipid/necrosis compared to what is seen in conventional T1w sequences. These results bridge the gap between pathologic studies demonstrating a higher association of intraplaque hemorrhage with stroke than lipid/necrosis and more recent research demonstrating MPRAGE signal in association with stroke.^{6–8,17}

For the MPRAGE sequence to transition from 3T to the 1.5T scanners used in many institutions for acute stroke work up, inter- and intraobserver agreement should remain high at both magnet strengths. In applying the MPRAGE sequence to a large clinical population at 1.5T with standard neck coils, this MPRAGE had very good inter- and intraobserver agreement similar to 3T. Even after adjusting for prevalence and bias, the PABAK values at 1.5T remained high, much like those at 3T. These results argue that carotid intraplaque hemorrhage detection with MRI, previously isolated to 3T research subjects, can be used at 1.5T.

Surprisingly, image quality was rated higher at 1.5T compared to 3T, even after controlling for confounders (BMI, age, male sex and MPRAGE positive signal). A significantly higher proportion of 3T images were degraded due to motion, local fat-saturation failure and flow artifact compared to the 1.5T images. These limitations of high-field-strength imaging are well known.²⁹ Identifying and eventually correcting these sequence limitations is important

as carotid MPRAGE is increasingly added to clinical evaluation of patients with suspected stroke.

Motion artifacts are secondary to view-to-view phase errors that occur when the signal amplitude at a specific location is not consistent between phase-encoding steps. The result is signal amplitude variation and ghosting artifact. The artifact intensity is proportional to motion amplitude, signal intensity of the moving structure, and magnetic field strength.

Furthermore, reduced efficacy of frequency-selective fat-saturation pulses is a known complication of 3T imaging and is secondary to B_0 inhomogeneity. The variations in susceptibility are twice as large at 3T than at 1.5T. These artifacts may be mitigated by improved field shimming and optimal positioning of the patient in the z -direction since these effects are more pronounced the further the image slice is positioned from isocenter.³⁰

Lastly, flow related artifacts can be worse at 3T because of both increased SNR and field inhomogeneity due to susceptibility variations.³¹ Because susceptibility-induced inhomogeneity increases with field strength, the resultant artifacts are also increased at 3T compared to 1.5T.³² Despite the initial optimization of the MPRAGE sequence at 3T by lowering the TI to minimize blood signal, flow artifacts were still increased at 3T compared to 1.5T.

We have recently developed the ability to limit cardiac pulsation and flow artifact in the MPRAGE sequence, by applying cardiac gating and post-processing to create a cineMPRAGE.³³ The cineMPRAGE sequence may reduce or eliminate artifact related to motion and flow, and may significantly improve interobserver agreement. Additional methods such as the slab-selective phase-sensitive inversion-recovery (SPI) or simultaneous noncontrast angiography and intraplaque hemorrhage (SNAP) sequences may also play a role by identifying flow related signal artifact and separating this from intraplaque hemorrhage.^{34,35}

In conclusion, intraplaque hemorrhage is a strong predictor of MPRAGE positive signal as opposed to lipid/necrosis. Image quality is retained and slightly higher at 1.5T compared to 3T. At both field strengths, the MPRAGE sequence has a very high inter- and intraobserver agreement and can therefore be used for both clinical and research applications.

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Fig 1.

Carotid MPRAGE positive plaque. MPRAGE positive plaque (*) was defined using a threshold of 2-fold signal intensity of a point value in the internal carotid artery (ICA) plaque compared to an ROI placed on the adjacent sternocleidomastoid (SCM). 859 mm × 455 mm (96 × 96 DPI).

Fig 2.

(A) Scatter plot demonstrating correlation between MPRAGE positive area and intraplaque hemorrhage area. (B) Scatter plot demonstrating correlation between MPRAGE positive area and lipid/necrosis area overlapping with intraplaque hemorrhage area. (C) Scatter plot demonstrating correlation between MPRAGE positive area and lipid/necrosis area without intraplaque hemorrhage. (D) Scatter plot demonstrating correlation between MPRAGE positive area and total plaque area.

Fig 3. Carotid MPRAGE comparison of intraplaque hemorrhage and lipid/necrosis. Representative MPRAGE positive plaque in a patient undergoing subsequent endarterectomy (left). H&E stain demonstrating recent intraplaque hemorrhage (solid line) and lipid/necrosis (dashed line) outlined by a pathologist (middle). Within the area outlined as recent intraplaque hemorrhage on H&E, PTAH positive staining indicates fibrin deposition (right).

Fig 4. Representative MPRAGE images in two patients undergoing subsequent endarterectomy. These images demonstrate carotid plaque with a large area of lipid/necrosis (right image, left ICA) versus a large area of intraplaque hemorrhage (left image, right ICA).

Table 1

MPRAGE area Multivariable Linear Regression (A) Final Model (B) Secondary Analysis

	Slope	P	95%CI	
(A) Final model				
Intraplaque hemorrhage (IPH)	.51	<.001	.36	.65
Lipid/necrosis w/IPH	.07	.392	-.09	.23
Lipid/necrosis w/o IPH	.07	.488	-.12	.25
Total plaque area	.05	.675	-.05	.16
(B) Secondary analysis				
Intraplaque hemorrhage (IPH)	.21	.001	.08	.33
Lipid/necrosis w/IPH	-.05	.562	-.20	.11
Lipid/necrosis w/o IPH	-.04	.512	-.15	.07
Total plaque area	-.02	.528	-.07	.03
MPRAGE area inferior	.62	<.001	.54	.70
MPRAGE area superior	.42	<.001	.36	.49

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Table 2

Interobserver Agreement					
	Obs 2		PABA		
	Overall	(-) (+)	(-) (+)	(-) (+)	(-) (+)
	(-)	648	10	(-) 348	10
	(+)	10	48	(+) 10	348
	3T				
Obs 1	(-)	121	2	(-) 73	5
	(+)	7	24	(+) 4	72
	1.5T				
	(-)	527	8	(-) 276	6
	(+)	3	24	(+) 5	275

Table 3

Interobserver Agreement

	Kappa	Std. error	2-sided 95%CI	
Overall	.812	.041	.732	.892
3T	.807	.062	.685	.929
1.5T	.803	.058	.689	.917

	PABAK	Std. error	2-sided 95%CI	
Overall	.944	.012	.920	.968
3T	.883	.038	.809	.957
1.5T	.961	.012	.937	.985

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Table 4

Intraobserver Agreement					
	Obs 1B		PABA		
	Overall	(-)	(+)	(-)	(+)
	(-)	651	4	(-) 353	6
	(+)	7	54	(+) 5	352
3T					
Obs 1A	(-)	121	2	(-) 75	2
	(+)	2	29	(+) 2	75
1.5T					
	(-)	530	2	(-) 278	4
	(+)	5	25	(+) 3	277

Table 5

Intraobserver Agreement

	Kappa	Std. error	2-sided 95%CI	
Overall	.899	.030	.840	.958
3T	.919	.040	.841	.997
1.5T	.871	.048	.777	.965

	PABAK	Std. error	2-sided 95%CI	
Overall	.969	.009	.951	.987
3T	.948	.026	.897	.999
1.5T	.975	.009	.957	.993

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Table 6

Carotid MPRAGE Image Quality

Observer 1 and 2		Mean	Stddev	SEM	n	2-tailed P
3T		3.87	.79	.06	154	
	1.5T	4.34	.71	.03	562	<.0001

Observer 1		Mean	Stddev	SEM	n	2-tailed P
3T		3.80	.87	.07	154	
	1.5T	4.46	.73	.03	562	<.0001

Observer 2		Mean	Stddev	SEM	n	2-tailed P
3T		3.95	.96	.08	154	
	1.5T	4.21	.90	.04	562	.002

Table 7

Linear Regression Model on Image Quality

	Slope	<i>P</i>	95%CI	
Magnet strength (3T vs. 1.5T)	-.46	<.001	-.60	-.33

Table 8

Multivariable Linear Regression Model on Image Quality

	Slope	<i>P</i>	95%CI	
Magnet strength (3T vs. 1.5T)	-.41	<.001	-.54	-.27
BMI	-.02	<.001	-.03	-.01
Age	-.01	<.001	-.01	-.002
Male sex	-.07	.203	-.18	.04
MPRAGE positive plaque	-.04	.703	-.25	.17

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Table 9

Decreased MPRAGE Image Quality at 3T Versus 1.5T

	Motion	P	Fat-sat	P	Flow	P	Signal	P	n
3T	36.4%		39.0%		22.7%		8.4%		154
1.5T	20.3%	<.002	2.8%	<.002	9.3%	<.002	.7%	NS	562