Correlation of Echocardiographic Findings With Cerebral Infarction in Elderly Adults
The AGES-Reykjavik Study

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Background and Purpose—Chronic effects of hypertension may be observed in multiple end organs. Previous reports suggest that cardiovascular morphological features can mirror cerebral infarction. In this cross-sectional analysis of elderly subjects, we investigated the relationship of a comprehensive set of echocardiographic measures with cerebral infarction detected by MRI.

Methods—We compared echocardiographically determined left ventricular (LV) mass, left atrial volume, aortic root diameter, mitral annular calcification, and measures of diastolic function with cerebral infarction determined by MRI using logistic regression in a random sample drawn from the Age Gene/Environment Susceptibility–Reykjavik Study cohort. The model was first adjusted for age and gender, and then for age, gender, and vascular risk factors.

Results—Among 692 subjects aged 75 (standard deviation, 6) years, 28% had at least 1 cerebral infarct. When adjusted for age and gender, the presence of cerebral infarction was modestly related to LV mass (odds ratio [OR], 1.01; 95% confidence interval [CI], 1.00–1.02) and left atrial volume (OR, 1.03; 95% CI, 1.01–1.05), as well as the lowest quartile of early-to-late pulsed Doppler velocity ratio (early-to-late pulsed Doppler velocity ratio <0.75; OR, 1.87; 95% CI, 1.22–2.87). The latter relation remained significant after adjustment for vascular risk factors and LV ejection fraction (OR, 1.82; 95% CI, 1.16–2.86).

Conclusion—Of all echocardiographic parameters, LV filling abnormality as indicated by low early-to-late pulsed Doppler velocity ratio displayed the strongest association with cerebral infarction and this relationship was independent of vascular risk factors. This simple marker of cerebral infarction may be useful when evaluating older patients. (Stroke. 2010;41:2223-2228.)

Key Words: aging ■ cerebral infarction ■ echocardiography ■ epidemiology ■ magnetic resonance imaging

Chronic hypertension leads to concomitant remodeling of the cardiac and vascular systems. Atherosclerotic changes in the cerebrovascular system, ultimately leading to incident stroke, are mirrored by development of left ventricular (LV) hypertrophy, resultant diastolic dysfunction, and heart failure with preserved systolic function. Thus, assessment of ventricular morphology and function may be indicative of the extent of vascular disease in less easily accessible sites such as the brain. Although the association of LV hypertrophy detected by ECG with stroke outcome was described many years ago,1 advanced imaging techniques including 2-dimensional and Doppler echocardiography have largely supplanted the ECG in measuring left ventricular mass (LVM) and provided further insights into associated changes of ventricular function.

Echocardiographic predictors of vascular outcome focused initially on LV morphology. LV hypertrophy was found to reflect the severity and chronicity of systemic hypertension.2 In related fashion, LVM and other cardiovascular measures have been reported to predict stroke risk.3 In fact, in the Framingham Study, for each 50-g increase in LVM, there was \( \approx 1.5 \)-fold increase in relative risk for subsequent cardiovascular events.4 Other echo findings associated with chronic hypertension and stroke include mitral annular calcification height and enlargement of the left atrium (LA) and aortic root.5–8

Much previous epidemiological work investigating the cardio-cerebrovascular relationship was limited by its reliance on single-dimensional or M-mode technology. Two-dimensional and 3-dimensional assessment is now available...
to better-delineate cardiac morphology, as are sensitive measures of diastolic and systolic ventricular function that may be affected by chronic hypertension. The latter includes pulsed and tissue Doppler measures of ventricular relaxation and filling. In addition, previous work focused on clinical stroke data that necessarily failed to evaluate the presence of silent cerebral infarction. Importantly, MRI screening can detect not only previous clinical but also subclinical cerebral infarction,9 which is consequential because clinical and silent cerebral infarctions have been related to cognitive decline and dementia in older subjects.10–12

We tested the relationship of newer measures of cardiac morphology and function to cerebral infarction detected by MRI in a well-characterized community-based cohort of older subjects (AGES-Reykjavik).13

**Patients and Methods**

AGES-Reykjavik is an extension of the Reykjavik Study, a community-based cohort established in 1967 to study cardiovascular disease prospectively in Iceland. The rationale and design of AGES-Reykjavik, which is cosponsored by the Icelandic Heart Association and the National Institute of Aging, National Institutes of Health, has been described elsewhere.13 AGES-Reykjavik has been approved by the Icelandic National Bioethics Committee and the National Institute of Aging Institutional Review Board. Between 2002 and 2006, 5764 men and women participated in detailed phenotypic evaluations of cardiovascular, neurocognitive, musculoskeletal, and metabolic systems. Within this cohort, all eligible subjects were offered a cerebral MRI, and 954 subjects were selected randomly for cardiac assessment by echocardiography. The brain and cardiac evaluations were performed within a 1-month interval.

**Cerebral Infarction**

High-resolution MRI images were acquired on a 1.5-T Signa Twinspeed system (General Electric Medical Systems). The image protocol consisted of axial T1-weighted 3-dimensional spoiled-gradient echo, T2*-weighted gradient echo-type echo planar images, proton density/T2-weighted fast-spin echo, and fluid-attenuated inversion recovery sequences.

Cerebral infarcts were identified by trained radiographers as defects in brain parenchyma with associated hyperintensity on T2 and fluid-attenuated inversion recovery images with a maximal diameter of at least 4 mm, with the exception of cerebellar and brain stem infarcts or infarcts with cortical involvement, which had no size criterion.14 Five percent of all scans were re-read by blinded master readers at Leiden University Medical Center, The Netherlands, to assess inter-reader reliability. A set of scans was also re-read by all readers for presence or absence of parenchymal defects and to calculate average intra-reader and inter-reader reliabilities, which were good (weighted kappa = 0.9 and 0.7, respectively).

**Echocardiographic Assessment**

Standard 2-dimensional pulsed and tissue Doppler imaging was performed in long and short parasternal and 3 apical views with standard equipment (Acuson Sequoia C512). Studies were acquired digitally using established imaging protocols15 during free breathing in the left lateral supine position and stored for off-line analysis (Digiview Workstation; Digisonics). Studies were read qualitatively by an experienced cardiologist. Linear point-to-point and area tracings were made from 2-dimensional studies by ultrasound technicians specially trained in image quantification. All Doppler studies were acquired at sweep speeds of 50 mm/sec. Subjects in atrial fibrillation during the study or with significant valvular heart disease were excluded.

**Echocardiographic Measures**

LVM was calculated using the American Society of Echocardiography modification of the Penn formula.16 LA volume was determined by modified Simpson biplane method of discs, which included apical 4- and 2-chamber views.17 The presence and height of mitral annular calcification were measured from 2-dimensional images.5 LV ejection fraction was assessed qualitatively by an experienced cardiologist.

**Assessment of Diastolic Function**

All LV diastolic filling was assessed during apical 4-chamber imaging. The pulsed Doppler sample volume was placed at the mitral leaflet tips to determine transmural blood velocities. The leading edge of the transmural Doppler velocity profile was measured to derive peak early (E) and late atrial phase (A) LV filling and their ratios. Tissue Doppler imaging of mitral annular velocities (E’, A’) were measured from septal and lateral edges of the mitral annulus.

A 10% sample of all studies was selected randomly and reviewed both qualitatively and quantitatively by echocardiographic physicians at the National Heart, Lung, and Blood Institute, National Institutes of Health, who also provided training and quality oversight. Interobserver agreements were good for the overall measures, with Spearman correlation coefficients varying from 0.70 for measurement of LV wall septal thickness to 0.98 for early-to-late pulsed Doppler velocity (E/A) ratio.

**Covariates**

We controlled for demographic and vascular risk factors associated with both cerebral infarction and cardiovascular disease. BMI was calculated as weight (kilograms) divided by height squared (meters). History of atrial fibrillation and use of anticoagulant or antiplatelet drugs were noted. Smoking status was determined by self-report and categorized as smoker (current or smoking within the past 12 months) or nonsmoker. Fasting HDL and LDL cholesterol levels were measured. Hypertension was defined as systolic blood pressure >140 mm Hg, diastolic blood pressure >90 mm Hg, or use of an antihypertensive medication. Diabetes was based on self-report, fasting plasma glucose concentration >7 mmol/L, or use of oral hypoglycemic medication or insulin. Previous coronary heart disease was defined as a documented history of coronary artery disease or coronary bypass surgery.

**Statistical Analysis**

General characteristics of subjects with and without cerebral infarction were compared using logistic regression. Echocardiographic characteristics of subjects with and without cerebral infarction were analyzed as continuous variables and as quartiles using logistic regression. LVM and LA volume were indexed to body surface area18 to allow comparison independent of obesity.

The relation between E/A ratio and cerebral infarction was analyzed using E/A ratio as a continuous variable and in a quadratic model. Because of a U-shape relation between E/A ratio and mortality with low and high E/A ratios (<0.75 and >1.5) associated with excess mortality,19,20 E/A ratio data were divided into quartiles; the quartile thresholds were 0.74, 0.88, and 1.03. Covariates for these quartiles of E/A ratio were examined using logistic regression for categorical variables and linear regression for continuous variables, adjusted for age and gender for variables other than age and gender.

The overall difference in odds ratio (OR) of cerebral infarction between the quartiles of E/A ratio was analyzed using logistic regression. Then, subjects in the lowest and highest quartiles were each compared with subjects in the 2 middle quartiles. Because the highest quartile included 130 subjects with E/A ratio <1.5 (reported normal range, 0.75–1.5),20 a sensitivity analysis was performed in which the lowest quartile (E/A ratio <0.75) was compared with the 3 other quartiles combined. Last, the lowest and highest E/A ratios (<0.75 and >1.5) were compared with the reported normal range of E/A ratio (0.75–1.5).20
The model was adjusted for age and gender (model I) and then adjusted for BMI, current systolic and diastolic blood pressures, hypertension, smoking status, HDL cholesterol level, diabetes, and previous documented coronary heart disease (model II). Because of the high prevalence of hypertension (79%), measured blood pressures were included in the model. Last, the model was adjusted for LV ejection fraction (model III). In models in which we found a significant association with overall cerebral infarction, we re-ran the analysis using clinical stroke by self-report as the dependent variable.

Data were expressed as mean (standard deviation). The OR and 95% confidence intervals (CI) were computed using SAS 9.1/SAS Enterprise Guide (v4.1). In all analyses, the conventional α-level of 0.05 was used for significance testing.

**Results**

Of the 954 subjects selected randomly from the AGES-Reykjavik cohort, 122 subjects did not undergo cerebral MRI because of contraindications, incomplete protocol to evaluate infarction, or logistic reasons including refusal or disability. Another 140 subjects were excluded because they had >2 missing echocardiogram values. Thus, the study population consisted of 692 subjects. Compared to those included, excluded subjects were significantly older (77 [6] vs 76 [6]; P<0.001), more likely to be men (58% vs 53%; P=0.001), and more likely to have cerebral infarction (41% vs 28%; P=0.01).

In the study population, 193 (28%) subjects had MRI evidence of cerebral infarction, but only 42 (6%) subjects described a previous clinical stroke. When adjusted for age and gender, subjects with cerebral infarction were significantly older (78 [6] vs 75 [5]; P<0.0001) and more likely to be male (55% vs 36%; P<0.0001). They were also more likely to have had a previous documented coronary event (26% vs 11%; P=0.0002) and to have diabetes (21% vs 10%; P=0.0001). Subjects with cerebral infarction had higher systolic blood pressure (146 [21] vs 141 [19]; P=0.02) and were more likely to use anticoagulant or antiplatelet agents (53% vs 35%; P=0.002).

Table 1 shows the association of cerebral infarction with echocardiographic parameters. In model I, adjusted for age and gender, LVM and LA volume indices were significantly higher in participants with cerebral infarction, but cerebral infarction was not significantly related to aortic root diameter, LV ejection fraction, or lateral E/E’ ratios, whether analyzed as continuous variables or as quartiles (quartile results not shown). Similarly, the presence of mitral annular calcification was not associated with cerebral infarction. In model II, adjusted for age, gender, and vascular risk factors, both LVM and LA volume indices became marginally significant. For a 10% increase in LVM or LA volume, the OR were 1.08 (95% CI, 1.00–1.16) and 1.06 (95% CI, 1.00–1.12), respectively. When subjects with a history of atrial fibrillation were excluded, LVM and LA volume remained marginally significant (OR, 1.01; 95% CI, 1.00–1.02; OR, 1.01; 95% CI, 1.00–1.13; model II), respectively.

Whether analyzed as a continuous variable or in a quadratic model, E/A ratio was not significantly related to the presence or absence of cerebral infarction (OR, 0.90 [0.33] vs 0.95 [0.31]; P=0.13 for continuous variable; quadratic results not shown). When E/A ratio was divided into quartiles, as specified in the Methods, increasing E/A ratio quartile was associated with younger age, reduced prevalence of diabetes, and lower diastolic blood pressure (Table 2). The second and third quartiles did not differ significantly except for serum glucose (5.92 [1.43] vs 5.63 [0.71]; P=0.01).

There was a significant overall relationship between E/A ratio quartiles and cerebral infarction (P=0.04). When compared with the 2 middle quartiles, the lowest E/A ratio quartile (<0.75) was significantly related to cerebral infarction in model I, model II, and model III (OR, 1.82; 95% CI, 1.16–2.86; Table 3).

When compared with the 3 other quartiles, E/A ratio <0.75 was also significantly related to cerebral infarction (OR, 1.98; 95% CI, 1.35–2.90; model III; Supplemental Table available online at http://stroke.ahajournals.org).

When compared with the normal range of E/A ratio (0.75–1.5), E/A ratio <0.75 was consistently significant in all models (OR, 2.03; 95% CI, 1.37–2.99; model III; Supplemental Table).

**Table 1. Association Between Echocardiographic Parameters and Cerebral Infarction.**

<table>
<thead>
<tr>
<th>Echocardiographic Parameters</th>
<th>Cerebral Infarction, Mean (SD)</th>
<th>OR (95% CI)*</th>
<th>OR (95% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV mass index, gm/m²</td>
<td>87.2 (21.3)</td>
<td>97.6 (29.6)</td>
<td>1.01 (1.00–1.02)</td>
</tr>
<tr>
<td>LA volume index, ml/m²</td>
<td>31.2 (8.82)</td>
<td>35.4 (12.2)</td>
<td>1.03 (1.01–1.05)</td>
</tr>
<tr>
<td>Aortic root diameter, cm</td>
<td>3.16 (0.45)</td>
<td>3.26 (0.48)</td>
<td>1.12 (0.72–1.75)</td>
</tr>
<tr>
<td>Mitral annular calcification, % (n)</td>
<td>30.9 (149)</td>
<td>34.2 (64)</td>
<td>0.93 (0.64–1.37)</td>
</tr>
<tr>
<td>E/E’ lateral</td>
<td>7.61 (2.26)</td>
<td>7.71 (3.15)</td>
<td>0.99 (0.92–1.07)</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.95 (0.31)</td>
<td>0.90 (0.33)</td>
<td>0.63 (0.35–1.14)</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>62.8 (5.8)</td>
<td>61.4 (8.8)</td>
<td>0.99 (0.96–1.01)</td>
</tr>
</tbody>
</table>

*Model I: logistic regression adjusted for age and gender.
†Model II: logistic regression adjusted for age and gender, current or recent smoker, body mass index, previous coronary artery disease, diabetes, hypertension, HDL cholesterol, and current systolic and diastolic blood pressures.

E/A indicates ratio of early (E) to late (A) diastolic transmitral Doppler flow velocity; E/E’ ratio of early diastolic (E) transmitral Doppler flow velocity and early diastolic (E’) tissue Doppler mitral annular velocity; LA, left atrium; LV, left ventricle.
For subjects with clinical stroke by self-report (n = 1100), the lowest quartile of E/A ratio had a similar point estimate but was not significant in any of the models (OR, 1.97; 95% CI, 0.95–4.08; model I). The highest E/A quartile was not significantly different from the 2 middle quartiles in unadjusted and adjusted models (Table 3) or from the normal range of E/A ratio of 0.75 to 1.5 in any model (OR, 1.51; 95% CI, 0.68–3.35; model I; Supplemental Table). When subjects with a history of atrial fibrillation were excluded, the results were unchanged.

Discussion

In this cross-sectional analysis, there was a robust association between a low E/A ratio (<0.75) and cerebral infarction.

Table 2. Characteristics of the Study Population Based on Quartiles of E/A Ratio

<table>
<thead>
<tr>
<th>General Characteristics, Mean (SD)</th>
<th>1st Quartile n=162</th>
<th>2nd Quartile n=163</th>
<th>3rd Quartile n=163</th>
<th>4th Quartile n=163</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>76.5 (5.7)</td>
<td>75.5 (5.5)</td>
<td>74.5 (5.6)</td>
<td>75.5 (5.5)</td>
<td>0.01</td>
</tr>
<tr>
<td>Men, % (n)</td>
<td>28.1 (73)</td>
<td>21.5 (56)</td>
<td>27.3 (71)</td>
<td>23.1 (60)</td>
<td>0.14</td>
</tr>
<tr>
<td>Height, cm</td>
<td>166.4 (9.8)</td>
<td>166.3 (8.9)</td>
<td>167.5 (10.2)</td>
<td>167.4 (9.4)</td>
<td>0.04</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>76.0 (14.8)</td>
<td>75.9 (13.5)</td>
<td>76.3 (13.8)</td>
<td>73.7 (14.8)</td>
<td>0.35</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.4 (4.5)</td>
<td>27.4 (4.0)</td>
<td>27.2 (4.3)</td>
<td>26.3 (4.8)</td>
<td>0.06</td>
</tr>
<tr>
<td>Current or recent smoking, % (n)</td>
<td>30.1 (104)</td>
<td>23.4 (81)</td>
<td>22.3 (77)</td>
<td>24.3 (84)</td>
<td>0.06</td>
</tr>
<tr>
<td>Previous coronary event, % (n)</td>
<td>28.0 (28)</td>
<td>19.0 (19)</td>
<td>21.0 (21)</td>
<td>32.0 (32)</td>
<td>0.15</td>
</tr>
<tr>
<td>Current or previous hypertension, % (n)</td>
<td>26.2 (135)</td>
<td>24.3 (125)</td>
<td>23.3 (120)</td>
<td>26.2 (135)</td>
<td>0.18</td>
</tr>
<tr>
<td>Diabetes mellitus, % (n)</td>
<td>26.6 (30)</td>
<td>28.0 (23)</td>
<td>14.6 (12)</td>
<td>20.7 (17)</td>
<td>0.03</td>
</tr>
<tr>
<td>Systolic BP†</td>
<td>143.4 (19.8)</td>
<td>144.2 (19.4)</td>
<td>140.8 (18.7)</td>
<td>142.7 (21.1)</td>
<td>0.5</td>
</tr>
<tr>
<td>Diastolic BP†</td>
<td>75.4 (8.8)</td>
<td>75.1 (8.5)</td>
<td>74.1 (7.5)</td>
<td>71.8 (8.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulse pressure†</td>
<td>68.0 (17.2)</td>
<td>69.1 (18.2)</td>
<td>66.7 (16.9)</td>
<td>70.9 (17.8)</td>
<td>0.25</td>
</tr>
<tr>
<td>History of atrial fibrillation, % (n)</td>
<td>0</td>
<td>14.3 (1)</td>
<td>14.3 (1)</td>
<td>71.4 (5)</td>
<td>0.26</td>
</tr>
<tr>
<td>Coumadin/antiplatelet drugs, % (n)</td>
<td>24.3 (59)</td>
<td>23.0 (56)</td>
<td>23.9 (58)</td>
<td>28.8 (70)</td>
<td>0.28</td>
</tr>
<tr>
<td>Antihypertensive drugs, % (n)</td>
<td>25.7 (100)</td>
<td>23.3 (91)</td>
<td>23.6 (92)</td>
<td>27.4 (107)</td>
<td>0.28</td>
</tr>
<tr>
<td>Statin use, % (n)</td>
<td>23.8 (34)</td>
<td>21.7 (31)</td>
<td>22.4 (32)</td>
<td>32.2 (46)</td>
<td>0.12</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>5.99 (1.12)</td>
<td>5.92 (1.43)</td>
<td>5.63 (0.71)‡</td>
<td>5.81 (1.34)‡</td>
<td>0.02</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L§</td>
<td>1.55 (0.50)</td>
<td>1.54 (0.39)</td>
<td>1.55 (0.39)</td>
<td>1.65 (0.44)§</td>
<td>0.06</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L§</td>
<td>3.54 (1.03)</td>
<td>3.61 (1.07)</td>
<td>3.70 (0.95)</td>
<td>3.44 (1.06)</td>
<td>0.36</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.26 (0.66)</td>
<td>1.21 (0.63)</td>
<td>1.22 (0.64)</td>
<td>1.13 (0.54)</td>
<td>0.25</td>
</tr>
<tr>
<td>High-sensitivity CRP, mg/L</td>
<td>4.56 (10.68)</td>
<td>3.3 (4.42)</td>
<td>3.39 (5.65)</td>
<td>3.16 (4.60)</td>
<td>0.23</td>
</tr>
</tbody>
</table>

*Linear and logistic models to compare overall difference between the four quartiles of E/A ratio, adjusted for age and gender.†Adjusted for antihypertensive drugs.‡P = 0.01 comparing 2nd and 3rd E/A quartiles adjusted for age and gender.§Adjusted for statin use.

BP indicates blood pressure (mm Hg); CRP, C-reactive protein; E/A, ratio of early (E) to late (A) diastolic transmitral Doppler flow velocity.

For subjects with clinical stroke by self-report (n = 42), the lowest quartile of E/A ratio had a similar point estimate but was not significant in any of the models (OR, 1.97; 95% CI, 0.95–4.08; model I). The highest E/A quartile was not significantly different from the 2 middle quartiles in unadjusted and adjusted models (Table 3) or from the normal range of E/A ratio of 0.75 to 1.5 in any model (OR, 1.51; 95% CI, 0.68–3.35; model I; Supplemental Table). When subjects with a history of atrial fibrillation were excluded, the results were unchanged.

Table 3. Association of Cerebral Infarction with Quartiles of E/A Ratio

<table>
<thead>
<tr>
<th>E/A Ratio Quartile*</th>
<th>Cerebral Infarction, % (n)</th>
<th>OR (95% CI)†</th>
<th>OR (95% CI)‡</th>
<th>OR (95% CI)§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lowest (first)</td>
<td>21.2 (102)</td>
<td>35.3 (60)</td>
<td>1.87 (1.22–2.87)</td>
<td>1.84 (1.17–2.88)</td>
</tr>
<tr>
<td>Second</td>
<td>26.6 (128)</td>
<td>20.6 (35)</td>
<td>1 (reference)</td>
<td>1 (reference)</td>
</tr>
<tr>
<td>Third</td>
<td>26.4 (127)</td>
<td>21.2 (38)</td>
<td>22.9 (38)</td>
<td>1.11 (0.70–1.76)</td>
</tr>
</tbody>
</table>

*Quartile thresholds: 0.74, 0.88, and 1.03.†Model I: logistic regression adjusted for age and gender.‡Model II: logistic regression adjusted for age and gender, current or recent smoker, BMI, previous coronary artery disease, diabetes, hypertension, LDL cholesterol, and current systolic and diastolic blood pressures.§Model III: model II + LV ejection fraction.

Discussion

In this cross-sectional analysis, there was a robust association between a low E/A ratio (<0.75) and cerebral infarction...
independent of age, gender, vascular risk factors, and LV ejection fraction. Even though LVM and LA volume, 2 well-known associations of chronic hypertension, were associated with cerebral infarction independently of age and gender, when the model was adjusted for vascular risk factors, the association became marginally significant. Using more advanced techniques, these results confirm some previous M-mode–based predictors of clinical stroke, including LA size and LVM. However, some, including aortic root size and mitral annular calcification measurements as determined with 2-dimensional point-to-point measurements, were not associated with cerebral infarction. Because of the spatial ambiguity associated with M-mode assessment, these new results seem more plausible but will require confirmation in future 2- and 3-dimensional evaluations. Another possibility is that the high prevalence of vascular risk factors in this elderly cohort obscured any independent association of aortic root diameter and mitral annular calcification with cerebral infarction.

Many earlier studies used M-mode echo data, and the diagnosis of stroke was based on clinical findings with or without cerebral imaging by CT or MRI.4,5,21–23 However, LVM has been related to cerebral infarction detected by MRI in blacks in whom prevalence of combined clinical and silent cerebral infarct was higher than that of classical stroke alone (20% vs 3%), and these findings are consistent with ours.9 Previous studies have already shown that abnormalities of LV early and late filling velocities (ie, low or high E/A ratios) are associated with increased all-cause mortality.20 Low E/A ratio (<0.6) was associated with higher all-cause and cardiac mortality in the Strong Heart Study of American Indians, but it was not an independent predictor after adjustment for covariates.9 The Strong Heart Study also described higher mortality in younger subjects (57 [7] years) with a restrictive filling pattern indicated by an E/A ratio >1.5. In this AGES-Reykjavik cohort, there were only 33 subjects with E/A ratio >1.5, so no conclusion can be drawn about any associations with the higher value of this parameter. This can also explain the absence of quadratic relation of E/A ratio with cerebral infarction.

E/A ratio describes flow velocities in early and late diastole. Besides describing phasic shifts of LV filling to late diastole, a low E/A ratio may reflect generalized changes in the vascular system, including alterations in laminar flow and a cascade of adverse effects on platelet aggregation and endothelial cell function.24 These maladaptive vascular changes, characterized by heightened oxidative stress and increased proinflammatory and prothrombotic states, could contribute to endothelial dysfunction.25,26 Thus, the mechanisms underlying the relationship between E/A ratio and stroke may be multifactorial.

E′, an indicator of LV passive stiffness,27 has been associated with ischemic stroke in subjects with atrial fibrillation,28 but it was not related to cerebral infarction in AGES-Reykjavik. However, this earlier study reported larger ranges and higher mean values for E/E′,28 suggesting stiffer ventricles than were present in AGES-Reykjavik, which could account for the differing results.

Study Limitations

The study has a number of limitations. The subjects are the survivors who are still alive 25 years after the Reykjavik Study was initiated, and so the relation of factors to a lethal disease can be underestimated. This limitation could explain why LVM and LA volume have only a modest association with cerebral infarction. Similarly, recall bias could affect characterization of covariates, but this effect should be equally distributed among the study groups. A second limitation is the cross-sectional design, which, in comparison with most previous studies of echocardiographic markers and stroke, does not allow determination of risk, identification of predictors, or sequence of events. The sample size is modest and power may have been reduced by exclusion of subjects without adequate echo measures.

Conclusion

This study demonstrates that even in older subjects, LV mass, LA volume, and low E/A ratio were associated with cerebral infarction detected by MRI. However, the association between low E/A ratio and cerebral infarction appears to be independent of concurrent cardiovascular risk factors, such as hypertension. This relationship is novel in that previous studies have associated cardiac morphology, not diastolic function, with stroke. Thus, E/A ratio, a simple marker of clinical and subclinical cerebral infarction, could be especially useful in elderly patients because a low ratio in patients with cognitive decline and dementia may suggest an etiology of cerebral ischemia.25

Acknowledgements

The authors thank the participants of the study and the Icelandic Heart Association clinic staff for their invaluable contribution.

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Disclosure

None.

References


老年人脑梗死与超声心动图结果的相关性

AGES-Reykjavik 研究

Correlation of Echocardiographic Findings With Cerebral Infarction in Elderly Adults

The AGES-Reykjavik Study

Dorothea McAreavey, MD; Jean-Sebastien Vidal, MD, PhD; Thor Aspelund, PhD; David S. Owens, MD; Timothy Hughes, MPH; Melissa Garcia, MPH; Sigurdur Sigurdsson, MSc; Halldora Bjornsdottir, MD; Tamara B. Harris, MD; Vilmundur Gudnason, MD, PhD; Lenore J. Launer, PhD; Jonathan F. Plehn, MD

背景和目的：观察发现高血压对多个靶器官具有慢性作用。过去研究表明心血管的形态学特点可反映脑梗死的情况。本横断面分析探讨老年人核磁共振成像(MRI)发现的脑梗死与超声心动图结果的相关性。

方法：从AGES-Reykjavik研究队列中随机抽取样本，用logistic回归分析比较MRI发现的脑梗死与超声心动图结果的关系，包括左心室体积(LVM)、左心房容积、主动脉根部直径、二尖瓣钙化、舒张功能指标。本模型首先调整了年龄与性别差异，然后调整了年龄、性别、血管危险因素。

结果：692例75岁(标准差，6)的个体中，28%至少曾出现一次脑梗死。当调整了年龄和性别后，脑梗死与LVM(优势比[OR], 1.01; 95%可信区间[CI], 1.00-1.02)、左心房容积(OR, 1.03; 95% CI 1.01-1.05)轻度相关，也与最小四分位数早期和晚期的脉冲多普勒的血流速度比值(早期和晚期的脉冲多普勒的血流速度比<0.75; OR, 1.87; 95% CI 1.22-2.87)相关。经过血管危险因素和左室射血分数调整后，后者的相关性仍然十分明显(OR, 1.82; 95% CI, 1.16-2.86)。

结论：在所有超声心动图参数中，早期和晚期的脉冲多普勒的血流速度比值提示的心室充盈异常与脑梗死相关性最大，且独立于血管危险因素之外。用这个简单的脑梗死指标评估中老年患者可能有价值。

关键词：老年化，脑梗死，超声心动图，流行病学，核磁共振成像

(Stroke, 2010;41:2223-2228. 暨南大学附属第一医院神经内科 黎泳欣 译 徐安定 校)
能力降低和痴呆相关。我们利用一个基于社区队列老年群体 (AGES-Reykjavik) 的研究资料, 观察了心脏形态学和功能的一些更新的指标和 MRI 发现脑梗死的相关性。

**患者和方法**

AGES-Reykjavik 是 Reykjavik 研究的延伸, 这是一个于 1967 年在冰岛建立的基于社区群体的心血管疾病的前瞻性研究。该研究由冰岛心脏协会、国际老年协会和冰岛健康协会共同申办, 其立题依据和设计已经在既往发表, 并被冰岛国家生物伦理学委员会和国家老年研究所批准。在 2002-2006 年, 共 5764 名男性和女性参加了包括心血管、神经认知、骨骼肌肉和代谢系统的详细的表型评估。在该人群中, 合适的人都进行了脑部 MRI 检查, 其中 954 人被随机抽取进行了超声心动图的心脏评估。脑和心脏评估的间隔时间控制在一个月内。

脑梗死

高分辨率 MRI 扫描采用 1.5-T Sigma 双速系统 (General Electric Medical Systems)。扫描程序包括轴向 T1 加权三维扰相梯度回波、T2* 加权梯度回波、质子密度/T2 加权快速自旋回波序列以及液体衰减反转恢复 (FLAIR) 序列。

脑梗死由训练有素的放射学人员诊断, 表现为脑实质区域 T2 及 FLAIR 成像高信号, 直径至少为 4 mm, 但小脑、脑干或脑皮质梗死没有大小判定标准。为了评估阅片者间一致性, 5% 的扫描在荷兰的莱顿大学医学中心被不知情的熟练的阅片者重读。阅片者间的一致性良好, 左心室壁中隔厚度、早期和晚期的脉冲多普勒的血流速度比值 (E/A) 的 Spearman 相关系数分别是 0.7 和 0.98。

超声心动图的测量

LVM 采用美国超声心动描记学会的 Penn 修正公式计算。左心房容积采用修正的双平面 Simpson 法测量, 其包括心尖 4 腔、2 腔视图。二尖瓣环状钙化的出现及其厚度由二维图像测量。左心室 (LV) 射血分数 (EF) 由有经验的心脏学家评估。舒张功能的测量

舒张功能的测量

左心室的舒张充盈在心尖 4 腔图像中测量。为了测定二尖瓣间血流速度, 脉冲多普勒取样容积定在二尖瓣小叶位置上测量。经二尖瓣多普勒速度分布图的主导边缘测量来取得 LV 充盈过程中早 (E) 和晚 (A) 心房位相峰值和它们的比值。二尖瓣环状流速 (E', A') 的组织多普勒图像从二尖瓣中隔和侧边测量。从所有研究中随机抽取 10% 样本, 由国家心、肺、血液协会和国家健康组织的超声心动图技师从数量和质量上进行回顾并提供训练和质量监督。阅片者间的一致性良好, 左心室壁中隔厚度、早期和晚期的脉冲多普勒的血流速度比值 (E/A) 的 Spearman 相关系数分别是 0.7 和 0.98。

协变量

我们控制了人口统计学的以及脑梗死、心血管疾病相关的血管危险因素。体重指数 (BMI) 以体重 (kg) / 身高 (m²) 计算。记录房颤病史、抗凝药物和抗血小板药物的使用。吸烟由自身报告, 归类为吸烟者 (现今或过去 12 个月有吸烟) 和非吸烟者。测定空腹高密度脂蛋白胆固醇 (HDL-C) 和低密度脂蛋白胆固醇 (LDL-C)。高血压定义为收缩压 >140 mmHg, 舒张压 >90 mmHg, 或有服用抗高血压药物史。糖尿病史基于自身报告, 空腹血糖浓度 >7 mmol/L 或有服用口服降糖药或使用胰岛素素。冠心病史定义为有明确医疗记录的冠心病病史或有冠状动脉旁路手术史。

统计分析

用 logistic 回归比较了有或没有脑梗死个体的超声心动图特点作为一个连续变量并用其四分位数做 logistic 回归分析。LVM 和左房容积用体表面积标化。
析 E/A 比率和脑梗死的关系。因为 E/A 比率与死亡率之间有 U 型关系 (E/A 比率 <0.75 或 >1.5 死亡率增加 [19-20])，E/A 比率资料被分成四分位数；这些四分位数的阈值为 0.74, 0.88 和 1.03。在调整性别和年龄因素后，用 logistic 回归对分类变量、直线回归对连续变量，对 E/A 比率四分位数的这些协变量进行检验。

E/A 四分位数之间的所有脑梗死 OR 值的变化用 logistic 回归检验。然后，最高和最低四分位数两组和中间两组四分位数进行比较。因为最高四分位数包括 130 个 E/A 比值 <1.5( 正常范围为 0.75-1.5) 的个体 [20]，用其他三组四分位数组合后与最低四分位数 (E/A 比值 <0.75) 进行了敏感度分析。最后，最低和最高 E/A 比值 (<0.75 和 >1.5) 与报告的正常 E/A 比值范围 (0.75-1.5) 进行了比较 [20]。

本模型首先调整了性别和年龄 (模型 I)，然后调整了 BMI、当时的收缩压和舒张压、高血压、吸烟情况、HDL-C、糖尿病和过去有医疗记录的冠心病史 (模型 II)。由于高血压的高患病率 (79%)，血压测量被包括在模型中。最后，模型调整了 LV 射血分数 (模型 III)。在这些与脑梗死有显著关系的模型中，我们用自身报告的临床卒中事件作为独立变量，重新进行了分析。

资料用平均数 ( 标准差 ) 描述。OR 值和 95% 可信区间 (CI) 用 SAS 9.1/SAS Enterprise Guide (v4.1) 计算。所有分析均采用 $\alpha=0.05$ 作显著性检验标准。

### 结果

从 AGES-Reykjavik 群体中随机抽取的 954 个个体中，122 人因禁忌症、或检查不完整以致不能评价是否存在梗死灶、或因患者拒绝或残疾，而未行 MRI 检查。其他 140 人因为有多于两项超声心动图指标缺失而被除外。最后，总研究人数 692 人。与入选的个体比较，被排除的个体年龄明显较大 (77 [6] vs 76 [6]; $P<0.001$)，其中男性较多 (58% vs 53%; $P=0.001$)，患脑梗死的可能性更大 (41% vs 28%; $P=0.01$)。

研究样本中，193 (28%) 人有脑梗死的 MRI 证据，但只有 42 (6%) 人描述临床卒中史。调整年龄和性别后，有脑梗死的个体年龄明显较大 (77 [6] vs 76 [6]; $P<0.001$)，其中男性较多 (58% vs 53%; $P=0.001$)，患脑梗死的可能性更大 (41% vs 28%; $P=0.01$)。

表 1 显示了脑梗死与超声心动图参数的关系。模型 I 中，调整了年龄和性别后，患脑梗死的参与者其 LVM 和左心房容积指标明显增高，但无论作为连续变量还是四分位数 (四分位数结果未显示) 分析，脑梗死与主动脉根部直径、左心室射血分数或侧瓣的 E/E' 比值无明显相关。二尖瓣环状钙化的出现与脑梗死无明显相关。在模型 II，调整了年龄、性别和血管危险因素后，LVM 和左房容积提高 10% 后，OR 值分别为 1.08(95% CI, 1.00-1.16) 和 1.06(95% CI, 1.00-1.12)。当有房颤病史的个体被排除后，LVM 和左房容积仍然显示有意义 ( 前者 OR, 1.01; 95% CI, 1.00-1.02; 后者 OR, 1.01; 95% CI, 1.00-1.39)。

### 表 1 超声心动图参数与脑梗死的关系

<table>
<thead>
<tr>
<th>超声心动图参数</th>
<th>否平均值 ( 标准差 )</th>
<th>是平均值 ( 标准差 )</th>
<th>OR(95% CI)*</th>
<th>OR(95% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVM，gm/m²</td>
<td>87.2 (21.3)</td>
<td>97.6 (29.6)</td>
<td>1.01 (1.00–1.02)</td>
<td>1.01 (1.00–1.02)</td>
</tr>
<tr>
<td>左房容积，ml/m²</td>
<td>31.2 (8.82)</td>
<td>35.4 (12.2)</td>
<td>1.03 (1.01–1.05)</td>
<td>1.02 (1.00–1.04)</td>
</tr>
<tr>
<td>主动脉根部直径，cm</td>
<td>3.16 (0.45)</td>
<td>3.26 (0.48)</td>
<td>1.12 (0.72–1.75)</td>
<td>1.23 (0.78–1.95)</td>
</tr>
<tr>
<td>二尖瓣环状钙化，%(n)</td>
<td>30.9 (149)</td>
<td>34.2 (64)</td>
<td>0.93 (0.64–1.37)</td>
<td>0.98 (0.66–1.45)</td>
</tr>
<tr>
<td>E/E' 侧瓣</td>
<td>7.61 (2.26)</td>
<td>7.71 (3.15)</td>
<td>0.99 (0.92–1.07)</td>
<td>0.97 (0.89–1.05)</td>
</tr>
<tr>
<td>E/A 比值</td>
<td>0.95 (0.31)</td>
<td>0.90 (0.33)</td>
<td>0.63 (0.35–1.14)</td>
<td>0.61 (0.33–1.11)</td>
</tr>
<tr>
<td>LV 射血分数，%</td>
<td>62.8 (5.8)</td>
<td>61.4 (8.8)</td>
<td>0.99 (0.96–1.01)</td>
<td>0.99 (0.96–1.02)</td>
</tr>
</tbody>
</table>

* 模型 I：对年龄和性别进行校正后的 logistic 回归分析。
† 模型 II：对年龄和性别、现今及近来有吸烟者、BMI、冠心病史、糖尿病、高血压、HDL-C、现今的收缩压和舒张压进行校正后的 logistic 回归分析。
E/A 指舒张期早期 (E) 和晚期 (A) 经二尖瓣多普勒血流速率；E/E' 指舒张期早期 (E) 经二尖瓣多普勒血流速率和舒张期早期 (E') 组织多普勒二尖瓣环状速度；LV 指左心室。
比值与脑梗死的出现与否无明显的相关性 (前者 OR, 0.90 [0.33] vs 0.95 [0.31]; 二次方程结果未显示)。如方法中所述, 当 E/A 比值在模型中被划分四分位数时, 增加的 E/A 比值四分位数与年龄较小、糖尿病的患病率减少、低舒张压有相关性 (表 2)。除了血糖值外, 第二和第三四分位数组无明显差异 (5.92 [1.43] vs 5.63 [0.71]; P=0.01)。

E/A 比值的四分位数与脑梗死之间有一个综合的关系 (P=0.04)。在模型 I、II、III 中, 与 E/A 比值中间的两组四分位数比较, 最低四分位数 (<0.75) 与脑梗死显示了明显的关系 (OR, 1.82; 95% CI, 1.16-2.86; 表 3)。

当与其他三组四分位数比较时, E/A 比值 (<0.75) 同样也与脑梗死有明显的关系 (OR, 1.98; 95% CI, 1.35-2.90; 模型 III; 补充的表格可在 http://stroke.ahajournals.org 查阅)

表 2  E/A 比值四分位数基础上的研究样本特征

<table>
<thead>
<tr>
<th>E/A 四分位数</th>
<th>第一四分位数</th>
<th>第二四分位数</th>
<th>第三四分位数</th>
<th>第四四分位数</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=162</td>
<td>N=163</td>
<td>N=163</td>
<td>N=163</td>
<td></td>
<td></td>
</tr>
<tr>
<td>年龄,岁</td>
<td>76.5 (5.7)</td>
<td>75.5 (5.5)</td>
<td>74.5 (5.6)</td>
<td>75.5 (5.5)</td>
<td></td>
</tr>
<tr>
<td>男性, % (n)</td>
<td>28.1 (73)</td>
<td>21.5 (56)</td>
<td>27.3 (71)</td>
<td>23.1 (60)</td>
<td>0.14</td>
</tr>
<tr>
<td>身高,cm</td>
<td>166.4 (9.8)</td>
<td>166.3 (8.9)</td>
<td>167.5 (10.2)</td>
<td>167.4 (9.4)</td>
<td>0.04</td>
</tr>
<tr>
<td>体重,kg</td>
<td>76.0 (14.8)</td>
<td>75.9 (13.5)</td>
<td>76.3 (13.8)</td>
<td>73.7 (14.8)</td>
<td>0.35</td>
</tr>
<tr>
<td>BMI,kg/m²</td>
<td>27.4 (4.5)</td>
<td>27.4 (4.0)</td>
<td>27.2 (4.3)</td>
<td>26.3 (4.8)</td>
<td>0.06</td>
</tr>
<tr>
<td>目前或近期吸烟,% (n)</td>
<td>30.1 (104)</td>
<td>23.4 (81)</td>
<td>22.3 (77)</td>
<td>24.3 (84)</td>
<td>0.06</td>
</tr>
<tr>
<td>冠脉病史,% (n)</td>
<td>28.0 (28)</td>
<td>19.0 (19)</td>
<td>21.0 (21)</td>
<td>32.0 (32)</td>
<td>0.15</td>
</tr>
<tr>
<td>高血压史,% (n)</td>
<td>26.2 (135)</td>
<td>24.3 (125)</td>
<td>23.3 (120)</td>
<td>26.2 (135)</td>
<td>0.18</td>
</tr>
<tr>
<td>糖尿病,% (n)</td>
<td>26.6 (30)</td>
<td>28.0 (23)</td>
<td>14.6 (12)</td>
<td>20.7 (17)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

表 3  E/A 比值四分位数间髓与脑梗死的关系

<table>
<thead>
<tr>
<th>E/A 比值四分位数 *</th>
<th>否</th>
<th>是</th>
<th>OR(95% CI)†</th>
<th>OR(95% CI)‡</th>
<th>OR(95% CI)§</th>
</tr>
</thead>
<tbody>
<tr>
<td>最低 (第一)</td>
<td>21.2 (102)</td>
<td>35.3 (60)</td>
<td>1.87 (1.22-2.87)</td>
<td>1.84 (1.17-2.88)</td>
<td>1.82 (1.16-2.86)</td>
</tr>
<tr>
<td>第二</td>
<td>26.6 (128)</td>
<td>20.6 (35)</td>
<td>1 (对照)</td>
<td>1 (对照)</td>
<td>1 (对照)</td>
</tr>
<tr>
<td>第三</td>
<td>26.4 (127)</td>
<td>21.2 (38)</td>
<td>1 (对照)</td>
<td>1 (对照)</td>
<td>1 (对照)</td>
</tr>
<tr>
<td>最高 (第四)</td>
<td>25.8 (124)</td>
<td>22.9 (39)</td>
<td>1.11 (0.70-1.76)</td>
<td>1.02 (0.63-1.66)</td>
<td>1.03 (0.63-1.67)</td>
</tr>
</tbody>
</table>

* 四分位数间隔: 0.74、0.88、1.03。
† 模型 I: 校正年龄和性别后的 logistic 回归分析。
‡ 模型 II: 校正年龄、性别、近来或现今吸烟者、BMI、冠状动脉病史、糖尿病、高血压、高密度脂蛋白胆固醇、现今的收缩压和舒张压后的 logistic 回归分析。
§ 模型 III: 模型 II+ 左心室射血分数。

E/A 指舒张期早期和晚期的经二尖瓣多普勒血流速度比值。
当和正常范围的 E/A 比值（0.75-1.5）比较时，E/A 比值 <0.75 在所有模型中始终有显著意义（OR, 2.03; 95% CI, 1.37-2.99；模型 III；补充表格中）。

对于自身报告有临床卒中的亚组（n=42），E/A 比值的最低四分位数有一个相似的点估计值，但在所有模型中都不显著（OR, 1.97; 95% CI, 0.95-4.08; 模型 I）。在未调整和调整的模型（表 3），或所有模型中 E/A 比值的正常范围中，E/A 最高四分位数与中间两组四分位数无明显差异（OR, 1.51; 95% CI, 0.68-3.35; 模型 I；补充表格）。有房颤史的个体被排除后，结果未见改变。

讨论

在本横断面研究中，低 E/A 比值（<0.75）和脑梗死之间有强大的关联，不依赖于年龄、性别、血管危险因子和左心室射血分数。虽然 LVM 和左房容量这两个与慢性高血压有显著关联的指标，与脑梗死有关联，并且不依赖于年龄和性别，当模型调整了血管危险因子后，这个关联变得更为明显。利用更先进的仪器，这些结果确定了一些过去 M 型超声基础上的临床卒中预测指标，包括左心房大小与 LVM。然而，一些指标包括主动脉根部大小和二尖瓣环状钙化厚度，由二维点对点指标确定时，这些新结果似乎更加合理但需要将来的二维或三维评估来证实。另一个可能性是，在这个老年群体中，血管危险因子的高患病率掩盖了一些主动脉根部直径或二尖瓣环状钙化与脑梗死的独立关系。

很多早期用 M 型超声资料和卒中诊断的研究是建立在有或无 CT 或 MRI 脑部成像的临床发现上的 [4,5,21–23]。然而，在黑人中，LVM 与 MRI 检测的脑梗死相关，其临床和亚临床的总患病率比单纯的临床卒中更高（20% vs 3%），而这些发现和我们的结论是一致的 [9]。过去的研究已经表明，左心室早晚充盈速率的异常（如，低或高 E/A 比值）与全因死亡率增高相关 [20]。在对美洲印第安人的 Strong Heart 研究中，低 E/A 比值（<0.6）与更高的全因死亡率和心源性死亡率相关，但调整了协变量后它并非是一个独立的预测指标。Strong Heart 研究同时表明，E/A>1.5 的限制充盈患者中，年轻个体中有更高的死亡率（57 [7] 岁）。在 AGES-Reykjavik 样本中，只有 33 个个体的 E/A 值>1.5，因此这些参数是否有更高的价值尚不能下定论。这也同时解释了 E/A 比值与脑梗死二次方关系的缺失。

E/A 比值提示早晚心室舒张期速率。除了描述舒张晚期左心室灌注的局部变化外，低 E/A 比值可反映心血管系统的一般变化，包括层流速变化和一系列关于心血管聚集和上皮细胞功能的不良反应 [20]。这些以高氧化应激、促炎症反应增加和高凝状态为特征的适应不良的血管变化，可造成上皮细胞功能不良 [25,26]。然而，E/A 比值和卒中关系的根本机制可能是多因素的。

E/E’，这个左心室被动硬度 [27] 的预测值，在有房颤的个体中与缺血性卒中有关，但在 AGES-Reykjavik 中未显示有关联。但是，这个早期研究报告了更大范围和更高的 E/E’ 平均值 [28]，其较 AGES-Reykjavik 更硬化的心室可能是导致差异的原因。

研究的局限性

本研究有一些局限性。样本为 Reykjavik 研究开始 25 年后仍然存活的个体，因此致命性疾病因子的相关性可能被低估了。这个局限性可解释为什么 LVM 和左房容量与脑梗死的相关性并不高。同样地，回忆偏倚会影响协变量的特征，但是这些影响在研究组中可平均分布。另一个局限性是，与大部分超声心动图与卒中关系的既往研究比较，交叉横断面设计并不能进行风险的确定、预测指标和事件后果的确定。本研究样本量不够大，而且其意义可能已被因没有足够超声指标而被排除的个体所减小。

结论

本试验证明，即使在老年个体中，LVM、左心房容积和低 E/A 比值都与 MRI 检测的脑梗死有关。然而，低 E/A 比值和脑梗死的关系表现出独立于并发的心血管危险因素，例如高血压。与既往心脏形态学与卒中的关系相比，本研究有新发现，即舒张期功能与卒中的关系。E/A 比值这个简单的临床与亚临床脑梗死因子在老年患者中可能是非常有意义的，因为 E/A 比值低的认知功能障碍和痴呆患者可能提示脑缺血 [12]。

参考文献


