# Short-term Prognosis After Emergency Department Diagnosis of TIA

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**Context** Management of patients with acute transient ischemic attack (TIA) varies widely, with some institutions admitting all patients and others proceeding with outpatient evaluations. Defining the short-term prognosis and risk factors for stroke after TIA may provide guidance in determining which patients need rapid evaluation.

**Objective** To determine the short-term risk of stroke and other adverse events after emergency department (ED) diagnosis of TIA.

**Design and Setting** Cohort study conducted from March 1997 through February 1998 in 16 hospitals in a health maintenance organization in northern California.

**Patients** A total of 1707 patients (mean age, 72 years) identified by ED physicians as having presented with TIA.

**Main Outcome Measures** Risk of stroke during the 90 days after index TIA; other events, including death, recurrent TIA, and hospitalization for cardiovascular events.

**Results** During the 90 days after index TIA, 180 patients (10.5%) returned to the ED with a stroke, 91 of which occurred in the first 2 days. Five factors were independently associated with stroke: age greater than 60 years (odds ratio [OR], 1.8; 95% confidence interval [CI], 1.1-2.7; $P=.01$), diabetes mellitus (OR, 2.0; 95% CI, 1.4-2.9; $P<.001$), symptom duration longer than 10 minutes (OR, 2.3; 95% CI, 1.3-4.2; $P=.005$), weakness (OR, 1.9; 95% CI, 1.4-2.6; $P<.001$), and speech impairment (OR, 1.5; 95% CI, 1.1-2.1; $P=.01$). Stroke or other adverse events occurred in 428 patients (25.1%) in the 90 days after the TIA and included 44 hospitalizations for cardiovascular events (2.6%), 45 deaths (2.6%), and 216 recurrent TIAs (12.7%).

**Conclusions** Our results indicate that the short-term risk of stroke and other adverse events among patients who present to an ED with a TIA is substantial. Characteristics of the patient and the TIA may be useful for identifying patients who may benefit from expeditious evaluation and treatment.

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TRANSIENT ISCHEMIC ATTACKS (TIAs) are common[1]: approximately 300 000 TIAs occur each year in the United States based on recent estimates of stroke incidence.[2,3] A recent survey suggested a much higher incidence, with 1 in 15 of those older than age 65 years reporting a history of TIA.[4] About 15% of patients experiencing stroke report a history of TIA.[5] Effective prevention of subsequent stroke in patients with TIAs could significantly reduce the overall stroke incidence.[6]

Evaluation and treatment of patients with stroke is generally rapid, in part because management of ongoing cerebral ischemia may reduce the final injury and because patients often have a disability that requires care.[7] The need for urgent intervention is less clear for patients with TIA, who have usually returned to their baseline level of function by the time they are evaluated. While some interventions are known to be effective after TIA, such as antiplatelet agents, anticoagulation for atrial fibrillation, and endarterectomy for symptomatic carotid artery stenosis,[8] it is not known whether more urgent therapy is justified. Hospitalization or rapid evaluation may be warranted only in patients with high short-term risk because urgent secondary prevention strategies are costly.[9-12]

Several studies have evaluated stroke risk after TIA,[13-24] but they have generally been small, have failed to examine the short-term risk separately, and have not identified which factors are associated with an increased risk of stroke. The most reliable data on short-term prognosis after TIA come from 2 population-based studies,[25,26] but their results have limited applicability to current clinical management because the studies were performed many years ago and included fewer than 200 cases each; 1 study did not include the risk of stroke in the first few days after the TIA.

It is often impossible to confirm a diagnosis of TIA since symptoms are transient and may have nonischemic etiologies such as seizure, syncope, and migraine. In fact, agreement between independent observers on TIA diagnosis is poor, even among neurologists.[27,28] Clinical decisions are based on the final diagnosis of the treating physician, who is often an emergency-department (ED) physician, internist, or fam-

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ily practitioner.[29] Since those patients given a diagnosis of TIA by the treating physician form the cohort currently recognized in practice, it is essential to study these patients regardless of whether the diagnosis is confirmed by a neurologist.

Lack of knowledge about the acute natural history of patients with TIA has led to variability in clinical practice.[30] Guidelines are generally vague about evaluation and treatment.[8,9] To determine the short-term risk of stroke and other adverse events after TIA and to identify risk factors, we performed a cohort study of patients diagnosed as having TIA in the EDs of hospitals belonging to a large health maintenance organization. We used the treating physicians' diagnoses of TIA to define the cohort so that results would be generalizable to institutions where neurologists do not routinely evaluate patients with TIA in the ED.

## METHODS

### Patients

Kaiser-Permanente Northern California is a health maintenance organization with 16 hospitals in the San Francisco Bay Area, Sacramento, and Santa Rosa, Calif. There are 2.9 million enrollees, with demographics similar to the regional population.[31] Beginning in March 1997, ED physicians at all 16 facilities were asked to select a primary diagnosis (including TIA) for all patient visits. These diagnoses were entered into a database.

Patients (n=1797) seen in the ED and diagnosed as having TIA from March 1997 through February 1998 were identified. Those who did not have ED records available (n=30), were not members of the health plan (n=27), had coded diagnoses other than TIA (n=25), or had a prior TIA treated in the ED during the study period (n=8) were excluded. Medical records were reviewed by trained analysts in conjunction with a neurologist. A neurologist blinded to follow-up events reviewed the medical records of all patients in whom the diagnosis of TIA was unclear based on physician documentation or analyst review. Definite TIA was defined based on World Health Organization criteria[32] as rapidly developed clinical signs of focal or global disturbance of cerebral function lasting fewer than 24 hours, with no apparent nonvascular cause. Analyses of definite TIA cases are reported separately from the primary analysis, which is based on the ED diagnosis.

Patient characteristics, medical history, TIA symptom details, medications, examination findings attributed to the TIA, and treatment plans were abstracted using predefined criteria. Emergency department physician notes were considered the primary source, supplemented with nursing notes when necessary. Subtypes of speech impairment and vision loss were not identified, given the difficulty of distinguishing dysarthria from aphasia and monocular blindness from a homonymous visual field deficit based on patient recollection. Heart rate and blood pressure were recorded from the initial ED measurement. A murmur was coded as absent if not mentioned in notation of cardiac auscultation. A bruit was coded as absent only if specifically noted in the record.

### Outcomes

Patients were followed up for 90 days after presentation. Strokes, TIAs, deaths, and hospitalizations for cardiovascular events were identified from computerized databases and review of medical records. Hospitalizations outside the system were recorded in a separate database, so it was possible to obtain very complete follow-up information.[33,34]

The primary outcome was stroke occurring within 90 days of TIA presentation. Stroke was defined as a rapidly developed focal or global disturbance of cerebral function, with no apparent nonvascular cause, lasting more than 24 hours or until death,[32] and distinguishable from the event leading to the initial TIA diagnosis. A final stroke diagnosis required independent confirmation by 2 neurologists, who also determined whether the stroke led to hospitalization or was disabling (defined as a Modified Rankin Score of $\geq$2).[35] There were initial disagreements in 2% of stroke diagnoses, 4% of hospitalization determinations, and 12% of disability assessments; all disagreements were resolved through discussion.

Diagnosis of recurrent TIA required confirmation by a reviewing neurologist and a written diagnosis in the medical record. Cardiovascular events requiring hospitalization included myocardial infarction, unstable angina, congestive heart failure, and ventricular arrhythmias. Deaths were identified from medical records, enrollment files, and the Social Security Death Index.

### Statistical Analysis

The risks of stroke, recurrent TIA, and cardiovascular events were determined as the percentage of patients with these events during the 90-day period after resolution of all TIA symptoms. For patients without documentation of symptom resolution, follow-up began at the time of ED discharge.

In univariate analysis, we identified clinical signs and symptoms that were significant risk factors for stroke using the Fisher exact test. Variables that were associated with stroke risk (at $P<.20$) were included in multivariable logistic regression models, and those no longer associated with risk (at $P>.10$) were removed in a stepwise fashion. To simplify the models, continuous variables were dichotomized at prespecified cutpoints (eg, diastolic blood pressure=90 mm Hg, systolic blood pressure=140 mm Hg). Because specific neurologic signs and symptoms were often correlated, neurologic abnormalities present either by history or on examination were combined (eg, weakness by history or on examination). Variables representing treatments initiated after ED evaluation were not included in the model since they were likely to be associated with perceived prognosis and could alter prognosis; these complex interactions would limit their interpretation. First-order interaction terms with combinations of all independent predictors were reintroduced into the multivariable model 1 at a time.

Kaplan-Meier life-table analysis was used to illustrate the timing of follow-up events. Since a stroke occurring after TIA but before evaluation would not have been included, resulting in an underestimation of stroke risk, we excluded person-time prior to evaluation in those patients whose symptoms resolved prior to ED arrival. Follow-up was terminated at death or endarterectomy. The protocol was approved by the institutional review board of Kaiser-Permanente Northern California. The risk of study participation was considered minimal, so informed consent was not required.

## RESULTS

Of the 1707 patients with a diagnosis of TIA, more than 99% (n=1697) arrived within 1 day of symptom onset. Mean age was 72 years and mean symptom duration was 207 minutes. The mean heart rate and blood pressure were 78/min and 161/83 mm Hg, respectively. Risk factors for vascular disease were common, and included diabetes mellitus, hypertension, and known history of vascular disease (TABLE 1). Symptoms were present on arrival in half of the patients (n=854).

Strokes occurred in 180 patients (10.5%) within 90 days of TIA presentation, 91 of which occurred during the first 2 days (FIGURE A). Strokes were fatal in 38 patients (21%), and disabling in another 115 (64%). Seventy-six percent of patients required hospitalization. Only 7% of strokes were not disabling and did not require hospitalization.

### Stroke Risk Factors

Age older than 60 years, diabetes mellitus, duration of the TIA, symptoms on arrival, and signs or symptoms of weakness, speech impairment, and gait disturbance were associated with increased risk of stroke in univariate analysis, while symptoms of numbness were associated with reduced risk (Table 1). Medications taken prior to evaluation did not influence prognosis.

A variety of treatments were initiated in the ED or at discharge (TABLE 2). Patients prescribed anticoagulation at discharge from the ED were more likely to have a stroke, but a perception of greater stroke risk may have prompted more aggressive therapy in these patients. When analysis was limited to the 918 patients who were not previously taking an anticoagulant or antiplatelet agent, those initiating an antiplatelet agent (n=775) tended to have lower

**Table 1.** Characteristics of Patients With TIA and Associated 90-Day Stroke Risk (N = 1707)*

| Variable | No. (%) | 90-Day Stroke Risk With Characteristic, % | | *P* Value† |
|---|---|---|---|---|
| | | Present | Absent | |
| Demographic characteristics | | | | |
| Age >60 y | 1325 (78) | 11 | 7 | .01 |
| Female sex | 899 (53) | 10 | 11 | .58 |
| Ethnicity | | | | |
| African American | 150 (10) | 13 | 11 | .41 |
| Asian American | 86 (6) | 15 | 11 | .11 |
| Hispanic | 72 (5) | 14 | 11 | .22 |
| White | 1226 (80) | 10 | 14 | .08 |
| Medical history | | | | |
| Diabetes | 332 (19) | 18 | 9 | <.001 |
| Hypertension | 988 (58) | 12 | 9 | .07 |
| Coronary artery disease | 414 (24) | 11 | 10 | .52 |
| Atrial fibrillation | 151 (9) | 13 | 10 | .27 |
| Known carotid artery disease | 97 (6) | 9 | 11 | .86 |
| Prior TIA | 452 (26) | 11 | 10 | .66 |
| Prior stroke | 385 (23) | 13 | 10 | .19 |
| Hypercholesterolemia | 250 (15) | 11 | 11 | .91 |
| Current cigarette smoking | 200 (14) | 13 | 11 | .46 |
| Medications at evaluation | | | | |
| Aspirin | 568 (33) | 11 | 10 | .50 |
| Ticlopidine | 59 (3) | 10 | 11 | >.99 |
| Warfarin | 153 (9) | 11 | 10 | .78 |
| TIA symptoms | | | | |
| Duration >10 min | 1441 (84) | 12 | 5 | .001 |
| Weakness | 784 (46) | 14 | 8 | <.001 |
| Numbness | 683 (40) | 8 | 12 | .02 |
| Confusion | 365 (21) | 13 | 10 | .18 |
| Change in vision | 222 (13) | 8 | 11 | .16 |
| Change in speech | 722 (42) | 14 | 8 | <.001 |
| Dizziness | 349 (20) | 8 | 11 | .14 |
| Vertigo | 54 (3) | 7 | 11 | .65 |
| Gait disturbance | 430 (25) | 14 | 9 | .01 |
| Symptoms on arrival | 854 (50) | 12 | 9 | .01 |
| Examination findings | | | | |
| Heart rate >80/min | 688 (40) | 11 | 10 | .33 |
| Systolic blood pressure ≥140 mm Hg | 1281 (75) | 11 | 9 | .14 |
| Diastolic blood pressure ≥90 mm Hg | 516 (30) | 11 | 10 | .44 |
| Murmur | 117 (7) | 13 | 10 | .43 |
| Bruit | 71 (8) | 9 | 10 | .84 |
| Weakness | 273 (16) | 15 | 10 | .007 |
| Numbness | 123 (7) | 7 | 11 | .17 |
| Gait abnormality | 83 (5) | 11 | 11 | .86 |
| Confusion | 54 (3) | 15 | 10 | .27 |
| Speech difficulty | 168 (10) | 16 | 10 | .02 |
| Aphasia | 65 (4) | 17 | 10 | .10 |
| Dysarthria | 100 (6) | 16 | 10 | .09 |

*TIA indicates transient ischemic attack.
†Calculated using the Fisher exact test.

stroke risk than those receiving no prophylactic medication (n=143) (9% vs 13%; *P*=.12). The same trend was apparent in evaluating any prophylactic therapy vs none (9% vs 13%; *P*=.17). In multivariable models adjusting for prognostic factors, neither of these variables was significant (new antiplatelet therapy vs none: odds ratio [OR], 0.63, 95% confidence interval [CI], 0.37-1.05, *P*=.10; any new prophylactic therapy vs none: OR, 0.66, 95% CI, 0.38-1.15, *P*=.14).

Five factors were independently associated with 90-day stroke risk (TABLE 3). A simple index (1 point for each risk factor) was useful in estimating risk, which varied from 0% in patients with no factors to 34% in those with all 5 (Figure B), though observations were sparse at the extremes (TABLE 4).

## Models Including First-Order Interactions

The only significant interaction term was one that represented speech impairment in patients with symptoms lasting more than 10 minutes (OR, 6.0; 95% CI, 1.9-6.5; *P*=.003). Symptom duration was no longer a significant predictor of stroke in that model, and speech impairment alone was associated with a lower risk of stroke (OR, 0.29; 95% CI, 0.09-0.95; *P*=.04). The magnitude of other predictors was unchanged in the model with the interaction term.

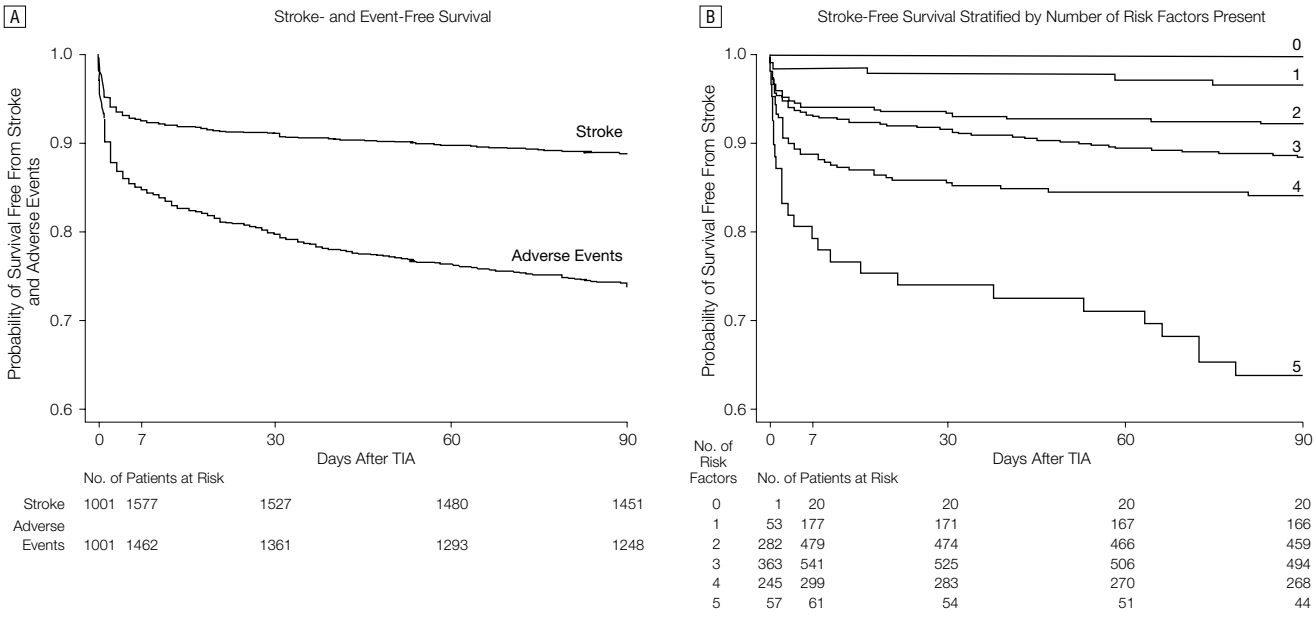
## Other Adverse Outcomes

Hospitalization for cardiovascular events occurred in 44 patients (2.6%) within 90 days of the TIA. Reasons for admission included congestive heart failure (n=22), myocardial infarction (n=13), ventricular arrhythmia (n=5), and unstable angina (n=4). Forty-five patients (2.6%) died within 90 days of the TIA; causes of death were strokes (n=20), cardiovascular events (n=9), infections (n=4), cancer (n=4), pancreatitis (n=1), and unknown (n=7). Recurrent TIA occurred in 216 patients (12.7%), and was associated with hospitalization in 106 patients (6.2%). An adverse event, including stroke, cardiovascular hospitalization, death, or recurrent TIA, occurred in 428 patients (25.1%) in the 90 days after the TIA. More than 50% of adverse events occurred within the first 4 days.

## Validation of the ED Diagnosis

The TIA diagnosis was thought improbable by a reviewing neurologist in 96 patients (5.6%), in whom symptoms were attributed to syncope (n=24), peripheral vestibulopathy (n=12), anxiety (n=11), migraine (n=10), seizure (n=6), medication effects (n=6), neuropathy (n=4), transient global amnesia (n=4), and other etiologies (n=19). Three of these patients had a stroke during 90-day follow-up. In an additional 182 patients (10.6%), neurologic symptoms improved but complete resolution within 24 hours was not documented; 19 of these patients (10.4%) had a stroke during follow-up. The stroke risk was not significantly different in the full cohort of patients with TIA and in the subgroup with TIAs confirmed by

**Figure.** Kaplan-Meier Life-Table Analysis of Survival Free From Stroke and All Adverse Events




Observations were indexed to the time of TIA symptom resolution. Time between symptom resolution and evaluation in the emergency department did not contribute to the analysis since a stroke occurring during this period would have excluded a case. Therefore, some patients entered the analysis after time zero. A, Adverse events include stroke, recurrent transient ischemic attack (TIA), hospitalization for a cardiovascular event, and death. B, Risk factors included age >60 years, duration of symptoms >10 minutes, diabetes mellitus, weakness associated with the transient ischemic attack (TIA), and speech impairment associated with the TIA. Follow-up was censored at the time of death or endarterectomy, so numbers do not reflect the total number of TIA patients at risk during the entire period.

neurologist review (10.5% vs 11.1%; $P=.64$ by the Fisher exact test). Compared with the full cohort, independent risk factors for stroke were similar in those with TIA diagnoses after neurologist review (age >60 years: OR, 1.8, 95% CI, 1.1-2.8; diabetes: OR, 2.4, 95% CI, 1.6-3.4; duration >10 min: OR, 2.5, 95% CI, 1.3-4.6; weakness: OR, 2.2, 95% CI, 1.6-3.2; speech impairment: OR, 1.3, 95% CI, 1.0-1.9).

## COMMENT

Transient ischemic attacks are ominous, carrying a substantial short-term risk of stroke, hospitalization for cardiovascular events, and death. The 90-day stroke risk was 10.5%, over 50 times that expected in a cohort of similar age.[2,36] Half of the strokes occurred within 2 days of the TIA. Short-term risks of cardiovascular events, death, and recurrent TIA were also high, with a combined 25.1% risk of an adverse event during the 3 months after a TIA.

Diagnosis of TIA is often problematic. It may be difficult to determine whether focal neurologic symptoms are due to ischemia from impaired cerebral circulation or due to seizure, migraine, or even syncope. Prior studies have required a neurologist's evaluation and strict entry criteria to establish the diagnosis of TIA,[25,26] but this does not solve the problem. Even among neurologists there is low interobserver agreement in the diagnosis of TIA.[27,28] Furthermore, evaluation by a neurologist is often not part of routine practice, in which ED and primary care physicians are primarily responsible for TIA diagnosis and management. In these settings, the diagnosis of TIA made by physicians caring for a patient is more useful for assessing natural history and defining stroke risk factors.[29] Therefore, we focused on the ED diagnosis of TIA. Based on neurologist review, these diagnoses appeared to be accurate in 94% of patients, although complete resolution of symptoms was not documented in an additional 11%. The prognosis of patients whose diagnoses were confirmed by neurologist review was not different from that of the entire cohort, suggesting that the ED diagnosis of TIA is reasonably reliable.

**Table 2.** Treatment of Patients With TIA and Associated 90-Day Stroke Risk (N = 1707)*

| Variable | No. (%) | 90-Day Stroke Risk With Characteristic, % | | P Value† |
|---|---|---|---|---|
| | | Present | Absent | |
| Hospital admission | 243 (14) | 10 | 7 | .07 |
| Aspirin | 1154 (68) | 10 | 12 | .15 |
| Ticlopidine | 199 (12) | 10 | 11 | .90 |
| Anticoagulation | 235 (14) | 14 | 10 | .04 |
| No medical therapy | 143 (8) | 13 | 10 | .27 |
| Discussed with neurologist | 446 (26) | 10 | 11 | .78 |
| Neurology consult | 60 (4) | 8 | 11 | .83 |

*Medications and interventions prescribed in the emergency department (ED) or at discharge. Hospital admissions include only those occurring directly from the ED at the initial evaluation. Stroke risks are unadjusted and likely reflect prognostic differences in patients chosen for treatment and should not be considered representative of treatment effects.
†Calculated using the Fisher exact test.

**Table 3.** Independent Risk Factors for Stroke Within 90 Days (N = 1707)*

| | Odds Ratio (95% CI) | P Value |
|---|---|---|
| Age >60 y | 1.8 (1.1-2.7) | .01 |
| Diabetes mellitus | 2.0 (1.4-2.9) | <.001 |
| Duration of episode >10 min | 2.3 (1.3-4.2) | .005 |
| Weakness with episode | 1.9 (1.4-2.6) | <.001 |
| Speech impairment with episode | 1.5 (1.1-2.1) | .01 |

*Based on logistic regression including all associated variables in univariate analysis ($P<.20$) with stepwise elimination of variables not contributing ($P>.10$). CI indicates confidence interval.

**Table 4.** 90-Day Stroke Risk by Number of Risk Factors*

| Risk Factors, No. | No. (%) | |
|---|---|---|
| | Patients | Stroke Within 90 Days |
| 0 | 22 (1) | 0 (0) |
| 1 | 179 (10) | 5 (3) |
| 2 | 509 (30) | 35 (7) |
| 3 | 584 (34) | 63 (11) |
| 4 | 337 (20) | 51 (15) |
| 5 | 76 (4) | 26 (34) |

*Risk factors are listed in Table 3.

The short-term risk that we observed was similar to that found in 2 smaller population-based studies of patients with TIA. Among 198 patients treated from 1955 to 1969 in Minnesota, the 3-month stroke risk was 10%,[25] similar to what we observed. Another study reported a 4% stroke risk in the first month among 184 patients enrolled a median of 3 days after the index TIA[26]; failure to include strokes that occurred during the first 3 days may account for the lower stroke risk in that study.

We studied patients who were evaluated in EDs of the hospitals of a health maintenance organization. It is possible that disincentives to using the ED led to selection of cases perceived to be at greater risk. Patients had longer duration of symptoms than previously reported for a sample that included patients identified in outpatient clinics.[37] The timing of presentation may also be important. Since more than half of strokes occurred within 2 days of the TIA, a patient arriving for evaluation 3 days after a TIA has already passed through the period of highest risk. Further study of patients diagnosed as having TIA in outpatient clinics is required to extend our findings to that group.

Currently available interventions for patients with TIA, including hospitalization and urgent carotid artery ultrasound, are expensive[10-12] and may not be cost-effective if used in all such patients. Stratification of stroke risk allows targeting expensive interventions to those at greatest short-term risk. We found 5 independent risk factors for stroke within 90 days after TIA: age older than 60 years, diabetes mellitus, duration of episode greater than 10 minutes, and weakness and speech impairment with the episode. These risk factors may identify patients whose symptoms are more likely due to cerebral ischemia or may indicate pathophysiologic conditions associated with greater risk. Combining these risk factors, we identified subgroups with minimal (0%) and very high (34%) short-term risk of stroke.

Prospective validation with an independent cohort will be required before these risk factors can be combined into a prediction model.[38] Validation also will be needed to test the possibility that the effects of symptom duration and speech impairment may be interdependent, as we observed in analyses that included interaction terms. Those analyses suggested that speech impairment may be a predictor only in those with symptoms lasting more than 10 minutes, or that symptom duration may be relevant only in those with speech impairment.

This observational study cannot provide reliable data on efficacy of therapies for TIA. It is likely that those perceived to be at highest risk of stroke were more often treated aggressively, and this would bias assessment of treatment benefit. Also, patients presented with TIAs despite any prophylactic antiplatelet or anticoagulant medications they were taking, so it is not surprising that prior medications did not reduce subsequent vascular events. Among patients who had not previously received an anticoagulant or antiplatelet medication, there was a nonsignificant trend toward reduced stroke risk in those who then received a prophylactic agent, but the size of the untreated group was small (n=143).

The short-term risk of stroke and other adverse vascular events is high among patients who present to the ED with a TIA. Urgent intervention in this group may be warranted, and the efficacy of such intervention should be studied. Stratification of short-term stroke risk appears practical, and may allow more cost-effective intervention and more efficient study of prevention strategies.

**Author Contributions:** Dr Johnston participated in the study concept and design, acquisition of data, analysis and interpretation of data, drafting of the manuscript, provided statistical expertise, obtained funding, provided administrative, technical, or material support, and supervised conduct of the study.

Dr Gress participated in the study concept and design, critical revision of the manuscript for important intellectual content, and supervised conduct of the study.

Dr Browner participated in study concept and design, analysis and interpretation of data, drafting of the manuscript, critical revision of the manuscript for important intellectual content, and provided statistical expertise.

Dr Sidney participated in the study concept and design, acquisition of data, analysis and interpretation of data, critical revision of the manuscript for important intellectual content, provided administrative, technical, or material support, and supervised conduct of the study.

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