By definition, symptoms of a transient ischemic attack (TIA) subside completely within 24 hours. Imaging studies show signs of persistent ischemic tissue damage in a substantial amount of patients with TIA. Cerebral infarction can cause permanent cognitive impairment. Whether permanent cognitive impairment occurs after TIA is unclear, as is its profile.

Methods—Patients with TIA aged 45 to 65 years without prior stroke or dementia underwent comprehensive neuropsychological testing within 3 months. Z scores per cognitive domain were obtained, based on the mean of a control group within the same age range. Cognitive impairment was defined as a domain $z$ score $<-1.65$. Patients underwent either computed tomography or MRI brain imaging.

Results—One hundred seven patients with TIA (63% women, mean age, 56.6 years) were included and compared with 81 controls (56% women, mean age, 52.9 years). Patients performed worse on all cognitive domains except episodic memory. Working memory (25%), attention (22%), and information processing speed (16%) were most frequently impaired and more often than in the control group (age- and sex-adjusted odds ratios, respectively, 22.5 [95% confidence interval, 2.9–174.3], 6.8 [1.9–24.3], 7.1 [1.5–32.5]). More than 35% of patients with TIA had impairment of $\geq 1$ cognitive domain. Presence of silent brain infarcts was related to worse executive functioning but did not explain the whole relationship between TIA and cognitive impairment.

Conclusions—More than a third of patients with TIA have impairment of $\geq 1$ cognitive domain within 3 months after their TIA. The affected domains fit in the vascular cognitive impairment profile. (Stroke. 2014;45:00-00.)
neuropsychological assessment was performed within 3 months after the qualifying event. The number of available slots for these assessments was restricted. However, no selection for neuropsychological assessments was used.

TIA was defined as a sudden onset focal neurological deficit of vascular origin with complete resolution of focal symptoms within 24 hours. Patients with prior stroke or dementia were excluded, whereas those with prior TIA were not. In addition, patients with incident stroke or carotid endarterectomy between TIA and cognitive testing were excluded as were those with a Mini-Mental State Examination score <24 because this was considered indicative for possible pre-existing dementia and would prevent a reliable cognitive assessment. All data were acquired as part of clinical care, and both storage and processing were conducted anonymously in agreement with the local ethical committee rules.

Control Group
Patients’ performances on all individual tests were compared with a control group, recruited among spouses, relatives, or social environment of patients attending our outpatient department. All participants were free from TIA, and the same exclusion criteria as for patients were applied. The stroke-free status was verified through a standardized, structured questionnaire. When a possible cerebrovascular event was reported, the general practitioner was contacted for additional information.

Only for the California Verbal Learning Test, performance was not compared with the control group but with a normative sample described in the test manual (n=164, age, 45–64 years). None of the subjects in the normative sample had a history of psychiatric or neurological disease.

Clinical Characteristics
Hypertension was considered present when (1) systolic blood pressure was >140 mmHg and diastolic blood pressure was >90 mmHg at both time of presentation and at 90-day follow-up, (2) antihypertensive medication was used, or (3) a previous diagnosis by a physician was recorded. Hypercholesterolemia was considered present when (1) fasting total cholesterol level was >6.5 mmol/L, (2) lipid-lowering drugs were used, or (3) a physician had previously made the diagnosis. Diabetes mellitus was defined as (1) the use of antidiabetic medication or (2) a previous diagnosis by a physician. Smoking status was determined current when a patient smoked or had stopped within the past 6 months and former when smoking was stopped earlier. Any prior myocardial infarction was noted, and the body mass index was calculated.

Brain Imaging
Brain imaging, either computed tomography or MRI, performed within 3 weeks after the qualifying event was evaluated for the presence and severity of age-related white matter changes (ARWMC) using a semiquantitative scale, as well as the presence of silent brain infarct (SBI). SBI was defined as an infarct on computed tomography or MRI at a location that did not correspond with the symptoms of the qualifying or any previous TIA and was classified as lacunar or nonlacunar. Two experienced raters (F.G.v.R. and E.J.v.D.) separately performed assessment of brain imaging. In case of disagreement, a consensus meeting was held.

Neuropsychological Assessment
A trained examiner administered the neuropsychological tests in a quiet, well-lit room and under standard circumstances. Executive functioning was assessed with a verbal fluency task (naming as many animals as possible within 60 seconds; response generation) and the interference score of the Abbreviated Stroop Color Word Test (response inhibition). Information processing speed was tested with cards I and II of the Abbreviated Stroop Color Word Test and the Symbol-Digit Modalities Test. The Paper and Pencil Memory Scanning Test (4 subtasks) was used to measure working memory, and attention was evaluated by the Verbal Series Attention Test.

Finally, verbal episodic memory was tested with the Dutch version of the California Verbal Learning Test, using both the total correct answers of 5 immediate recall trials and the difference between trial 5 and long-term recall (consolidation). For tests requiring both speed and precision, a speed accuracy trade-off score was calculated by dividing the percentage of correct answers by the time taken to complete the test. This applied to the Verbal Series Attention Test, cards I and II of the Stroop Color Word Test, and all subtasks of the Paper and Pencil Memory Scanning Test. The Stroop interference score was computed by dividing the speed accuracy trade-off scores of card III by the mean of the speed accuracy trade-off scores of cards I and II.

Subjective Cognitive Failures
In addition, a 15-item semistructured interview based on the Cognitive Failures Questionnaire was administered to identify subjective cognitive failures (SCF) experienced the month before. Responses were added to provide a sum score with a maximum of 25. SCF reported in remembering, word finding, planning, concentration, and slowness in thought were given a higher weight in the sum scores (range 0–3: none, mild, moderate, severe) than the other items (0–1). If ≥1 moderate problem (score ≥2) on an item with a score range of 0 to 3 or a score of 1 on a dichotomous item was reported, SCF was considered present.

Other Measurements
Age, sex, and level of education were recorded. The presence of depressive symptoms was defined as a Hospital Anxiety and Depression Scale depression subscale score ≥8.

Statistical Analysis
All analyses were done with IBM SPSS Statistics version 20.0 (IBM Corp, Armonk, NY). Differences in characteristics between patients and controls were compared using Student t test, Pearson χ², and age- and sex-adjusted ANCOVA when appropriate. Bonferroni correction for multiple testing was applied with α set at 0.01.

Individual z scores were computed for each neuropsychological test using the mean and SD of the control group, and domain z scores were calculated by averaging z scores of individual tests. Per cognitive domain, a z score <−1.65 of the control group was used as a cutoff to determine domain-specific impairment (ie, corresponding to a performance below the lower fifth percentile). Domain-specific age- and sex-adjusted odds ratios (OR) of cognitive impairment after TIA were obtained by logistic regression. No OR of episodic memory impairment was calculated because California Verbal Learning Test results were not compared with the same control group.

In case of missing neuropsychological test results (maximum 12.2%), the domain score was based on the remaining tests, or if no tests were performed within a cognitive domain, the domain z score was not used in further analyses.

Results
Between September 2004 and December 2010, 246 patients with TIA aged 45 to 65 years were registered, of whom 114 underwent neuropsychological testing within 3 months after the qualifying event (mean [SD], 56 [14.7] days; range, 26–91 days). Patients with and without neuropsychological assessment did not differ on age and sex (Student t test and Pearson χ²; P=0.92 and 0.43, respectively). Subsequently, 5 patients were excluded because of a history of stroke and 2 because of a Mini-Mental State Examination score <24. Characteristics of included patients (n=107) are summarized in Table 1. The control group included 81 individuals. No differences were
present between patients and controls for sex, low level of education, Hospital Anxiety and Depression Scale depression subscale; IQR, interquartile range; N/A, not applicable; and TIA, transient ischemic attack.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients With TIA</th>
<th>Controls</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td>67 (62.6)</td>
<td>45 (55.6)</td>
<td>0.41</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>56.6 (6.3)</td>
<td>52.9 (6.2)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low level of education</td>
<td>28 (26.2)</td>
<td>17 (21.0)</td>
<td>0.52</td>
</tr>
<tr>
<td>HADS-D, mean (SD)</td>
<td>3.2 (3.9)</td>
<td>2.6 (2.7)</td>
<td>0.22</td>
</tr>
<tr>
<td>HADS-D score ≥8</td>
<td>13 (12.1)</td>
<td>6 (7.4)</td>
<td>0.35</td>
</tr>
<tr>
<td>Body mass index, mean (SD)</td>
<td>26.8 (4.0)</td>
<td>27.4 (4.9)</td>
<td>0.36</td>
</tr>
<tr>
<td>Hypertension</td>
<td>52 (48.6)</td>
<td>27 (33.3)</td>
<td>0.05</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>31 (29.0)</td>
<td>15 (18.5)</td>
<td>0.14</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5 (4.7)</td>
<td>1 (1.2)</td>
<td>0.36</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>8 (7.5)</td>
<td>1 (1.2)</td>
<td>0.10</td>
</tr>
<tr>
<td>Smoking status</td>
<td>0.32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>19 (17.8)</td>
<td>22 (27.2)</td>
<td></td>
</tr>
<tr>
<td>Former</td>
<td>53 (49.5)</td>
<td>35 (43.2)</td>
<td></td>
</tr>
<tr>
<td>ARWMC score, median (IQR)</td>
<td>1 (3)</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Silent brain infarct</td>
<td>18 (18.2)</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>17 (15.9)</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Lacunar</td>
<td>14 (13.1)</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>DWI lesion only MRI</td>
<td>16 (27.1)</td>
<td>N/A</td>
<td></td>
</tr>
</tbody>
</table>

Numbers are n (%) unless stated otherwise. ARWMC indicates age-related white matter changes; DWI, diffusion-weighted imaging; HADS-D, Hospital Anxiety and Depression Scale, depression subscale; IQR, interquartile range; N/A, not applicable; and TIA, transient ischemic attack.

*Difference using Student t test and Pearson χ² (continuity correction) when appropriate.

Table 3. Odds Ratios for Cognitive Impairment Within 3 Months After TIA Compared With Controls Without TIA (n=81)

<table>
<thead>
<tr>
<th>Cognitive Domain</th>
<th>All Patients With TIA (n=107)</th>
<th>Patients With TIA Without SBI Only (n=89)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive Impairment, OR (95% CI)*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Executive function</td>
<td>3.5 (0.7–16.7)</td>
<td>1.5 (0.3–8.7)</td>
</tr>
<tr>
<td>Information processing speed</td>
<td>7.1 (1.5–32.5)</td>
<td>4.8 (1.0–23.4)</td>
</tr>
<tr>
<td>Working memory</td>
<td>22.5 (2.9–174.3)</td>
<td>16.2 (2.0–128.7)</td>
</tr>
<tr>
<td>Attention</td>
<td>6.8 (1.9–24.3)</td>
<td>5.7 (1.5–20.9)</td>
</tr>
<tr>
<td>≥1 cognitive domain</td>
<td>5.9 (2.4–14.5)</td>
<td>5.4 (2.1–13.3)</td>
</tr>
</tbody>
</table>

Cognitive impairment defined as domain z score <−1.65. CI indicates confidence interval; OR, odds ratio; SBI, silent brain infarct; and TIA, transient ischemic attack.

*Age- and sex-adjusted logistic regression; †P<0.01; ‡P<0.001.
Discussion

The main findings of our study are that (1) more than a third of patients aged 45 to 65 years who had a TIA in the previous 3 months have impairment of function in ≥1 cognitive domain, (2) working memory, attention, and information processing speed are the most affected cognitive domains, whereas global memory functions remain relatively intact, (3) this cognitive impairment is only partly related to cerebrovascular damage on conventional neuroimaging, and (4) subjective cognitive complaints are not more frequently reported by patients with TIA than by healthy individuals.

This study was not without limitations. Given the cross-sectional design of the study without information on cognitive performance before the qualifying event, neither definite causal relations between cognitive function and TIA nor its time course could be established. Not all patients with TIA seen during the study period underwent cognitive assessment because of restricted availability of slots. To prevent selection bias, patients were assigned to cognitive assessment irrespective of clinical data. Furthermore, California Verbal Learning Test was not performed in the control group, and patients’ results for this test were compared with normative data derived from a different age-adjusted reference group published in the test manual. Although this prevented calculation of ORs for overall cognitive impairment, the composition of the cognitive profile of patients with TIA separately could be described. In addition, not all patients completed the neuropsychological test battery. By computing a domain-specific compound z score based on less than all associated tests when necessary and by not using missing values in further analyses, we might have reduced statistical power of our results. Despite swift analysis after referral, patient delay caused initial brain imaging to take place ≤3 weeks after the qualifying event, which could have influenced diffusion-weighted imaging lesion prevalence. Also, controls were on average slightly younger than the patients. However, they were all from the same relatively narrow age range, and although statistically significant, the mean difference was small and analyses were adjusted for differences in age. Therefore, we think that this potential factor has been adequately accounted for and cannot entirely explain our findings. Finally, to reduce the influence of concomitant cognitive disorders, we excluded older patients and those with low Mini-Mental State Examination performance. This limits the generalizability of our results to the whole TIA population and might have excluded representatives of the more severe spectrum of cognitive impairment after TIA.

Previous data on cognitive function after TIA are scarce and heterogeneous. Patent characteristics, definition of cognitive impairment, and delay from TIA to assessment of cognition differ widely between studies. Furthermore, cognitive assessment is mostly limited to screening tools such as Mini-Mental State Examination and Montreal Cognitive Assessment, which are not sensitive to mild cognitive deficits after stroke and do not assess specific cognitive domains. Previously reported prevalence of cognitive impairment after TIA varies from 30% to 57%. Compared with our study, these studies included substantially older patients, performed cognitive assessment much later after TIA, and did not exclude patients with previous stroke. In contrast, we aimed to minimize the effect of possible concomitant causes of cognitive impairment by performing cognitive testing within 3 months after the qualifying event in patients aged <65 years and excluding those with a history of stroke. Despite our rigorous measures to minimize the effects of neurodegenerative or previous vascular cognitive disorders, we found a prevalence of 38% of cognitive impairment in a relative young cohort of patients with a recent TIA.

The cognitive profile after TIA showed prominent impairment in the domains of working memory, attention, and information processing speed, whereas global memory functions remained within normal ranges. This nonamnestic cognitive impairment is compatible with the vascular cognitive impairment profile and mainly driven by subcortical brain damage disrupting subcortical–frontal connections. Only a few studies have previously described the cognitive profile of patients with TIA and found prominent deficits in executive functioning, visuconstruction, and attention. However, one of these studies included only patients with internal carotid artery occlusion and did not exclude patients with prior stroke, whereas the others performed Montreal Cognitive Assessment instead of a more comprehensive neuropsychological evaluation.

Presence of SBI was related only to worse executive functioning, whereas ARWMC were not related to any cognitive impairment after TIA. Because of limited numbers, we were unable to assess the relationship of different types of SBI with cognitive function. The prevalence of SBI and the severity of ARWMC were low compared with population-based studies, probably related to our relatively young study population. Both SBI and ARWMC are markers for cerebral small vessel disease, indicating that in our study population the role of small vessel disease in cognitive impairment after TIA seems limited. This is further strengthened by the robust association of TIA with cognitive impairment after excluding patients with SBI. The influence of concomitant neurodegeneration on cognitive function was minimized through a rigorous age restriction. This suggests a role for TIA itself in cognitive impairment afterward, the mechanism of which remains to be elucidated. Transient ischemia might lead to microstructural damage and loss of white matter structural integrity, giving rise to subcortical–frontal disconnection in a similar fashion as the vascular cognitive impairment construct. Verification of this potential pathway was not possible in our study because imaging modalities assessing the structural integrity of white matter were not performed.

In contrast to the high prevalence of objective cognitive dysfunction, patients with TIA did not report more SCF than controls. The prevalence of SCF among patients with TIA is remarkably lower than in elderly persons with white matter lesions and is comparable to the one study that previously reported cognitive complaints in patients with TIA. However, those results were not separately reported for TIA and minor stroke patients, and assessment of SCF was limited. The discrepancy between prevalence of objective and subjective cognitive dysfunction in TIA might mean that only relatively minor difficulties in everyday life are perceived.
because patients with TIA in our study were relatively young, a large proportion would still be working and be socially active. Even minor cognitive decline might, therefore, have impact.

The causes of cognitive impairment after TIA remain unknown. Future studies should include advanced brain imaging techniques to identify microstructural and functional cerebrovascular damage and perform longitudinal assessment of cognitive function after TIA to observe whether cognitive impairment is transient, stationary, or progresses over time. Nevertheless, our results show the extent of cognitive impairment after TIA in relatively young adults and warrant the need for more clinical awareness of this problem.

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This work was supported by a grant received by Dr van Dijk; Dutch Brain Foundation, grant number F2009(1)-16.

Disclosures
None.

References
Persistent Cognitive Impairment After Transient Ischemic Attack
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SUPPLEMENTAL MATERIAL

Persistent cognitive impairment after transient ischemic attack

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Short title: Persistent cognitive impairment after TIA

Key words: Transient ischemic attack, cognition, vascular cognitive impairment

Supplementary Table I. Associations of silent brain infarct and age-related white matter changes with cognitive impairment within three months after TIA.

<table>
<thead>
<tr>
<th>Cognitive domain</th>
<th>SBI (yes/no)</th>
<th>ARWMC (per 1 point increase)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Executive function</td>
<td>10.7 (2.5-44.8)†</td>
<td>1.0 (0.8-1.3)</td>
</tr>
<tr>
<td>Information processing speed</td>
<td>4.8 (1.3-17.5)</td>
<td>0.7 (0.5-1.0)</td>
</tr>
<tr>
<td>Working memory</td>
<td>3.5 (1.0-11.9)</td>
<td>0.7 (0.5-1.0)</td>
</tr>
<tr>
<td>Attention</td>
<td>3.4 (1.0-11.4)</td>
<td>0.8 (0.6-1.0)</td>
</tr>
<tr>
<td>≥1 cognitive domain</td>
<td>2.4 (0.8-7.2)</td>
<td>0.8 (0.7-1.0)</td>
</tr>
</tbody>
</table>

Cognitive impairment defined as z-score < -1.65.

*age- and sex-adjusted logistic regression; †p-value < 0.01.

OR Indicates odds ratio; CI, confidence interval; SBI, silent brain infarct; ARWMC, age-related white matter changes.

References:


TI A 后持续性认知功能障碍
Persistent Cognitive Impairment After Transient Ischemic Attack

Frank G. van Rooij, MD, Pauline Schapsteentruit, MSc, Noortje A.M. Maaijwee, MD,
Dirk A.H.J. van Duijnhoven, MSc, Frank E. de Leeuw, PhD, Roy P.C. Kessels, PhD

Ewoud J. van Dijk, PhD

1. 研究假设 TIA 后患者的认知能力下降，并有血管性认知功能障碍特征。
2. 为减轻老年化认知缺损的偏移影响，研究对象限制在 45 到 65 岁之间。

本研究除外既往患有卒中或痴呆症的患者。另外，TIA 患者在认知测验中卒中发作或需要行颈动脉内膜切除术，则排除在外。所有数据都是临床治疗的一部分，数据存储和处理都是匿名进行，符合当地伦理委员会的标准。

方法

1. 根据定义，短暂性脑缺血发作（TIA）指伴有局灶症状的短暂的脑血液循环受阻，症状在 24 小时内消失。
2. 本研究连续登记了 2004 到 2010 年，内梅亨医疗中心卒中单元及 TIA 门诊 45 到 65 岁 TIA 患者的情况。我们主要是记录那些在 TIA 发作后 3 个月内进行综合神经心理测试的患者。
3. 神经心理测试

- 根据定义，短暂性脑缺血发作（TIA）24 小时内症状消退。脑成像研究表明绝大部分 TIA 患者有持续缺血组织的损伤迹象。
- 脑成像

- 短暂性脑缺血发作患者(校正年龄和性别后差异非常显著)。
- 无症状脑梗死可能表现为执行能力受损，但工作记忆(25%)，注意力(22%)和信息处理速度(16%)时常受损，远高于对照组(年龄和性别校正后优势比分别为 22.5[95% 置信区间,2.9-174.3],6.8[1.9-24.3],7.1[1.5-32.5])。超过 30% 的短暂性缺血发作患者。

数据分析

- 对照组与患者组的区别通过 T 检验，非参数 χ2 检验以及适合
- 表 1 TIA 组及对照组的临床特征

<table>
<thead>
<tr>
<th>对照组</th>
<th>TIA 患者组</th>
</tr>
</thead>
<tbody>
<tr>
<td>女性</td>
<td>67(62.6)</td>
</tr>
<tr>
<td>教育水平</td>
<td>28(26.2)</td>
</tr>
<tr>
<td>年龄, 均数 (SD)</td>
<td>56.6(6.3)</td>
</tr>
<tr>
<td>女性</td>
<td>67(62.6)</td>
</tr>
<tr>
<td>教育水平</td>
<td>28(26.2)</td>
</tr>
<tr>
<td>年龄, 均数 (SD)</td>
<td>56.6(6.3)</td>
</tr>
<tr>
<td>女性</td>
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</tr>
<tr>
<td>年龄, 均数 (SD)</td>
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</tr>
<tr>
<td>女性</td>
<td>67(62.6)</td>
</tr>
<tr>
<td>教育水平</td>
<td>28(26.2)</td>
</tr>
<tr>
<td>年龄, 均数 (SD)</td>
<td>56.6(6.3)</td>
</tr>
</tbody>
</table>

所有认知领域中，主观认知障碍与低 z 值相关。短暂性脑缺血发作患者(校正年龄和性别后的协方差分析 ; P<0.01)
- 没有差别。 59% 的短暂性脑缺血发作患者主观认知障碍与对照组相同。

结论

1. 随着年龄和性别校正 AGE 值进行比较。运用 Bonferroni 多重测验校正 P 值到 0.01。
2. 本文的局限性包括研究设计为横断面研究，没有纳入特定认知领域的问题，这种认知障碍仅仅部分与脑血管损害有关。
3. 主要发现有: (1) 年龄为 45 至 65 岁短暂性脑缺血患者发作后 3 个月内有超过三分之一的患者存在 1 个以上认知领域障碍 ; (2) 工作记忆和注意力和信息处理速度是受损影响最显著的认知领域，而患者早期诊断。短暂性脑缺血发作患者主观认知障碍与低 z 值相关。短暂性脑缺血发作患者(校正年龄和性别后的协方差分析 ; P<0.01)。在预约背景中，我们仍不能完全解释 TIA 与认知功能障碍的所有关系。
急性脑出血治疗的血液动力学

背景与目的：最近，急性脑出血防治试验2 (INTERACT2) 中的研究显示了急性脑出血时降低血压可改善预后。然而，血压的降低程度和降低血压的时机尚未明确。

目的：本研究旨在分析急性脑出血患者降压治疗时机和血压降低程度与预后的关系。

方法：本研究为队列研究，纳入所有在脑出血后24小时内接受降压治疗的急性脑出血患者。将患者分为低血压组（血压<90mmHg）和正常血压组（血压≥90mmHg）。根据降压治疗开始的时间，将患者分为早期降压组（开始时间<1小时）和延迟降压组（开始时间≥1小时）。主要结局为3个月的神经功能恶化。

结果：共纳入344例患者，其中低血压组和正常血压组各172例，早期降压组和延迟降压组各172例。结果显示，低血压组和早期降压组的预后显著优于正常血压组和延迟降压组。

结论：本研究提示，急性脑出血患者应尽早开始降压治疗，并将血压降至正常水平，以改善预后。