Electrocardiographic Left Atrial Abnormality and Risk of Stroke
Northern Manhattan Study

Hooman Kamel, MD; Madeleine Hunter; Yeseon P. Moon, MS; Shadi Yaghi, MD; Ken Cheung, PhD; Marco R. Di Tullio, MD; Peter M. Okin, MD; Ralph L. Sacco, MD; Elsayed Z. Soliman, MD, MSc, MS; Mitchell S.V. Elkind, MD, MS

Background and Purpose—Electrocardiographic left atrial abnormality has been associated with stroke independently of atrial fibrillation (AF), suggesting that atrial thromboembolism may occur in the absence of AF. If true, we would expect an association with cryptogenic or cardioembolic stroke rather than noncardioembolic stroke.

Methods—We conducted a case-cohort analysis in the Northern Manhattan Study, a prospective cohort study of stroke risk factors. P-wave terminal force in lead V1 was manually measured from baseline ECGs of participants in sinus rhythm who subsequently had ischemic stroke (n=241) and a randomly selected subcohort without stroke (n=798). Weighted Cox proportional hazard models were used to examine the association between P-wave terminal force in lead V1 and stroke etiologic subtypes while adjusting for baseline demographic characteristics, history of AF, heart failure, diabetes mellitus, hypertension, tobacco use, and lipid levels.

Results—Mean P-wave terminal force in lead V1 was 4452 (±3368) μV*ms among stroke cases and 3934 (±2541) μV*ms in the subcohort. P-wave terminal force in lead V1 was associated with ischemic stroke (adjusted hazard ratio per SD, 1.20; 95% confidence interval, 1.03–1.39) and the composite of cryptogenic or cardioembolic stroke (adjusted hazard ratio per SD, 1.31; 95% confidence interval, 1.08–1.58). There was no definite association with noncardioembolic stroke subtypes (adjusted hazard ratio per SD, 1.14; 95% confidence interval, 0.92–1.40). Results were similar after excluding participants with a history of AF at baseline or new AF during follow-up.

Conclusions—ECG-defined left atrial abnormality was associated with incident cryptogenic or cardioembolic stroke independently of the presence of AF, suggesting atrial thromboembolism may occur without recognized AF. (Stroke. 2015;46:3208-3212. DOI: 10.1161/STROKEAHA.115.009989.)

Key Words: atrial fibrillation ▪ cardiomyopathies ▪ cohort studies ▪ embolism ▪ stroke

One third of ischemic strokes are of unknown cause.1 Many of these cryptogenic strokes seem to arise from a distant thrombus, prompting the recent definition of an entity called embolic stroke of undetermined source.2 Recent data suggest that many of these embolic-appearing cryptogenic strokes result from left atrial thromboembolism in the absence of atrial fibrillation (AF)—currently the only form of atrial disease with a recognized association with stroke.3 P-wave terminal force in lead V1 (PTFV1)—a standard electrocardiographic marker of left atrial abnormality4—has been associated with stroke5 and radiographic evidence of brain infarction6 even in the absence of known AF. Because PTFV1 is an established marker of left atrial pathophysiological changes such as hypertrophy and elevated filling pressures,4 these associations suggest that an atrial cardiopathy may lead to thromboembolism without necessarily manifesting with AF. If true, we would expect markers of atrial cardiopathy to be associated specifically with cryptogenic or cardioembolic strokes, as opposed to definite noncardioembolic strokes, such as those from in situ small-vessel occlusion or artery-to-artery embolism from atherosclerotic plaques. This would support the hypothesis that an unrecognized atrial cardiopathy may be responsible for some proportion of strokes that are currently of unknown cause. We therefore examined the association between PTFV1 and ischemic stroke subtypes, with a prespecified hypothesis that PTFV1 would be more strongly associated with cardioembolic or cryptogenic stroke as opposed to other types of stroke.
Methods

Design and Participants

The Northern Manhattan Study (NOMAS) prospectively enrolled a population-based sample of stroke-free men and women ≥39 years of age residing in Northern Manhattan, as previously described. All participants underwent a baseline in-person examination at home or the Columbia University Medical Center. The Columbia University Medical Center and University of Miami institutional review boards approved the NOMAS study, and all participants provided written, informed consent. For this study, we used a case-cohort design, in which the analytic cohort was a subsample of all NOMAS participants with available baseline ECG data. This analytic cohort comprised a 30% sample of the entire cohort (termed the subcohort), chosen at random using a random number generator in SAS version 9.3 (SAS Institute, Cary, NC), in addition to all participants with ischemic stroke during follow-up. This case-control design precluded the need for manual review of all ECGs while still allowing efficient comparison of the association between PTFV1 and multiple different stroke etiologic subtypes, although the incidence rates of individual stroke subtypes were low.

Measurements

The predictor variable was PTFV1, defined as the absolute value of the depth (μV) of the downward deflection (terminal portion) of the P wave in ECG lead V1, multiplied by its duration (ms; Figure). A single investigator (M.H.) blinded to participants’ stroke status during follow-up manually obtained these measurements from the baseline ECG of participants in our case-cohort sample. Digital calipers were used to measure P-wave features in mm from paper ECG, and were then converted to μV and ms using the ECG calibration of 10 mm/mV and 25 mm/s. Previous studies have shown excellent intrarater correlations and moderate inter-rater correlations for manual measurements of P-wave morphology. To confirm this in our cohort, a second investigator (S.Y.) independently performed blinded measurements of a random sample of 30 ECGs to allow assessment of intrarater and inter-rater reliability of PTFV1 measurements. In cases where we could not obtain baseline PTFV1 measurements because of inadequate ECG quality or absent P waves because of AF on the ECG, we coded participants’ PTFV1 value as missing and excluded them from our analyses.

The primary outcome was ischemic stroke of any type, and secondary outcomes were the following ischemic stroke subtypes: cryptogenic, cardioembolic, a composite of cryptogenic and cardioembolic, and all other subtypes combined (termed noncardioembolic). Stroke was defined as the first occurrence of an event fulfilling the World Health Organization definition of stroke. Participants were screened for stroke via annual telephone calls to identify hospitalizations or symptoms consistent with stroke, which was then confirmed through review of hospital records. Diagnostic evaluations from the stroke hospitalization included neuroimages, electrocardiography, extracranial duplex Doppler ultrasound, transcranial Doppler, and 2-dimensional echocardiogram, and when necessary, follow-up neuroimages, Holter monitor, transesophageal echocardiogram, and conventional cerebral angiogram. The stroke subtype was then determined by a diagnostic committee that reviewed all the available data and classified each ischemic stroke by causal mechanism based on a modified NINDS Stroke Data Bank scheme.

Statistical Analysis

We tabulated baseline characteristics by cases and the subcohort using frequencies and percentages for dichotomous variables and means with SDs or medians with interquartile ranges for continuous variables. Weighted Cox proportional hazard models were used to calculate the hazard ratio (HR) and 95% confidence interval (CI) for the association between PTFV1 and stroke while adjusting for the covariates above. Consistent with previous studies, the primary predictor (PTFV1) was used as a continuous variable in 1-SD increments. We built models incrementally to gauge the degree of changes in associations after the inclusion of model covariates. Model 1 included PTFV1 alone, model 2 additionally included baseline demographic characteristics, and model 3 additionally included all the covariates listed above. We used bootstrapping to calculate differences in the associations between PTFV1 and various stroke subtypes. In sensitivity analyses, we entirely excluded participants with a history of AF at baseline or new AF diagnosed during follow-up. Among the subset of participants with available baseline echocardiographic data, we performed sensitivity analyses adjusted for left atrial size and left ventricular hypertrophy to determine whether ECG-defined left atrial abnormality signals a heightened risk of thromboembolism apart from these other factors. We calculated the power based on the size of our sample (n=866, 30% of the full cohort) by comparing the upper quartile of PTFV1 versus the rest of our sample. The observed HR was 1.5 and the observed ischemic stroke incidence was 0.083; using these values, we had power of 80% to detect a significant difference with α=0.05. Even with 86 participants excluded because of unusable ECGs, the power was 78%. All statistical analyses were performed with SAS version 9.3 (SAS Institute, Cary, NC) and R version 3.1.

Results

Of 3298 NOMAS participants, 2887 (87.5%) had available baseline ECGs. Of these, 866 were included in our subcohort and 272 had an ischemic stroke during follow-up. Eighty-six participants had unusable ECGs and were thus excluded. Therefore, the final sample for this analysis included 241 participants with ischemic stroke and a subcohort of 798 participants (Table 1). All participants had normal sinus rhythm on the baseline ECG. Stroke mechanisms were classified as 33% cardioembolic, 27% small-vessel occlusion, 14% large-artery atherosclerosis, 21% cryptogenic, 3% other defined mechanisms, and 2% missing.

Intraclass correlation coefficients for PTFV1 measurements in the subset of 30 ECGs that were independently
Table 1. Baseline Characteristics of NOMAS Participants With Ischemic Stroke Versus a Random Subcohort

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Stroke (n=241)</th>
<th>Subcohort (n=798)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>70.2 (8.5)</td>
<td>67.1 (9.6)</td>
</tr>
<tr>
<td>Male</td>
<td>100 (41.5)</td>
<td>328 (41.1)</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>128 (53.1)</td>
<td>439 (55.0)</td>
</tr>
<tr>
<td>Black</td>
<td>66 (27.4)</td>
<td>195 (24.5)</td>
</tr>
<tr>
<td>White</td>
<td>42 (17.4)</td>
<td>147 (18.4)</td>
</tr>
<tr>
<td>Other</td>
<td>5 (2.1)</td>
<td>17 (2.1)</td>
</tr>
<tr>
<td>High school education or greater</td>
<td>111 (46.1)</td>
<td>383 (48.0)</td>
</tr>
<tr>
<td>Current tobacco use</td>
<td>42 (17.4)</td>
<td>136 (17.0)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>90 (37.3)</td>
<td>177 (22.2)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>200 (83.0)</td>
<td>605 (75.8)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>17 (7.1)</td>
<td>46 (6.1)</td>
</tr>
<tr>
<td>History of atrial fibrillation†</td>
<td>7 (2.9)</td>
<td>31 (3.9)</td>
</tr>
<tr>
<td>Low-density lipoprotein, mean (SD), mg/dL</td>
<td>128.1 (38.1)</td>
<td>130.0 (36.3)</td>
</tr>
<tr>
<td>High-density lipoprotein, mean (SD), mg/dL</td>
<td>44.9 (13.5)</td>
<td>46.2 (14.6)</td>
</tr>
</tbody>
</table>

NOMAS indicates Northern Manhattan Study.
*Data are presented as number (%) unless otherwise specified.
†All participants were in normal sinus rhythm on the baseline ECG.

Table 2. Associations Between P-Wave Terminal Force in ECG Lead V1 and Incident Ischemic Stroke Subtypes

<table>
<thead>
<tr>
<th>Outcome*</th>
<th>Model 1†</th>
<th>Model 2‡</th>
<th>Model 3§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any ischemic stroke</td>
<td>1.24 (1.07–1.42)</td>
<td>1.21 (1.04–1.39)</td>
<td>1.20 (1.03–1.39)</td>
</tr>
<tr>
<td>Ischemic stroke subtypes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptogenic or cardioembolic</td>
<td>1.31 (1.10–1.55)</td>
<td>1.28 (1.07–1.53)</td>
<td>1.31 (1.08–1.58)</td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>1.29 (0.99–1.68)</td>
<td>1.25 (0.95–1.65)</td>
<td>1.29 (0.96–1.72)</td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>1.32 (1.07–1.62)</td>
<td>1.30 (1.05–1.62)</td>
<td>1.23 (0.97–1.56)</td>
</tr>
<tr>
<td>Noncardioembolic</td>
<td>1.14 (0.94–1.40)</td>
<td>1.12 (0.92–1.37)</td>
<td>1.14 (0.92–1.40)</td>
</tr>
<tr>
<td>Sensitivity analyses</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excluding patients with any atrial fibrillation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any ischemic stroke</td>
<td>1.34 (1.12–1.59)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptogenic or cardioembolic</td>
<td>1.56 (1.25–1.96)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>1.31 (0.97–1.77)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>1.97 (1.42–2.75)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noncardioembolic</td>
<td>1.17 (0.93–1.47)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted for left atrial size on echocardiogram</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any ischemic stroke</td>
<td>1.08 (0.87–1.34)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptogenic or cardioembolic</td>
<td>1.17 (0.90–1.51)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cryptogenic</td>
<td>1.25 (0.82–1.91)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>1.10 (0.80–1.51)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noncardioembolic</td>
<td>0.95 (0.70–1.28)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Results are reported as the hazard ratio (95% confidence interval) for each 1-SD increase in P-wave terminal force in lead V1.
†Adjusted for age, sex, race, and level of formal education.
‡Adjusted for Model 2 covariates plus baseline smoking status, diabetes, hypertension, lipid levels, history of atrial fibrillation, and heart failure.
§Adjusted for Model 2 covariates plus baseline smoking status, diabetes, hypertension, lipid levels, history of atrial fibrillation, and heart failure.

Discussion

In a multiethnic population-based cohort, we found an association between ECG-defined left atrial abnormality and subsequent ischemic stroke. These associations appeared most marked for cryptogenic and cardioembolic strokes as opposed to noncardioembolic strokes, although this difference was not statistically significant, potentially because of low numbers of individual stroke subtypes. Our findings were unchanged or stronger when excluding patients with AF diagnoses. The associations between ECG-defined left atrial abnormality and most stroke subtypes were attenuated after adjustment for left atrial size on echocardiogram, indicating that PTFV1 partly reflects left atrial dilatation, which has been previously associated with stroke.13–17 These findings may be of particular relevance to currently unexplained racial disparities in stroke incidence because certain minority groups such as blacks have a higher rate of stroke18 but a lower rate of AF19 and a lower proportion of strokes that are attributable to AF.20

Previous studies have identified associations between markers of left atrial dysfunction and stroke. ECG-defined left atrial abnormality, left atrial size, left atrial pump function, and levels of amino terminal pro-B-type natriuretic peptide have been associated with stroke risk in the absence of apparent AF.5,6,13,16,21,22

assessed by 2 investigators demonstrated excellent intrarater reliability (0.87; 95% CI, 0.79–0.92) and moderate inter-rater reliability (0.69; 95% CI, 0.45–0.80).

Participants with ischemic stroke had a significantly higher PTFV1 (1452±3368 μV·ms) than those in the subcohort (3934±2541 μV·ms). In an unadjusted model, PTFV1 was associated with a higher risk of ischemic stroke (HR per SD, 1.20; 95% CI, 1.03–1.39; Table 2). This association did not substantially change after inclusion of potentially confounding or mediating covariates, including a history of AF (adjusted HR per SD, 1.20; 95% CI, 1.03–1.39; Table 2).

In adjusted models, we found an association between PTFV1 and a composite of cryptogenic or cardioembolic stroke (adjusted HR per SD, 1.31; 95% CI, 1.08–1.58). The findings were not statistically significant when stroke subtypes were considered separately as cryptogenic stroke (HR per SD, 1.29; 95% CI, 0.96–1.72), cardioembolic stroke (HR per SD, 1.23; 95% CI, 0.97–1.56), or noncardioembolic stroke (HR per SD, 1.14; 95% CI, 0.92–1.40). Associations between PTFV1 and stroke were unchanged or stronger when we entirely excluded participants who had a baseline history of AF or were diagnosed with AF during follow-up (Table 2). For cardioembolic or cryptogenic stroke, PTFV1 was associated with a 50% increase in risk (adjusted HR per SD, 1.56; 95% CI, 1.25–1.96). Among the 73% of participants with available baseline echocardiography data, the inclusion of left atrial size in our models attenuated the association between ECG-defined left atrial abnormality and most stroke subtypes were attenuated after adjustment for left atrial size on echocardiogram, indicating that PTFV1 partly reflects left atrial dilatation, which has been previously associated with stroke.13–17 These findings may be of particular relevance to currently unexplained racial disparities in stroke incidence because certain minority groups such as blacks have a higher rate of stroke18 but a lower rate of AF19 and a lower proportion of strokes that are attributable to AF.20

Previous studies have identified associations between markers of left atrial dysfunction and stroke. ECG-defined left atrial abnormality, left atrial size, left atrial pump function, and levels of amino terminal pro-B-type natriuretic peptide have been associated with stroke risk in the absence of apparent AF.5,6,13,16,21,22

In a multiethnic population-based cohort, we found an association between ECG-defined left atrial abnormality and subsequent ischemic stroke. These associations appeared most marked for cryptogenic and cardioembolic strokes as opposed to noncardioembolic strokes, although this difference was not statistically significant, potentially because of low numbers of individual stroke subtypes. Our findings were unchanged or stronger when excluding patients with AF diagnoses. The associations between ECG-defined left atrial abnormality and most stroke subtypes were attenuated after adjustment for left atrial size on echocardiogram, indicating that PTFV1 partly reflects left atrial dilatation, which has been previously associated with stroke.13–17 These findings may be of particular relevance to currently unexplained racial disparities in stroke incidence because certain minority groups such as blacks have a higher rate of stroke18 but a lower rate of AF19 and a lower proportion of strokes that are attributable to AF.20

Previous studies have identified associations between markers of left atrial dysfunction and stroke. ECG-defined left atrial abnormality, left atrial size, left atrial pump function, and levels of amino terminal pro-B-type natriuretic peptide have been associated with stroke risk in the absence of apparent AF.5,6,13,16,21,22

In a multiethnic population-based cohort, we found an association between ECG-defined left atrial abnormality and subsequent ischemic stroke. These associations appeared most marked for cryptogenic and cardioembolic strokes as opposed to noncardioembolic strokes, although this difference was not statistically significant, potentially because of low numbers of individual stroke subtypes. Our findings were unchanged or stronger when excluding patients with AF diagnoses. The associations between ECG-defined left atrial abnormality and most stroke subtypes were attenuated after adjustment for left atrial size on echocardiogram, indicating that PTFV1 partly reflects left atrial dilatation, which has been previously associated with stroke.13–17 These findings may be of particular relevance to currently unexplained racial disparities in stroke incidence because certain minority groups such as blacks have a higher rate of stroke18 but a lower rate of AF19 and a lower proportion of strokes that are attributable to AF.20

Previous studies have identified associations between markers of left atrial dysfunction and stroke. ECG-defined left atrial abnormality, left atrial size, left atrial pump function, and levels of amino terminal pro-B-type natriuretic peptide have been associated with stroke risk in the absence of apparent AF.5,6,13,16,21,22
Our results build on these studies by demonstrating that left atrial abnormality is not associated with noncardioembolic stroke subtypes, which provides reassurance that the overall association with stroke is not simply the result of residual confounding from global vascular risk factors. Instead, the stronger association with cardioembolic and cryptogenic strokes supports a specific link with left atrial thromboembolism.

Our study has several limitations. First, we relied on manual rather than electronic measurements of PTFV1. Given the blinded nature of our measurements, however, this would be expected to cause nondifferential misclassification and bias our results toward the null hypothesis. Second, without long-term heart-rhythm monitoring, we cannot rule out that subclinical AF explains the relationship between ECG-defined left atrial abnormality and stroke. However, several factors argue against this. In this and several previous studies, the inclusion of AF as a covariate in our models did not decrease the association between PTFV1 and stroke, whereas the inclusion of a mediator typically attenuates the relationship between a predictor and outcome. Indeed, our findings were stronger after excluding patients with clinically apparent AF, which is a more severe form than subclinical AF. Furthermore, 70% of patients with cryptogenic stroke in a recent trial manifested no AF even after 3 years of continuous heart-rhythm monitoring, indicating that paroxysmal AF does not account for most cryptogenic strokes and that other thromboembolic sources are at play. Finally, recent data indicate that the stroke risk seen with AF cannot be entirely explained by the dysrhythmia itself because 31% of patients with both AF and stroke in 2 recent studies did not manifest the dysrhythmia until after their stroke despite undergoing many months of continuous heart-rhythm monitoring beforehand. These findings are better explained by a mechanistic model in which AF reflects an atrial cardiopathy that causes thromboembolism, rather than by the traditional understanding that AF and of itself causes thromboembolism.

Even if AF does mediate the association between ECG-defined left atrial abnormality and stroke, markers of left atrial dysfunction that can be ascertained at a single visit may prove to be more clinically valuable, cost effective, and reliable for identifying thromboembolic risk than prolonged heart-rhythm monitoring to screen for AF. 12-lead ECG measures are especially appealing because of their low cost and broad applicability. Determination of stroke risk at a single point in time could preclude the need for long-term monitoring for AF, during which time the patient would remain at risk for stroke. Therefore, future studies may be warranted to identify the best markers of left atrial thromboembolism and to compare different antithrombotic strategies in patients at high risk even if they have no apparent AF.

Sources of Funding

This research was funded by grant K23NS082367 (Dr Kamel) and R37NS29993 (Drs Elkind and Sacco) from the National Institute of Neurological Disorders and Stroke. This publication was also supported by the National Center for Advancing Translational Sciences, National Institutes of Health, through Grant Number UL1TR000040. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH.

Disclosures

Dr Elkind receives compensation for providing consultative services for Biotlemetry/Cardionet, BMS-Pfizer Partnership, Boehringer-Ingelheim, Daiichi-Sankyo, and Janssen Pharmaceuticals; receives royalties from up to date for a chapter related to cryptogenic stroke; and serves a member of the American Stroke Association Cryptogenic Stroke Initiative Oversight Committee. Dr Okin serves as a consultant to and has received research grants from Novartis Pharmaceuticals. Dr Sacco received consultative compensation from Boehringer-Ingelheim for a trial on secondary stroke prevention with dabigatran. The other authors report no conflicts.

References


Electrocardiographic Left Atrial Abnormality and Risk of Stroke: Northern Manhattan Study
Hooman Kamel, Madeleine Hunter, Yeseon P. Moon, Shadi Yaghi, Ken Cheung, Marco R. Di Tullio, Peter M. Okin, Ralph L. Sacco, Elsayed Z. Soliman and Mitchell S.V. Elkind

Stroke. 2015;46:3208-3212; originally published online September 22, 2015;
doi: 10.1161/STROKEAHA.115.009989
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2015 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/46/11/3208

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2016/05/30/STROKEAHA.115.009989.DC1

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/
心電図で検出された左房異常と脳卒中リスク
Northern Manhattan 試験

Electrocardiographic Left Atrial Abnormality and Risk of Stroke
Northern Manhattan Study

Hooman Kamel, MD1; Madeleine Hunter2; Yeseon P. Moon, MS2, et al.

1Department of Neurology, Feil Family Brain and Mind Research Institute, Weill Cornell Medical College, New York; and 2Department of Neurology, Columbia College of Physicians and Surgeons, New York.

脳卒中を予防するためには、脳卒中の予測因子を把握することが重要である。この研究の目的は、心電図（ECG）から左心房の異常を検出し、それが脳卒中のリスクを増加させるかどうかを検証することである。

方法：本研究は、ニューヨーク市のNorthern Manhattan地域での追跡調査である。1,000人以上の成人を対象に、1986年から1988年にかけての当たり前の生活習慣を調査した。調査結果から、左心房の電図異常と脳卒中のリスクの関連性を評価した。

結果：左心房の電図異常は、脳卒中のリスクを増加させるとの結果が得られた。特に、Q波の形状異常や心室期延長が脳卒中のリスクを増加させるとされる傾向が観察された。

結論：心電図から左心房の異常を検出し、それが脳卒中のリスクを増加させることが示唆された。今後は、より多くのデータを用いて、この関連性をさらに詳しく検討する必要がある。