Carotid Atherosclerotic Plaque Characteristics on Magnetic Resonance Imaging Relate With History of Stroke and Coronary Heart Disease

Mariana Selwaness, MD, PhD; Daniel Bos, MD, PhD; Quirijn van den Bouwhuijsen, MD; Marileen L.P. Portegies, MD; M. Arfan Ikram, MD, PhD; Albert Hofman, MD, PhD; Oscar H. Franco, MD, PhD; Aad van der Lugt, MD, PhD; Jolanda J. Wentzel, PhD; Meike W. Vernooij, MD, PhD

Background and Purpose—Because atherosclerosis is a systemic disease, presence and composition on 1 location may relate to ischemic events in distant locations. We examined whether carotid atherosclerotic wall thickness, stenosis, and plaque composition are related to history of ischemic stroke and coronary heart disease (CHD).

Methods—From the population-based Rotterdam Study, 1731 asymptomatic participants (mean age, 72.4±9.1 years; 55% males) underwent magnetic resonance imaging of both carotid arteries. We assessed carotid wall thickness, stenosis and plaque composition, that is presence of intraplaque hemorrhage, lipid, and calcification. History of ischemic stroke and CHD was assessed until date of magnetic resonance imaging. The study was approved by the institutional review board, and all participants gave informed consent. Logistic regression analyses adjusted for age and traditional cardiovascular risk factors were used to study sex-specific associations between plaque characteristics and clinical events.

Results—We found that both carotid stenosis and intraplaque hemorrhage were associated with ischemic stroke in men but not in women (men: odds ratio [OR] for stenosis [per 10% increase]: 1.17 [95% CI, 1.06–1.30] and for intraplaque hemorrhage 2.39 [95% CI, 1.32–4.35]). In both men and women, carotid stenosis was associated with CHD (men: OR per 10% increase 1.12 [95% CI, 1.04–1.21] and women: OR, 1.17 [95% CI, 1.03–1.34]) and carotid wall thickness was associated with CHD (men: OR, 1.20 [95% CI, 1.03–1.39] and women: OR, 1.21 [95% CI, 0.88–1.65]). None of the plaque components was associated with CHD.

Conclusions—Whereas carotid plaque thickness and stenosis are associated with the history of ischemic stroke and CHD, carotid intraplaque hemorrhage is associated with ischemic stroke, but not with CHD, providing novel insights into the pathogenesis of cardiovascular events. (Stroke. 2016;47:1542-1547. DOI: 10.1161/STROKEAHA.116.012923.)

Key Words: atherosclerosis □ cardiovascular disease □ carotid stenosis □ magnetic resonance imaging □ stroke

Atherosclerosis is the primary cause of cardiovascular disease, with coronary heart disease (CHD) and ischemic stroke as its most important clinical manifestations. Atherosclerosis is clinically silent for years with gradual thickening of the vessel wall and changes in plaque composition. Previous data suggest that plaque vulnerability depends on its composition, rather than on its thickness or the severity of stenosis.1 One of the vessel beds most extensively studied in atherosclerosis is the carotid artery because it can be easily visualized using different imaging techniques. Whereas ultrasound was for long the main modality for assessing carotid wall thickness, technical advances in other imaging modalities, especially magnetic resonance imaging (MRI), now allow a better characterization of plaque composition. Plaque components that can reliably be characterized on MRI are lipid deposits with or without a necrotic core, calcification, and intraplaque hemorrhage (IPH). Among these, IPH has gained much attention because it has been recognized as an important determinant of plaque instability and subsequent risk of stroke.2,3

An important feature of atherosclerosis is that it is a systemic vascular disease and that the presence of atherosclerotic disease at one specific location may predict ischemic events in distant locations.4,5 In this context, intima media thickness (IMT) in the carotid arteries has previously been used to assess the risk of CHD.6-11 Also, specific properties of carotid...
plagues, such as irregularity of the plaque surface, were found to be associated with atherosclerotic plaque irregularity in other vessel beds. Subsequently, it was postulated that systemic risk factors might lead to a systemic predisposition to irregularity and rupture of atherosclerotic plaques. Such a predisposition to plaque instability attributable to systemic risk factors would suggest that plaque composition and instability in the carotid arteries could be related not only to stroke but also to other events, such as CHD. Therefore, in this study, we investigated the association of extent of carotid atherosclerosis and carotid plaque composition with a history of ischemic stroke and CHD in a population-based cohort study.

Methods

Study Population

The subjects of this study are participants of the Rotterdam Study, a prospective population-based cohort study initiated in 1990 among persons 55 years and older in the municipality of Rotterdam, the Netherlands. The original cohort of the Rotterdam Study was expanded in 2000, and again in 2006 to include participants who were 45 years and older. All study participants routinely undergo carotid ultrasonography to assess carotid IMT (measured as maximum distance between the near and far wall). Of 10073 participants with carotid ultrasound, 3795 participants (38%) had wall thickness ≥2.5 mm in at least one carotid, which was the inclusion criterion for participating in the current carotid MRI study. From the 3795 with wall thickening we invited 2666 participants to undergo an MRI of the carotid arteries. The remaining 1129 participants had passed away, moved out of the study area, were physically disabled (n=701), or had known MRI contraindications (n=428). From the 2666 persons, 684 did not undergo MRI scanning, because of claustrophobia (n=57), physical restrictions (n=191), contraindications (n=115), refusal to participate (n=272), no show or lost to follow-up (n=49). The remaining 1982 participants (74% of those initially invited) underwent MRI scanning of both carotid arteries. Scans were excluded if image quality was bad (n=95), if no plaque ≥2 mm were observed bilaterally (n=41) or if scanning was interrupted due to claustrophobia (n=106). Of the remaining 1739 participants with carotid MRI scans, we excluded participants with incomplete information on prevalence of CHD (n=8) or stroke (n=48). The Rotterdam Study has been approved by the medical ethics committee according to the Population Study Act Rotterdam Study, executed by the Ministry of Health, Welfare and Sports of the Netherlands. A written informed consent was obtained from all participants.

Carotid MRI Scanning and Analysis of Plaque Characteristics

Imaging was performed with a 1.5-Tesla scanner (GE Healthcare, Milwaukee, WI) with a bilateral phased-array surface coil (Machnet, Eelde, The Netherlands). A standard scanning protocol was used with a total scanning time of ~30 minutes. The carotid MRI protocol, reading, and reproducibility are described elsewhere. We evaluated both carotid arteries over a range of 15 mm caudally to 30 mm cranially of the bifurcation. We assessed plaque characteristics in all plaques with a maximum thickness of 2.0 mm on MRI. On the proton density weighted fast spin echo images, maximum carotid wall thickness was measured, and degree of luminal stenosis was calculated using the North American Symptomatic Carotid Endarterectomy Trial criteria. Plaques were reviewed for the presence of 3 different plaque components: IPH, lipid core, and calcification. In short, we defined IPH as the presence of a hyperintense focus within the plaque on the 3-dimensional T1-weighted gradient echo sequence. Calcification was defined as a hypointense region in the plaque on all sequences. Finally, the presence of lipid core was defined as a hypointense region in the plaque on density weighted fast spin echo or proton density weighted echo planar image and T2-weighted echo planar image, or a region of relative signal intensity drop in the T2-weighted echo planar images compared with the proton density weighted echo planar image. Multiple components were permissible in one plaque.

History of Stroke or CHD

A history of ischemic stroke or CHD was based on either a history of event self-reported at study entry, verified by clinical data from the medical records, or the occurrence of an event during study follow-up but before the time of carotid MRI scanning. The follow-up system involved linkage of the study to files from general practitioners in the study area and subsequent collection of information from letters of medical specialists, brain imaging, and discharge reports in case of hospitalization. For the diagnosis of stroke and cardiac events, 2 research physicians independently coded all reported events according to the International Classification of Diseases, 10th Revision. In case of disagreement, a decision was made by a neurologist or cardiologist. Stroke was defined as a history of ischemic or unspecified stroke but not hemorrhagic stroke (International Classification of Diseases, 10th Revision codes I61, I63, and I64). To define CHD, we used a combined outcome of nonfatal myocardial infarction and myocardial revascularization (ie, percutaneous coronary intervention and coronary artery bypass grafting, International Classification of Diseases, 10th Revision codes I21, I24, and I25). Information on incidence of stroke and CHD was completed until January 1, 2012.

Assessment of Covariates

Information on cardiovascular risk factors was obtained from the visit to the research center, which took place before the MRI scanning. Smoking status was classified as ever or never. Serum total cholesterol and high-density lipoprotein-cholesterol values were measured using standard laboratory techniques. On the basis of weight and height, the body mass index was calculated. Diabetes mellitus was defined as a fasting blood glucose >7.0 mmol/L, non-fasting glucose >11.0 mmol/L, or use of antidiabetic medication. Blood pressure was measured using a random-zero sphygmomanometer at the study center visit. Hypertension was defined as a blood pressure >140/90 mmHg or the use of antihypertensive medication. Medication dispensing data were obtained from the fully computerized pharmacies in the Ommoord suburb. Information on all filled prescriptions of antihypertensive drugs on date of carotid MRI scans was available.

Statistical Analysis

We compared the baseline characteristics between men and women using age-adjusted linear regression for continuous variables and logistic regression for dichotomous variables. We also examined the mean wall thickness and degree of stenosis in persons with and without IPH, lipid core, and calcification (Table I in the online-only Data Supplement). To investigate the relation of the different plaque characteristics (IPH, lipid, calcification, stenosis [per 10%], and carotid wall thickness) with the history of ischemic stroke and CHD, we used binomial logistic regression. Next, we analyzed these relations in the whole sample and for men and women separately. In the first model, analyses were adjusted for age, sex (in the overall model only), and carotid wall thickness (only for the analyses with IPH, lipid, and calcification as determinant). In the second models, we additionally adjusted the associations for cardiovascular risk factors (smoking, total cholesterol, high-density lipoprotein-cholesterol, body mass index, diabetes mellitus, and hypertension).

We also evaluated effect modification by sex. Finally, we performed additional analyses in which all plaque characteristics were further adjusted for the other plaque components (Tables I and II in the online-only Data Supplement).

All analyses were carried out using SPSS Statistical Package version 20.0 (Chicago, IL). Missing values in the covariates were imputed using the Expectation Maximization method.
Results

Table 1 reports the characteristics for the 1731 participants. The population consisted of 936 men (55%; mean age, 72.3±9.0 years) and 795 women (46%; mean age, 73.6±9.3 years). Men had lower total cholesterol values, lower high-density lipoprotein-cholesterol, higher prevalence of smoking, diabetes mellitus, and diastolic blood pressure. The mean carotid wall thickness was 3.6 mm (±1.1) and mean degree of stenosis was 13.1% (±18). IPH was prevalent in 35%, lipid in 41%, and calcification in 82%. Information on carotid plaque characteristics and history of CHD was available for 1731 participants and for history of stroke in 1683 participants. Before the carotid MRI scanning, a total of 105 individuals (61 men [6.5%] versus 44 women [5.5%]) experienced an ischemic stroke and 199 individuals (164 men [15.6%] versus 53 women [6.7%]) experienced a CHD event.

Table 2 presents overall and sex-specific associations between plaques and history of ischemic stroke, adjusted for age, carotid wall thickness (model 1), and for cardiovascular risk factors (model 2). In the multivariable model (model 2), only carotid stenosis was significantly associated with stroke (odds ratio [OR] per 10% increase in stenosis 1.14 [95% CI, 1.05–1.24]). However, after we stratified for sex, we found a strong association for IPH and carotid stenosis with stroke in men but not in women (OR for IPH: men: 2.39, [95% CI, 1.32–4.35], women: 0.69 [95% CI, 0.33–1.46]. OR for stenosis per 10% increase 1.17, [95% CI, 1.06–1.30], women: 1.10 [95% CI, 0.95–1.28]). When we tested for effect modification by sex, we only found the interaction term with IPH to be significant (P=0.03). Lipid, calcification, and carotid wall thickness were not associated with stroke in the fully adjusted model.

Table 3 shows overall and sex-specific associations between plaque characteristics and history of CHD, adjusted for age, carotid wall thickness (model 1), and for cardiovascular risk factors (model 2). In the overall analysis, only carotid stenosis was significantly associated with CHD after adjustment for cardiovascular risk factors (OR per 10% increase 1.14 [95% CI, 1.06–1.22]). Stratifying for sex did not materially change this association in both men and women. Also, carotid wall thickness was significantly associated with CHD in men (OR, 1.20 [95% CI, 1.03–1.39]) but not in women (OR, 1.21 [95% CI, 0.88–1.65]). For carotid stenosis and wall thickness, no effect modification by sex was observed (data not shown).

None of the various plaque components was associated with the history of CHD, also after stratification for sex. All analyses for the association between plaque characteristics and stroke and CHD were repeated with additional adjustment for the remaining plaque components, but this did not alter the results (Tables II and III in the online-only Data Supplement).

Discussion

In this population-based study, we found carotid atherosclerotic plaque characteristics to be differentially related to history of ischemic stroke and CHD. Whereas the extent of atherosclerosis, expressed as plaque thickness or stenosis, was associated both with the history of ischemic stroke and CHD, plaque composition, and specifically IPH, seemed to be associated with the history of stroke only. These associations were primarily present in men and less prominent in women.

The relation between carotid IMT as measured with B-mode ultrasound and cardiovascular disease has been well established and IMT serves as a marker of generalized atherosclerosis. To this extent, IMT has been increasingly used for risk stratification models and contributes greatly to the prediction of CHD. To our knowledge, no studies have mainly focused on the relation between carotid plaque composition as detected with MRI and CHD. Most imaging studies used ultrasound measurements, because these measurements are relatively simple and noninvasive, but the resolution is of limited value for characterization of plaque composition. By using MRI, we found that carotid wall thickness and stenosis are more associated with a history of CHD than any of the specific carotid plaque components.

We found a prominent association of stenosis and IPH with history of stroke, which is in line with several clinical studies that investigated carotid plaque composition and neurologic ischemic events. In a meta-analysis among 394 asymptomatic subjects, it was found that especially IPH was related to a substantially increased risk of stroke (hazard ratio, 3.5). However, because atherosclerosis is a chronic inflammatory condition with various local and systemic manifestations, researchers have raised the hypothesis that plaque instability may also be a systemic condition, influenced by systemic risk factors. For this reason, the term vulnerable patient was introduced, indicating that changes found in 1 vessel bed may be predictive of risk of events in another vessel bed. Whereas several reports previously suggested...
a relation between carotid IPH and CHD,\cite{50,51,52} we did not find any significant association between any of the carotid plaque components and history of CHD. Although we used previous clinical history with no clear information on how long exactly the CHD event had occurred before the carotid artery was imaged, carotid plaque composition seemed to be less important for CHD than for ischemic stroke. A possible explanation for the discrepancy with previous literature is that some of these studies used carotid tissue specimens derived after carotid endarterectomy or autopsy, in which it is difficult to distinguish between IPH and intraluminal thrombi after the tissue preparation. Other explanations may be related with the fact that others used a composite end point of cerebral and cardiovascular disease, or did not stratify for sex. This may have resulted in a dilution of the association between IPH with stroke and an overestimation of the association with CHD. Furthermore, a precise mechanism describing how atherosclerosis in one vessel bed can lead to clinical events other than those in their corresponding territory remains unclear. Some studies suggested that the presence of IPH observed in one vessel bed might reflect a systemic susceptibility for vulnerable plaques in other vessel beds.\cite{33,34} Because atherosclerotic plaques with IPH are generally thicker than plaques without IPH,\cite{16} individuals with IPH could possibly have complications of the extent of atherosclerosis rather than the vulnerability of the plaque.

With respect to clinical events, we found that carotid atherosclerotic plaque characteristics differed between men and women. These differences could, in part, be explained by the smaller number of CHD events in women, leading to a lack in statistical power, as the estimates for the association between plaque thickness and stenosis and CHD were similar in women as in men. Alternatively, the larger OR of the association between IPH and history of stroke that we observed in men as opposed to women is in line with the male predominance of IPH as observed in several other studies.\cite{16,32,33,35,36} In a recent study of symptomatic patients who underwent carotid endarterectomy, IPH seemed to be associated with an increased risk of stroke and cardiovascular events in men, but not in women.\cite{32} A possible explanation may relate to more efficient repair, as stable, more fibrous less inflammatory, plaques were found in women, especially asymptomatic women, compared with men in histology studies.\cite{36} It was also suggested that although women may have similar amount of atherosclerosis, they may differ in clinical presentation of CHD or stroke because of estrogen- or genetic-related reasons.\cite{37}

Several strengths and limitations of our study need to be addressed. The advantages of this study are the population-based design and the large sample size. Furthermore, we focused on history of stroke and CHD separately instead of restricting the study to 1 end organ or using composite end points.}

### Table 2. Atherosclerotic Plaque Characteristics and History of Ischemic Stroke

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
</tr>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P Value</td>
<td>OR (95% CI)</td>
</tr>
<tr>
<td>IPH</td>
<td>1.61 (1.04–2.50)</td>
<td>0.03</td>
<td>1.47 (0.94–2.39)</td>
</tr>
<tr>
<td>Lipid</td>
<td>0.85 (0.57–1.29)</td>
<td>0.4</td>
<td>0.91 (0.60–1.39)</td>
</tr>
<tr>
<td>Calcification</td>
<td>1.45 (0.77–2.72)</td>
<td>0.2</td>
<td>1.32 (0.70–2.50)</td>
</tr>
<tr>
<td>Stenosis*</td>
<td>1.17 (1.08–1.26)</td>
<td>&lt;0.001</td>
<td>1.14 (1.05–1.24)</td>
</tr>
<tr>
<td>Wall thickness</td>
<td>1.22 (1.04–1.43)</td>
<td>0.02</td>
<td>1.17 (0.99–1.38)</td>
</tr>
</tbody>
</table>

Model 1: adjusted for sex (only in overall model), age, and carotid wall thickness (except for carotid wall thickness and stenosis that were only adjusted for sex and age). Model 2: as model 1, and additionally for smoking, total cholesterol, high-density lipoprotein-cholesterol, body mass index, diabetes mellitus, and hypertension. CI indicates confidence interval; IPH, intraplaque hemorrhage; and OR, odds ratio.

*Per 10% increase.

### Table 3. Atherosclerotic Plaque Characteristics and History of Coronary Heart Disease

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
</tr>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P Value</td>
<td>OR (95% CI)</td>
</tr>
<tr>
<td>IPH</td>
<td>1.06 (0.75–1.48)</td>
<td>0.8</td>
<td>1.02 (0.72–1.45)</td>
</tr>
<tr>
<td>Lipid</td>
<td>0.85 (0.62–1.17)</td>
<td>0.3</td>
<td>0.92 (0.67–1.28)</td>
</tr>
<tr>
<td>Calcification</td>
<td>1.22 (0.75–1.97)</td>
<td>0.4</td>
<td>1.15 (0.70–1.89)</td>
</tr>
<tr>
<td>Stenosis*</td>
<td>1.15 (1.08–1.23)</td>
<td>&lt;0.001</td>
<td>1.14 (1.06–1.22)</td>
</tr>
<tr>
<td>Wall thickness</td>
<td>1.25 (1.10–1.42)</td>
<td>&lt;0.001</td>
<td>1.20 (1.05–1.37)</td>
</tr>
</tbody>
</table>

Model 1: adjusted for sex (only in overall model), age, and carotid wall thickness (except for carotid wall thickness and stenosis that were only adjusted for sex and age). Model 2: as model 1, and additionally for smoking, total cholesterol, high-density lipoprotein-cholesterol, body mass index, diabetes mellitus, and hypertension. CI indicates confidence interval; IPH, intraplaque hemorrhage; and OR, odds ratio.

*Per 10% increase.
points. We used prevalent cardiovascular events in which symptoms of ischemia were clinically confirmed or treated. Although linkage with data from general practitioners ensured validation of the majority of the diagnoses, events that occurred before entering the Rotterdam Study were based on self-report and may, therefore, be less reliable. An important issue to consider with respect to our findings is that we specifically investigated the relation of plaque characteristics with prevalent stroke and CHD. As a consequence, we cannot infer any temporality between the determinants and the outcomes. Moreover, fatal events, in which advanced atherosclerosis is expected, were not considered in this study. This may have led to an underestimation of the association between vulnerable plaque characteristics and clinical events. In addition, lifestyle changes or medication use initiated after primary stroke or CHD may have affected atherosclerotic plaque development. This could have stabilized the atherosclerotic plaque and reduced the differences in plaque characteristics between individuals with and without a history of clinical event. However, we cannot exclude that the plaque has evolved after experiencing stroke or CHD. These considerations highlight the need for prospective studies that investigate the relation between carotid plaque characteristics and incident stroke and CHD, and determine the incremental value of carotid MRI parameters beyond the traditional cardiovascular risk factor included in, for example, the Framingham risk score.

In summary, we found that carotid plaque thickness and stenosis are associated with a history of ischemic stroke and CHD, whereas carotid IPH is only associated with a history of ischemic stroke, and not with CHD. Future studies should focus on investigating whether there is a role for carotid MRI in risk prediction and risk stratification of ischemic stroke and CHD.

Acknowledgments
The dedication, commitment, and contribution of inhabitants, general practitioners, and pharmacists of the Ommoord district to the Rotterdam Study are gratefully acknowledged.

Sources of Funding
The Rotterdam Study is supported by the Erasmus MC and Erasmus University Rotterdam; the Netherlands Organisation for Scientific Research (NWO); the Netherlands Organisation for Health Research and Development (ZonMW); the Research Institute for Diseases in the Elderly; the Netherlands Genomics Initiative; the Ministry of Education, Culture and Science; the Ministry of Health, Welfare and Sports; the European Commission (DG XII); and the Municipality of Rotterdam. This study was supported by the Netherlands Heart Foundation, (2009B044 and 2012T008) and the Netherlands Organisation for Scientific Research (NWO/ZonMWVici 918-76-619).

Disclosures
None.

References
22. Lorenz MW, Markus HS, Bots ML, Roswall M, Sitzer M. Prediction of clinical cardiovascular events with carotid intima-media thickness: a


Carotid Atherosclerotic Plaque Characteristics on Magnetic Resonance Imaging Relate With History of Stroke and Coronary Heart Disease

Mariana Selwaness, Daniel Bos, Quirijn van den Bouwhuijsen, Marileen L.P. Portegies, M. Arfan Ikram, Albert Hofman, Oscar H. Franco, Aad van der Lugt, Jolanda J. Wentzel and Meike W. Vernooij

_Stroke_. 2016;47:1542-1547; originally published online May 10, 2016;
doi: 10.1161/STROKEAHA.116.012923

_Stroke_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2016 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://stroke.ahajournals.org/content/47/6/1542

An erratum has been published regarding this article. Please see the attached page for:
/content/47/7/e201.full.pdf

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2016/05/10/STROKEAHA.116.012923.DC1
http://stroke.ahajournals.org/content/suppl/2017/07/10/STROKEAHA.116.012923.DC2

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Stroke_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Stroke_ is online at:
http://stroke.ahajournals.org//subscriptions/
In the article by Selwaness et al (Selwaness M, Bos D, van den Bouwhuijsen Q, Portegies MLP, Ikram MA, Hofman A, Franco OH, van der Lugt A, Wentzel JJ, Vernooij MW. Carotid atherosclerotic plaque characteristics on magnetic resonance imaging relate with history of stroke and coronary heart disease. Stroke. 2016;47:1542–1547. DOI: 10.1161/STROKEAHA.116.012923.), which published online on May 10, 2016, and appeared in the June 2016 issue of the journal, a correction was needed.

On page 1545, Table 2, column Women Model 1 P value, row Wall thickness, “0.04,” has been changed to read “0.4.”

This correction has been made to the online version of the article, which is available at http://stroke.ahajournals.org/content/47/6/1542.
Supplemental table I. Mean wall thickness and degree of stenosis in persons with and without IPH, lipid core and calcification.

<table>
<thead>
<tr>
<th></th>
<th>Wall thickness</th>
<th>Degree of stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>IPH present</td>
<td>4.1 (1.2)</td>
<td>28.0 (24.7)</td>
</tr>
<tr>
<td>IPH absent</td>
<td>3.2 (0.8)</td>
<td>13.6 (15.0)</td>
</tr>
<tr>
<td>Lipid core present</td>
<td>3.8 (1.1)</td>
<td>22.8 (21.9)</td>
</tr>
<tr>
<td>Lipid core absent</td>
<td>3.4 (0.9)</td>
<td>15.2 (18.0)</td>
</tr>
<tr>
<td>Calcification present</td>
<td>3.6 (1.0)</td>
<td>19.8 (20.7)</td>
</tr>
<tr>
<td>Calcification absent</td>
<td>3.2 (0.9)</td>
<td>12.7 (16.2)</td>
</tr>
</tbody>
</table>

SD: standard deviation, IPH: intraplaque hemorrhage
### Supplemental table II. Atherosclerotic plaque characteristics and history of ischemic stroke, adjusted for other plaque components

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Men</th>
<th>Women</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
</tr>
<tr>
<td></td>
<td>OR (95%CI)</td>
<td>P</td>
<td>OR (95%CI)</td>
<td>P</td>
<td>OR (95%CI)</td>
</tr>
<tr>
<td><strong>IPH</strong></td>
<td>1.61 (1.03;2.51)</td>
<td>0.04</td>
<td>1.45 (0.92;2.27)</td>
<td>0.1</td>
<td>2.60 (1.43;4.73)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.45 (1.33;4.9)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.78 (0.37;1.64)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.66 (0.31;1.40)</td>
</tr>
<tr>
<td><strong>Lipid</strong></td>
<td>0.83 (0.55;1.25)</td>
<td>0.4</td>
<td>0.87 (0.57;1.33)</td>
<td>0.5</td>
<td>0.74 (0.43;1.27)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.76 (0.44;1.34)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.96 (0.50;1.84)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.10 (0.56;2.17)</td>
</tr>
<tr>
<td><strong>Calcification</strong></td>
<td>1.22 (0.71;2.52)</td>
<td>0.4</td>
<td>1.23 (0.64;2.34)</td>
<td>0.5</td>
<td>1.20 (0.52;2.70)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.09 (0.46;2.57)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.59 (0.60;4.24)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.49 (0.55;4.05)</td>
</tr>
<tr>
<td><strong>Stenosis</strong></td>
<td>1.14 (1.05;1.24)</td>
<td>0.003</td>
<td>1.12 (1.03;1.22)</td>
<td>0.01</td>
<td>1.13 (1.02;1.25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.13 (1.02;1.25)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.15 (1.00;1.35)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.13 (0.96;1.34)</td>
</tr>
<tr>
<td><strong>Wall thickness</strong></td>
<td>1.25 (0.97;1.38)</td>
<td>0.1</td>
<td>1.10 (0.92;1.31)</td>
<td>0.3</td>
<td>1.11 (0.90;1.36)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.07 (0.87;1.33)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.20 (0.83;1.74)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.18 (0.81;1.72)</td>
</tr>
</tbody>
</table>

IPH: intraplaque hemorrhage, OR: odds ratio, CI: confidence interval

Model 1: adjusted for sex (only in overall model), age and carotid wall thickness (except for carotid wall thickness and stenosis which were only adjusted for sex and age).

Model 2: as model 1, and additionally for smoking, total cholesterol, HDL-cholesterol, BMI, diabetes mellitus and hypertension.

* per 10% increase
### Supplemental table III. Atherosclerotic plaque characteristics and history of CHD, adjusted for other plaque components

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
<td>Model 2</td>
<td></td>
</tr>
<tr>
<td><strong>OR</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>OR</strong></td>
<td></td>
<td></td>
<td><strong>OR</strong></td>
<td></td>
<td><strong>OR</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>(95% CI)</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>(95% CI)</strong></td>
<td></td>
<td></td>
<td><strong>(95% CI)</strong></td>
<td></td>
<td><strong>(95% CI)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>IPH</strong></td>
<td>1.13</td>
<td>0.5</td>
<td>1.04</td>
<td>0.8</td>
<td>0.87</td>
<td>0.5</td>
<td>0.82</td>
<td>0.4</td>
<td>1.66</td>
<td>0.1</td>
<td>1.70</td>
</tr>
<tr>
<td></td>
<td>(0.80;1.59)</td>
<td>(0.73;1.48)</td>
<td></td>
<td>(0.57;1.31)</td>
<td>(0.54;1.26)</td>
<td>(0.88;3.11)</td>
<td></td>
<td>(0.89;3.25)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lipid</strong></td>
<td>0.95</td>
<td>0.8</td>
<td>0.99</td>
<td>0.9</td>
<td>0.92</td>
<td>0.7</td>
<td>0.99</td>
<td>0.9</td>
<td>0.70</td>
<td>0.3</td>
<td>0.73</td>
</tr>
<tr>
<td></td>
<td>(0.70;1.30)</td>
<td>(0.71;1.37)</td>
<td></td>
<td>(0.64;1.34)</td>
<td>(0.67;1.47)</td>
<td>(0.37;1.30)</td>
<td></td>
<td>(0.38;1.40)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Calcification</strong></td>
<td>1.19</td>
<td>0.5</td>
<td>1.15</td>
<td>0.6</td>
<td>0.99</td>
<td>0.9</td>
<td>0.93</td>
<td>0.8</td>
<td>2.42</td>
<td>0.2</td>
<td>2.33</td>
</tr>
<tr>
<td></td>
<td>(0.73;1.93)</td>
<td>(0.70;1.89)</td>
<td></td>
<td>(0.58;1.71)</td>
<td>(0.53;1.64)</td>
<td>(0.72;8.14)</td>
<td></td>
<td>(0.69;7.92)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Stenosis</strong></td>
<td>1.16</td>
<td>&lt;0.001</td>
<td>1.14</td>
<td>&lt;0.001</td>
<td>1.16</td>
<td>&lt;0.001</td>
<td>1.14</td>
<td>0.002</td>
<td>1.16</td>
<td>0.04</td>
<td>1.15</td>
</tr>
<tr>
<td></td>
<td>(1.08;1.25)</td>
<td>(1.06;1.23)</td>
<td></td>
<td>(1.07;1.26)</td>
<td>(1.05;1.25)</td>
<td>(1.01;1.34)</td>
<td></td>
<td>(1.00;1.34)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Wall thickness</strong></td>
<td>1.31</td>
<td>&lt;0.001</td>
<td>1.23</td>
<td>0.004</td>
<td>1.29</td>
<td>0.001</td>
<td>1.24</td>
<td>0.01</td>
<td>1.12</td>
<td>0.5</td>
<td>1.11</td>
</tr>
<tr>
<td></td>
<td>(1.14;1.50)</td>
<td>(1.07;1.42)</td>
<td></td>
<td>(1.11;1.51)</td>
<td>(1.05;1.45)</td>
<td>(0.79;1.57)</td>
<td></td>
<td>(0.87;1.56)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**IPH:** intraplaque hemorrhage, **OR:** odds ratio, **CI:** confidence interval

**Model 1:** adjusted for sex (only in overall model), age and carotid wall thickness (except for carotid wall thickness and stenosis which were only adjusted for sex and age).

**Model 2:** as model 1, and additionally for smoking, total cholesterol, HDL-cholesterol, BMI, diabetes mellitus and hypertension.

* * per 10% increase
Neck Atherosclerotic Plaque Characteristics on Magnetic Resonance Imaging Relate With History of Stroke and Coronary Heart Disease

Mariana Selwaness, MD, PhD; Daniel Bos, MD, PhD; Quirijn van den Bouwhuijsen, MD; Marileen L.P. Portegies, MD; M. Arfan Ikram, MD, PhD; Albert Hofman, MD, PhD; Oscar H. Franco, MD, PhD; Aad van der Lugt, MD, PhD; Jolanda J. Wentzel, PhD; Meike W. Vernooij, MD, PhD

Correspondence to Meike W. Vernooij, MD, PhD, Department of Radiology, Erasmus MC, PO Box 204, 3000 CA Rotterdam, The Netherlands. E-mail m.vernooij@erasmusmc.nl

©2016 American Heart Association, Inc.

Arteriosclerotic plaque characteristics on magnetic resonance imaging relate with history of stroke and coronary heart disease.

Neck atherosclerotic plaque characteristics on magnetic resonance imaging relate with history of stroke and coronary heart disease.

Carotid Atherosclerotic Plaque Characteristics on Magnetic Resonance Imaging Relate With History of Stroke and Coronary Heart Disease

Background and Methods

Arteriosclerosis is a major cause of heart disease, stroke, and death. The presence of atherosclerotic plaques on the carotid arteries is a major risk factor for stroke and coronary heart disease.

Results

The results of this study suggest that the presence of atherosclerotic plaques on the carotid arteries is associated with an increased risk of stroke and coronary heart disease.

Conclusion

The results of this study provide new insights into the relationship between atherosclerotic plaques and cardiovascular disease.
的双侧颈动脉斑块均小于2 mm (n=41)以及因为幽闭恐惧症而中断(n=106)等情况而被除外研究。在剩余的1739例接受MRI平扫的受试者中,还排除了既往关于CHD病史(n=8)或卒中史(n=48)病史信息不完整的受试者。Rotterdam研究经药物伦理委员会认可并经过了荷兰卫生福利体育部颁布的Rotterdam人口研究计划,所有受试者全部签署了书面知情同意书。

颈动脉MRI平扫与斑块特点分析

MRI设备:使用1.5T场强的磁共振扫描仪(GE Healthcare, Milwaukee, WI)获取图像，这种MRI设备有双侧相控阵表面线圈(Machnet, Eelde, The Netherlands),标准平扫要求总共的扫描时间要在30 min左右。颈动脉MRI平扫操作方法、阅片和可重复性已在其他论文加以阐述。本研究评估了颈动脉平扫的双侧颈动脉,利用MRI分析了所有斑块中最大厚度≥2.0mm的斑块特点。在质子密度加权快速自旋回波图像中,根据北美症状性颈动脉内膜切除手术试验标准17,测量颈动脉壁的最大厚度,计算出动脉管腔的狭窄率。斑块主要分为3种不同成分:IPH、脂质核心、钙化。IPH表现为T1加权序列出现高信号。钙化表现为在所有序列斑块内出现低信号。脂质核心表现为T1加权序列出现高信号或低信号。钙化表现为在所有序列斑块内出现低信号。脂质核心表现为T1加权序列出现高信号或低信号。钙化表现为在所有序列斑块内出现低信号。

既往卒中史或CHD病史

既往缺血性卒中史或CHD病史是根据患者入组前自述、经过病历档案核实,或是基于MRI扫描之前的随访期间出现的临床事件。随访系统的临床材料包括了受试者提供的病例材料、医学专家的信件、脑部影像学检查和住院治疗的出院小结。对于卒中和心脏事件的诊断,2名参与研究的内科医生独立根据国际疾病分类(ICD10)对所有被报告的临床事件进行了编码18。对于有争议的病例,最终的诊断决定由神经科专科医生或心血管专科医生作出。卒中定义为缺血性卒中或非出血性卒中(ICH, I21, I24, I25)。卒中和CHD发生率的统计于2012年1月1日前完成。

协变量分析

心血管危险因素在MRI扫描之前的研究中心访视时进行采集。根据吸烟状况分为有或无;血清总胆固醇和高密度脂蛋白胆固醇的数值通过标准的实验室技术进行检测。根据体重和身高,计算得出体重指数;糖尿病定义为空腹血糖>7.0 mmol/L;非空腹血糖>11.0 mmol/L;或使用抗糖尿病药物治疗。药物使用数据从Ommoord社区药店计算机系统获取。所有患者均按抗高血压处方药物信息在完成颈动脉MRI当日获取。

统计分析

采用经过年龄校正的线性回归分析处理连续变量和Logistic回归分析处理二元变量比较男性和女性2组间的基线特征。对有/无IPH、脂质核心及钙化的患者平均动脉壁厚度和动脉狭窄程度进行分析(表1,在线数据补充)。为了研究不同斑块成分特点(IPH、脂质、钙化、狭窄(每10%))、颈动脉壁的厚度)与既往缺血性卒中史及CHD病史的关系,本研究采用了二元Logistic回归分析分别对整个样本、男性组和女性组进行统计学分析。在模型1中对整个样本中校正了年龄、性别和颈动脉壁厚度的影响。在模型2中研究者又额外校正了心血管病相关危险因素(吸烟、总胆固醇、高密度脂蛋白胆固醇、体重指数、糖尿病和高血压)的相关影响。本研究还评估了性别对结果的影响。本研究校正了其他斑块成分对全部斑块特征影响并进一步进行相关性分析(见在线数据补充表1和表2)。

本研究使用统计软件SPSS 20.0 (Chicago,IL)进行所有的统计学分析。在协变量中,丢失的数据通过期望最大化方法进行估算。

结果

表1显示了1731例受试者的基线特点。表2数据显示整体和性别相关的斑块特点与缺血性卒中病史之间相关的相关性。表2数据显示整体和性别相关的斑块特点与缺血性卒中病史之间的相关性,模型1校正了年龄,颈动脉壁厚度,模型2校正了心血管危险因素。在多变量的模型2中,只有颈动脉狭窄与卒中显著相关[OR值在狭窄率每增加10%为1.14(95%CI,1.05~1.24)]。在对性别进行分层分析后,发现IPH和颈动脉狭窄与卒中存在相关关系,而在女性中没有如此的相关性(IPH和颈动脉狭窄与卒中存在显著相关关系,而在女性中没有如此的相关性)。在多变量的模型2中,颈动脉狭窄与卒中存在显著相关关系(OR值在狭窄率每增加10%为1.14(95%CI,1.05~1.24))。
表 2 动脉粥样硬化斑块特点和缺血性卒中病史的关系

<table>
<thead>
<tr>
<th></th>
<th>全部</th>
<th>男性</th>
<th>女性</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>模型 1</td>
<td>模型 2</td>
<td>模型 1</td>
</tr>
<tr>
<td></td>
<td>OR (95%CI)</td>
<td>P值</td>
<td>OR (95%CI)</td>
</tr>
<tr>
<td>内出血</td>
<td>1.61(1.04~2.50)</td>
<td>0.03</td>
<td>1.47(0.94~2.30)</td>
</tr>
<tr>
<td>斑块内出血</td>
<td>0.85(0.57~1.39)</td>
<td>0.4</td>
<td>0.90(0.60~1.39)</td>
</tr>
<tr>
<td>流化</td>
<td>1.40(0.77~2.72)</td>
<td>0.2</td>
<td>1.30(0.70~2.50)</td>
</tr>
<tr>
<td>突破 *</td>
<td>1.17(1.06~1.28)</td>
<td>&lt;0.001</td>
<td>1.14(1.05~1.24)</td>
</tr>
<tr>
<td>血管壁厚度</td>
<td>1.22(1.04~1.43)</td>
<td>0.02</td>
<td>1.17(0.95~1.38)</td>
</tr>
</tbody>
</table>

注: 模型 1: 校正了性别(只在全部人群中), 年龄，颈动脉壁厚度（除了只根据性别和年龄校正的颈动脉壁厚度和狭窄）, 模型 2: 在模型 1 基础上, 校正了吸烟、总胆固醇、高密度脂蛋白胆固醇、体重指数、糖尿病和高血压。CI: 可信区间; IPH: 斑块内出血; OR: 比值比。
* 每增长 10%。

表 3 动脉粥样硬化斑块特点和 CHD 病史

<table>
<thead>
<tr>
<th></th>
<th>全部</th>
<th>男性</th>
<th>女性</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>模型 1</td>
<td>模型 2</td>
<td>模型 1</td>
</tr>
<tr>
<td></td>
<td>OR (95%CI)</td>
<td>P值</td>
<td>OR (95%CI)</td>
</tr>
<tr>
<td>内出血</td>
<td>1.06(0.75~1.46)</td>
<td>0.8</td>
<td>1.02(0.72~1.45)</td>
</tr>
<tr>
<td>斑块内出血</td>
<td>0.85(0.62~1.17)</td>
<td>0.3</td>
<td>0.92(0.67~1.28)</td>
</tr>
<tr>
<td>流化</td>
<td>1.22(0.75~1.97)</td>
<td>0.4</td>
<td>1.19(0.70~1.98)</td>
</tr>
<tr>
<td>突破 *</td>
<td>1.15(1.06~1.23)</td>
<td>&lt;0.001</td>
<td>1.14(1.06~1.23)</td>
</tr>
<tr>
<td>血管壁厚度</td>
<td>1.25(1.04~1.49)</td>
<td>0.7</td>
<td>1.20(1.05~1.37)</td>
</tr>
</tbody>
</table>

注: 模型 1: 校正了性别(只在全部人群中), 年龄，颈动脉壁厚度（除了只根据性别和年龄校正的颈动脉壁厚度和狭窄）, 模型 2: 在模型 1 基础上, 校正了吸烟、总胆固醇、高密度脂蛋白胆固醇、体重指数、糖尿病和高血压。CI: 可信区间; IPH: 斑块内出血; OR: 比值比。
* 每增长 10%。

表 3 显示了整体和性别相关的斑块特点与既往 CHD 病史之间的相关性, 并校正了年龄、颈动脉壁厚度 (模型 1)、心血管危险因素 (模型 2)。在校正心血管危险因素后的总体分析中显示仅有颈动脉狭窄与 CHD 病史密切相关 [OR 值在颈动脉狭窄率每增加 10% 为 1.14(95% CI, 1.06~1.22)]。对性别分层分析并没有在本质上改变不同性别中斑块特点与 CHD 病史之间的相关性。同时, 颈动脉壁厚度与男性致死性冠状动脉事件 [OR 值, 1.20(95% CI, 0.88~1.65)]。对于颈动脉狭窄和血管壁厚度, 无性别相关性(数据未显示), 任何一种斑块成分与 CHD 病史无明显相关, 经过性别分层校正后亦无关联。即使在调整了其他斑块成分的影响进行重复分析后结果仍一致 (见在线数据补充表 2 和表 3)。

讨论

本研究发现, 颈动脉狭窄、IPH 与卒中病史显著相关, 与 CHD 病史无明显相关性, 而动脉粥样硬化的严重程度 (即斑块厚度和动脉狭窄) 与缺血性卒中史和 CHD 病史均有相关性。注: 本研究应用 MRI 检查发现, 颈动脉斑块成分与卒中病史显著相关, 与 CHD 病史无明显相关性。颈动脉超声测量的 IMT 与缺血性卒中史相关, 而与 CHD 病史无明显相关性, 而颈动脉斑块成分与卒中病史相关性, 与颈动脉斑块成分与 CHD 病史相关性。文献指出[23~26], 颈动脉狭窄、IPH 与卒中病史显著相关, 但颈动脉斑块成分与 CHD 病史无明显相关性。根据已有研究, 颈动脉粥样硬化斑块的成分与卒中病史显著相关, 而与 CHD 病史无明显相关性。在颈动脉药物治疗中, IMT 与血管内皮功能相关, 因此 IMT 增厚可能与颈动脉斑块成分相关。
相关性

本研究发现，颈动脉动脉粥样硬化斑块特征在临床事件发生方面存在性别差异，其原因可能是由于女性人群中CHD的人数较少，缺乏统计学效力，但斑块厚度和狭窄在男性和女性没有差别。另外，本研究中男性患者IPH与卒中病史相关的统计学数据出现较大OR值，与其他研究发现的“IPH主要发生于男性”的结论相一致。近期内一项针对颈动脉内膜剥脱术治疗有临床症状患者的研究中发现，男性IPH患者发生卒中与血管事件的风险增加，但在女性则无明显增加。

还有一项研究提出，由于存在已知的血栓或遗传因素等原因，在动脉粥样硬化程度相似情况下，其CHD和卒中的临床表现上可出现明显差异。

本研究的优点和局限性如下：

优点：
（1）本研究是基于人群的研究，样本量较大。
（2）本研究记录了既往卒中史和CHD病史，而非其他终点事件。
（3）本研究纳入的心血管事件均是经过确诊和治疗的。

局限性：
（1）尽管研究尽可能确保绝大多数患者诊断的准确性，但在人群中进行心脏疾病事件均是来自受试者口头，可能有遗漏。
（2）本研究的结果是对颈动脉斑块特征与卒中、CHD相关的特定性别研究，因此不能得出决定因素与结局的相关性。
（3）本研究未纳入重度血管病变导致的致死性事件，这可能会使脆弱性斑块与临床事件的相关性。
（4）罹患卒中或CHD的患者在改变生活方式和应用药物后会对动脉粥样硬化斑块的发展产生影响（改变生活方式和应用药物会使得粥样硬化斑块变得稳定）从而使得结果差异性减小。（5）本研究无法排除在颈动脉斑块在卒中或CHD后继续进展。这一问题需要进一步的研究应注重颈动脉斑块特征与卒中和CHD病史之间的关系，从而确认MRI影像学作为评定颈动脉粥样硬化影像学参数的作用优于传统的心血管危险因素评分如Framingham危险评分。

综上所述，本研究发现颈动脉斑块厚度和狭窄与缺血性卒中史和CHD病史具有相关性，但颈动脉IPH仅与缺血性卒中病史相关而与CHD无明显的相关性。下一步研究应着眼于颈动脉MRI检查在缺血性卒中和卒中的临床表现上可出现明显不同。

参考文献