For over 20 years, determination of carotid bulb sources of stroke (carotid-source stroke) has been based primarily on lumen stenosis. In 1991, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) cemented the role of carotid stenosis as a stroke predictor, leading to our current approach for intervention.1 Carotid stenosis is measured using the formula 

\[
\frac{a-b}{a} \times 100\% 
\]

where (b) is the diameter at the level of maximal stenosis and (a) is the diameter of the internal carotid artery (ICA) distal to the stenosis.2 The calculation of percent stenosis is subject to 2 main pitfalls. First is identifying near-occlusions, when the ICA diameter (a) is decreased beyond an extreme stenosis, making any ratio calculation fallacious.2–4 The second pitfall is measuring the ICA diameter (a), which must be well beyond ICA bulb where the walls are parallel.2 In symptomatic patients with ≥70% stenosis, NASCET found that surgery outperformed medical therapy with a number needed to treat of 6. Soon after NASCET, the Asymptomatic Carotid Artery Stenosis (ACAS) trial found surgery of benefit in asymptomatic patients with ≥60% stenosis with a higher number needed to treat of 19.5 Despite proven benefit of endarterectomy, most patients with carotid disease will not have a subsequent stroke. If stenotic but stable plaques could be identified, many surgeries and healthcare expenses could, therefore, be eliminated.

Since NASCET, advances have been made in noninvasive carotid imaging. Computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) are as accurate as digital subtraction angiography (DSA) in determining lumen...
stenosis and both much more accurate than duplex ultrasound. Carotid plaque ulceration has long been an indicator for surgery but can be difficult to detect with DSA. Ulceration can be detected with CTA at a threshold of ≥2 mm with high sensitivity (87%) and specificity (99%) compared with histology. Intraluminal thrombus is an additional lumen marker highly associated with stroke and can be detected on CTA as a donut sign. Despite these advances, stenosis remains the primary measurement to determine carotid sources of stroke, future stroke risk, and potential benefit of surgical versus medical therapy.

In the early 2000s, high carotid wall signal on MRI was first shown to detect complex atheromas by Moody et al. Recently, MRI sequences including the heavily T1-weighted magnetization-prepared rapid acquisition gradient echo (MPRAGE) sequence have been used to detect intraplaque hemorrhage (IPH) and are much more sensitive and specific compared with conventional time-of-flight (TOF) and T1-weighted images. MPRAGE-positive plaque is now an additional predictor of carotid-source stroke, with increased stroke risk for all degrees of stenosis. Still, it is unclear whether IPH is only a surrogate for other markers of unstable plaque, including plaque thickness, ulceration, or intraluminal thrombus. Recent research suggests that CTA-detected ulceration may be used as a surrogate for IPH. If ulceration or other lumen markers could be used as true surrogates for IPH, carotid MRI may not add significant value in detecting stroke sources. Until now, however, no studies have addressed the relative contributions of IPH along with all other available imaging and clinical markers of stroke risk in a multivariable analysis.

With this in mind, our goal was to determine which carotid imaging characteristics best predict ipsilateral carotid-source stroke. We initiated the MPRAGE sequence as part of the clinical protocol in November 2009. The data were gathered and analyzed retrospectively, as a cross-sectional study. Our hypothesis was that IPH would be an essential imaging marker of stroke, even after controlling for known clinical and imaging markers of stroke risk. To evaluate the potential importance of carotid IPH, we included all patients undergoing acute stroke workup with carotid MRA over the course of 4.5 years. Using a multivariable logistic regression model, essential imaging and clinical markers of carotid-source stroke were determined.

Methods

Clinical Study Design

Institutional review board approval was obtained for this retrospective cross-sectional study on patients undergoing stroke evaluation with brain MRI/carotid MRA at the University of Utah Medical Center from November 2009 to January 2014. The carotid MPRAGE sequence was obtained in all patients (578) undergoing carotid MRA stroke workup beginning in November 2009. All patients were scanned within 1 week of symptom onset. This resulted in 1156 carotid artery-ipsilateral brain image pairs.

Exclusions were determined after reviewing electronic medical records for noncarotid plaque stroke sources, those outside of the 2 cm above and 2 cm below the carotid bifurcation. A total of 420 carotid-brain image pairs were excluded, including craniocervical dissections (118), atrial fibrillation (94), intracardiac/extracardiac shunt (86), cardiac thrombus (26), recent aortic or mitral valve replacement (16), vasculitis (14), global hypoxic/ischemic injury (10), recent cardiac or neurovascular catheterization (10), recent cardiovascular surgery (8), dural venous sinus thrombosis (8), fibromuscular dysplasia or lupus vasculopathy (8), proximal carotid stenosis >50% (6), rheumatic heart disease (4), brain neoplasm (4), endocarditis (2), idiopathic hypertrophic subaortic stenosis (2), aortic graft complication (2), and distal vessel atherosclerosis (2). We also excluded carotid occlusions (7) and near-occlusions (3) because lumen markers cannot accurately be assessed. Seven hundred twenty-six carotid-brain image pairs were used in the final analysis. Although a few scans exhibited mild motion artifacts primarily from swallowing, these artifacts were not sufficient to exclude any carotid images from interpretation.

MRI/MRA Clinical Protocol

Images were obtained on Siemens 3-T and 1.5-T MRI scanners with standard head/neck coils. Standard clinical MRI/MRA protocol included brain MRI (axial diffusion-weighted images [DWI], axial T2-weighted, axial fluid attenuation inversion recovery, and sagittal T1-weighted images), brain MRA (3-dimensional [3D] axial TOF), and neck MRA (2D axial TOF, coronal precontrast T1-weighted, coronal postcontrast arterial and venous phase images). Neck MRA was obtained from the aortic arch through the circle of Willis. Total scan time was ≈45 minutes, of which MPRAGE required ≈5 minutes. In cases when renal insufficiency precluded intravenous contrast (glomerular filtration rate <30 mL/min per 1.73 m2), postcontrast MRA images were replaced with 3D noncontrast TOF with 1 mm slice thickness combined with duplex ultrasound.

Carotid MPRAGE Sequence

MPRAGE parameters were first optimized at 3T and were as follows: 3D, repetition time/echo time/time to inversion=6.39/2.37/370 ms, flip angle=15°, field of view=130×130×48 mm3, matrix=256×256×48, voxel size=0.50×0.50×0.70 mm3, fat saturation, time saturation = 5 minutes. The time to inversion was initially optimized for 3T and transferred to 1.5T. An initial time to inversion of ≈500 ms was chosen based on prior computer simulations at 3T and was adjusted down to a time to inversion of 370 ms to maximize contrast between hemorrhage and inflowing blood in human volunteers as described previously. Images were obtained from 20 mm below to 20 mm above the carotid bifurcation at 1.0 mm slice thickness. To produce 3D 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.

Imaging Markers of Carotid-Source Stroke

All carotid imaging markers were determined by consensus review of 2 reviewers (J.S.M. and M.S.M.), blinded to brain MRI findings and clinical covariates. In addition, IPH was determined independent of other carotid imaging markers of stroke risk. Lumen markers included stenosis, maximum plaque thickness, ulceration, and intraluminal thrombus.

Percent diameter stenosis was determined using NASCET criteria on contrast MRA, using the formula [(a−b)/a]×100% where (b) is the diameter of maximal stenosis and (a) is the diameter of the ICA distal to the stenosis. Carotid stenosis was measured at the narrowest segment of the carotid plate (b) on the axial images, perpendicular to the long axis of the vessel on multiplanar reformats using a sub-mm measurement tool (Figure 1A). The distal ICA diameter (a) was measured beyond the bulb where the walls are parallel and no longer tapering per NASCET. We performed the multivariable regression analysis using both NASCET stenosis [(a−b)/a]×100% and mm stenosis (b), adapted from the mm stenosis method first described on CTA. Near-occlusions were defined using the following criteria: visible bulb stenosis, distal ICA diameter ≤3 mm, and distal ICA/distal external carotid artery ratio of ≤1.25. These criteria were adapted from Bartlett et al to recognize near-occlusions on CTA, and originally adapted from DSA.
Ulceration was determined on contrast MRA images using a size threshold of 2 mm, previously described with CTA.\textsuperscript{15} Intraluminal thrombus was determined using contrast MRA multiplanar reformats and defined by a noncontrast filling defect outlined by lumen contrast. On axial images, this often manifests as a donut sign, previously described on CTA.\textsuperscript{3} Each of the 19 subjects with intraluminal thrombus had a contrast MRA study. Moreover, the majority of these patients had confirmation with another imaging method CTA (15), DSA (1), or ultrasound (1). In arteries from patients with renal failure (glomerular filtration rate <30 mL/min per 1.73 m\textsuperscript{2}, 68/726 or 9.4\% of carotid-brain image pairs), the above imaging markers were determined from 3D noncontrast TOF with 1 mm slice thickness combined with duplex ultrasound. Maximum plaque thickness was measured in the transverse plane on 3-dimensional magnetization-prepared rapid acquisition gradient echo (MPRAGE) images \textsuperscript{D}. Intraplaque hemorrhage was defined by MPRAGE-positive plaque, using a signal threshold of 2-fold signal intensity over adjacent sternocleidomastoid muscle \textsuperscript{D}, right carotid is MPRAGE-positive*, image left).

\section*{Acute Stroke Determination}
Recent carotid territory infarct was defined using the DWI technique derived from diffusion tensor imaging (DTI) trace images from our standard clinical imaging protocol, as previously described.\textsuperscript{12} DTI trace images have been shown to be superior to conventional diffusion-weighted sequences in detecting recent infarcts.\textsuperscript{19} Briefly, DTI parameters were 2D, 128x128 matrix, 3 mm slice thickness, B value=2000, 22 directions. Although the referring clinician suspected a possible recent infarct, only objective DWI images were used to determine if a recent infarct was present. DWI positivity was defined as hyper intense signal on DTI trace corresponding to a recent infarct at the time of the scan.\textsuperscript{20,21} Embolic infarcts were defined as those involving the cortex or subcortical white matter, whereas microvascular infarcts were defined as those involving only the basal ganglia or adjacent white matter. Only DWI positive embolic infarcts in the ipsilateral ICA territory were placed in the DWI positive category and presumably represented carotid-source strokes after excluding other stroke sources under our clinical study design. Brain DWI images were interpreted by a subspecialty trained, certificate of added qualification–certified neuroradiologist blinded to carotid imaging.

\section*{Statistical Analysis}
A mixed effects multivariable logistic regression model was used and accounted for 2 vessels per patient. The multivariable logistic regression model was fitted for the outcome of ipsilateral stroke. Carotid imaging predictors included stenosis, maximum plaque thickness, ulceration, intraluminal thrombus, and IPH. Clinical covariates included age, male sex, diabetes mellitus, hypertension, hyperlipidemia, and body mass index. Cardiovascular medication confounders included antihypertension, antiplatelet, anticoagulation, and statin medication classes. Magnet strength (3T or 1.5T) was also included as a potential confounder in the logistic regression model. A backwards elimination method was used to determine the final model, in which all remaining predictors had a \(P<0.10\). Odds ratios (ORs) and \(P\) values were reported for each factor alone and for the factors found to be significant from the backward elimination. Receiver operating characteristic comparison analysis was performed to determine the discriminatory value of the final model (with IPH) compared with (1) the final model without IPH, (2) a model using only percent stenosis as a continuous variable, and (3) a model using only percent stenosis with standard clinical cutoffs of 50\% and 70\%. All statistical analyses were performed with Stata version 13.1.

\section*{Results}

\subsubsection*{Patient Demographics}
Patient demographics are listed by vessel and depicted in Table 1.

\subsection*{Carotid Plaque Markers and Stroke Imaging}
The carotid plaque features measured in this study included percent diameter stenosis, mm stenosis, maximum plaque thickness, ulceration, intraluminal thrombus, and IPH. Carotid stenosis was measured using percent diameter stenosis \([(a−b)/a]\times100\%\) and mm stenosis (b) in Figure 1A. Ulceration was determined on contrast MRA images using a 2 mm measurement threshold in Figure 1B. Intraluminal thrombus was defined as a filling defect on contrast MRA images in Figure 1C. Maximum plaque thickness was measured in the transverse plane on 3D MPRAGE images in Figure 1D. IPH was defined by MPRAGE-positive plaque, using a signal threshold of 2-fold signal intensity over adjacent sternocleidomastoid muscle in Figure 1D, with an MPRAGE-positive right carotid plaque, image left. Recent carotid territory embolic stroke was defined using DWI (DTI trace) in Figure 2.
Carotid plaque imaging markers

Intraplaque hemorrhage, n/total n (%) 65/726 (9.0)
Intraluminal thrombus, n/total n (%) 19/726 (2.6)
Stenosis, mean (SD), mm 3.0 (1.6)
Percent stenosis, mean (SD) 12.2 (23.1)
Anticoagulation, n/total n (%) 74/726 (10.2)
Statin, n/total n (%) 316/726 (43.5)
Antiplatelet, n/total n (%) 294/726 (40.5)
Statin, n/total n (%) 316/726 (43.5)
Antihypertension, n/total n (%) 412/726 (56.8)
Diabetes mellitus, n/total n (%) 227/726 (31.3)
Hyperlipidemia, n/total n (%) 358/726 (49.3)
Hypertension, n/total n (%) 499/726 (68.7)
Smoking, n/total n (%) 138/726 (19.0)
Antihypertension, n/total n (%) 316/726 (43.5)

Table 1. Patient Demographics

Cardiovascular risk factors

Male sex, n/total n (%) 387/726 (53.3)
Age, mean (SD), y 64.2 (15.6)
Body mass index (BMI), mean (SD), kg/m² 28.4 (6.4)
Smoking, n/total n (%) 158/726 (21.8)
Never smoker
Current smoker
Prior smoker

Cardiovascular medications

Antihypertension, n/total n (%) 316/726 (43.5)
Antiplatelet, n/total n (%) 294/726 (40.5)
Anticoagulation, n/total n (%) 74/726 (10.2)
Statin, n/total n (%) 316/726 (43.5)
Antihypertension, n/total n (%) 412/726 (56.8)

Figure 2. Embolic stroke detected with diffusion-weighted images (DWI; diffusion tensor imaging [DTI] trace). Recent carotid territory embolic stroke was determined using DWI (DTI trace, left) with apparent diffusion coefficient (ADC) map (right) detecting true diffusion restriction (arrow).

Multivariable Logistic Regression

Carotid-source stroke predictors are depicted in Table 2, with ORs adjusted using multivariable logistic regression.

Final Multivariable Logistic Regression Model

The final model for predictors of carotid-source stroke is depicted in Table 3. After backward elimination with a threshold of $P<0.10$, the remaining significant factors predicting carotid-source stroke included intraluminal thrombus (OR=103.6; $P<0.001$, 95% confidence interval [CI]=12.5–858.1), IPH (OR=25.2; $P<0.001$, 95% CI=11.8–53.7), current smoking (OR=2.78; $P=0.004$, 95% CI=1.39–5.56), and maximum thickness (OR=1.24; $P=0.020$, 95% CI=1.03–1.48). Of these, intraluminal thrombus had the highest positive predictive value of carotid-source stroke (18/19=94.7%). Prior smoking was not a significant predictor of carotid-source stroke (OR=1.69; $P=0.135$, 95% CI=0.85–3.35) but was included in the final regression model because current smoking was significant. Of the imaging findings, neither stenosis nor ulceration was left in the final model because these were not significant predictors of carotid-source stroke.

Receiver Operating Characteristic Comparison Analysis

Receiver operating characteristic comparison analysis is shown in Figure 3. The final model discriminatory value was excellent (area under the curve [AUC]=0.862). The final model discriminatory value (AUC) with IPH was significantly higher than the final model without IPH (AUC=0.814, $P<0.001$). The final model AUC was also significantly higher than a model using only stenosis as a continuous variable (AUC=0.753) or a model using only stenosis with cutoffs of 50% and 70% (AUC=0.683), $P<0.001$.

Discussion

Determining stroke pathogenesis is critically important because treatment directed at stroke sources can prevent future stroke. This study is the first to demonstrate that IPH adds significant discrimination of carotid-source stroke even when using all available lumen markers (stenosis, thickness, ulceration, and intraluminal thrombus). These results suggest that carotid MRI and IPH detection be used in the determination of stroke sources. Currently, CTA use far outweighs the use of MRA and is used $4x$ as often as MRA at our institution. Although CTA has a higher resolution than MRA, both detect lumen stenosis with a similar, high accuracy compared with DSA. In addition, plaque density on CTA is a poor IPH predictor. It is clear from this research that stroke source workup is incomplete if one uses only lumen information from CTA or MRA without IPH-sensitive sequences, such as MPRAGE.

Of potential concern with CT stroke workup is radiation dose, given the carcinogenesis risk extrapolated from other studies. Over the past 30 years, medical radiation sources have increased the average radiation dose per person in the United States from an average of 3.6 mSv in the 1980s to 6.2 mSv in 2006. Extrapolating data from Hiroshima, Three Mile Island, Chernobyl, and radiation workers to current CT use, it has been estimated that 1.5% to 2.0% of all cancers in the United States may be attributable to CT radiation. Acute stroke workup with CT/CTA/CT perfusion results in a mean effective dose of 16.4 mSv, imparting a low cancer risk of 1 in 1200 patients. Effective dose is calculated from the dose length product (mGycm) taking into account the radiosensitivity of imaged organs, 80% of which is attributed to the
One reason for this low risk is that most patients undergoing stroke workup are within the sixth to ninth decades, and exposure in this age group carries a low risk of excess cancer mortality because of decreased dose length product conversion factors. Although the individual risk of cancer with CT stroke workup seems to be small, databases have been developed for future large-scale epidemiological studies addressing CT-radiation risk.

Although both intraluminal thrombus and IPH were required for optimal stroke-source determination in our final regression model, IPH added no further discriminatory value in the relatively rare instance of intraluminal thrombus detection. These findings are perplexing because carotid-source stroke has always been understood to occur by initial thrombus formation and subsequent fragmentation and embolic stroke. These thrombi likely also form with IPH, but they may be below the limits of detection or they may have already embolized to the brain. Confirmation of IPH-induced microthrombi could be obtained with future studies using high-resolution methods including optical coherence tomography.

Table 2. Multivariable Logistic Regression

<table>
<thead>
<tr>
<th>Carotid-Stroke Predictor</th>
<th>Stroke+ (100)</th>
<th>Stroke− (626)</th>
<th>OR</th>
<th>P Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male sex, n/total n (%)</td>
<td>70/100 (70.0)</td>
<td>317/626 (50.6)</td>
<td>1.01</td>
<td>0.979</td>
<td>0.53–1.92</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>68.9 (14.0)</td>
<td>63.5 (15.7)</td>
<td>1.02</td>
<td>0.241</td>
<td>0.99–1.04</td>
</tr>
<tr>
<td>BMI, mean (SD), kg/m²</td>
<td>27.7 (4.2)</td>
<td>28.5 (6.7)</td>
<td>1.00</td>
<td>0.890</td>
<td>0.95–1.06</td>
</tr>
<tr>
<td>Smoking, n/total n (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td>32/100 (32.0)</td>
<td>106/626 (16.9)</td>
<td>3.18</td>
<td>0.003</td>
<td>1.47–6.86</td>
</tr>
<tr>
<td>Prior smoker</td>
<td>28/100 (28.0)</td>
<td>130/626 (20.8)</td>
<td>1.63</td>
<td>0.189</td>
<td>0.79–3.38</td>
</tr>
<tr>
<td>Hypertension, n/total n (%)</td>
<td>68/100 (68.0)</td>
<td>431/626 (68.9)</td>
<td>0.59</td>
<td>0.201</td>
<td>0.26–1.32</td>
</tr>
<tr>
<td>Hyperlipidemia, n/total n (%)</td>
<td>59/100 (59.0)</td>
<td>299/626 (47.8)</td>
<td>0.93</td>
<td>0.829</td>
<td>0.48–1.80</td>
</tr>
<tr>
<td>Diabetes mellitus, n/total n (%)</td>
<td>36/100 (36.0)</td>
<td>191/626 (30.5)</td>
<td>1.19</td>
<td>0.598</td>
<td>0.62–2.28</td>
</tr>
<tr>
<td>Cardiovascular medications, number/total number (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antihypertension</td>
<td>62/100 (62.0)</td>
<td>350/626 (55.9)</td>
<td>1.52</td>
<td>0.282</td>
<td>0.71–3.27</td>
</tr>
<tr>
<td>Statin</td>
<td>50/100 (50.0)</td>
<td>266/626 (42.5)</td>
<td>0.92</td>
<td>0.826</td>
<td>0.43–1.95</td>
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<tr>
<td>Antiplatelet</td>
<td>42/100 (42.0)</td>
<td>252/626 (40.3)</td>
<td>0.61</td>
<td>0.200</td>
<td>0.29–1.29</td>
</tr>
<tr>
<td>Anticoagulation</td>
<td>7/100 (7.0)</td>
<td>67/626 (10.7)</td>
<td>0.50</td>
<td>0.224</td>
<td>0.16–1.53</td>
</tr>
<tr>
<td>Carotid plaque imaging markers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stenosis, mean (SD), %</td>
<td>36.3 (32.6)</td>
<td>7.4 (18.6)</td>
<td>7.44</td>
<td>0.191</td>
<td>0.37–150.5</td>
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<tr>
<td>Stenosis, mean (SD), mm</td>
<td>3.1 (1.6)</td>
<td>4.3 (1.0)</td>
<td>1.33</td>
<td>0.325</td>
<td>0.75–2.34</td>
</tr>
<tr>
<td>Thickness, mean (SD), mm</td>
<td>4.76 (2.1)</td>
<td>2.8 (1.4)</td>
<td>1.18</td>
<td>0.101</td>
<td>0.98–1.45</td>
</tr>
<tr>
<td>Ulceration, n/total n (%)</td>
<td>39/100 (39.0)</td>
<td>57/626 (9.1)</td>
<td>1.02</td>
<td>0.963</td>
<td>0.45–2.33</td>
</tr>
<tr>
<td>Intraluminal thrombus, n/total n (%)</td>
<td>18/100 (18.0)</td>
<td>1/626 (0.2)</td>
<td>78.3</td>
<td>&lt;0.001</td>
<td>6.84–710.8</td>
</tr>
<tr>
<td>Intraplaque hemorrhage, n/total n (%)</td>
<td>48/100 (48.0)</td>
<td>17/626 (2.7)</td>
<td>24.0</td>
<td>&lt;0.001</td>
<td>10.1–57.0</td>
</tr>
<tr>
<td>Magnet strength=3T, n/total n (%)</td>
<td>15/100 (15.0)</td>
<td>43/626 (6.9)</td>
<td>0.66</td>
<td>0.424</td>
<td>0.23–1.84</td>
</tr>
</tbody>
</table>

BMI indicates body mass index; CI, confidence interval; and OR, odds ratio.

Table 3. Final Model

<table>
<thead>
<tr>
<th>Carotid Stroke Predictor</th>
<th>OR</th>
<th>P Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraluminal thrombus</td>
<td>103.6</td>
<td>&lt;0.001</td>
<td>12.5–858.1</td>
</tr>
<tr>
<td>Intraplaque hemorrhage</td>
<td>25.2</td>
<td>&lt;0.001</td>
<td>11.8–53.7</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2.78</td>
<td>0.004</td>
<td>1.39–5.56</td>
</tr>
<tr>
<td>Prior smoker</td>
<td>1.69</td>
<td>0.135</td>
<td>0.85–3.35</td>
</tr>
<tr>
<td>Plaque thickness</td>
<td>1.24</td>
<td>0.020</td>
<td>1.03–1.48</td>
</tr>
</tbody>
</table>

Cl indicates confidence interval; and OR, odds ratio.
established that current smoking activates a proembolic component of carotid plaque. Evidence suggests that smoking results in platelet activation and thrombogenesis in atherosclerotic plaque. Our data further support the role of current smoking in thromboembolism from carotid stroke sources.

Despite our recent research demonstrating a significant difference between 3-T and 1.5-T MPRAGE image quality and intraobserver and interobserver agreement, magnet strength did not significantly confound the association of IPH and stroke. This is likely because the effect of magnet strength on gross MPRAGE positive or negative status is small, on the order of 10% in our recent study. In addition, 3-T magnets are used far less often than 1.5-T magnets in the evaluation of acute stroke at our institution. The results of this study further suggest that IPH detection can and should be used as part of stroke source workup at both magnet strengths.

Although these data are somewhat limited because of their cross-sectional nature, they nonetheless argue against using only stenosis to determine carotid stroke sources. With advances in imaging, evidence supporting stroke risk stratification based on IPH is rapidly accumulating. Furthermore, our data suggest that MRA-detected ulceration is not a significant determinate of carotid-source stroke when IPH is taken into account. These data suggest that carotid MRI and IPH detection be used in carotid-source stroke workup.

**Summary**

Optimal discrimination of carotid-source stroke required information on IPH, intraluminal thrombus, maximum plaque thickness, and smoking history. Ulceration and stenosis do not contribute when other imaging and clinical factors are taken into account. These data suggest that carotid MRI and IPH detection be used in carotid-source stroke workup.

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**Disclosures**

None.

**References**


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