Recanalization of Spontaneous Carotid Artery Dissection

Krassen Nedeltchev, MD; Stefan Bickel, MD; Marcel Arnold, MD; Hakan Sarikaya, MD; Dimitrios Georgiadis, MD; Matthias Sturzenegger, MD; Heinrich P. Mattle, MD; Ralf W. Baumgartner, MD

Background and Purpose—We set out to investigate the predictors and time course for recanalization of spontaneous dissection of the cervical internal carotid artery (SICAD).

Methods—We prospectively included 249 consecutive patients (mean age, 45±11 years) with 268 SICAD. Ultrasound examinations were performed at presentation, during the first month, and then at 3, 6, and 12 months, and clinical follow-ups after 3, 6, and 12 months.

Results—Of 268 SICADs, 20 (7.5%) presented with middle-aged patients.1,2 The main mechanism is arterio-arterial embolism or aspirin for stroke prevention.5,6 The time course of recanalization of the cervical internal carotid artery (SICAD) is a frequent cause of stroke in young to middle-aged patients.1,2 The main mechanism is arterio-arterial embolism,3,4 and most clinicians recommend the administration of anticoagulants or aspirin for stroke prevention.5,6 To enable a more sophisticated antithrombotic treatment of patients with spontaneous cervical artery dissection, the CADISP group recently reviewed the mechanism of brain ischemia, clinical experiences, and a systematic meta-analysis about antithrombotic agents in these patients.7 There is, however, no consensus about the duration of antithrombotic treatment. Many clinicians feel that antithrombotic therapy is no longer necessary once the flow in the dissected artery is reestablished.8

Combined extracranial and transcranial ultrasound is useful, particularly in patients presenting with carotid territory ischemia for assessing carotid stenosis and occlusion in patients with SICAD, both in the acute phase and during recanalization.9,10

We undertook this study to investigate the predictors and the time course of recanalization of the cervical internal carotid artery using ultrasound in patients with SICAD.

Spontaneous dissection of the cervical internal carotid artery (SICAD) is a frequent cause of stroke in young to middle-aged patients.1,2 The main mechanism is arterio-arterial embolism,3,4 and most clinicians recommend the administration of anticoagulants or aspirin for stroke prevention.5,6 To enable a more sophisticated antithrombotic treatment of patients with spontaneous cervical artery dissection, the CADISP group recently reviewed the mechanism of brain ischemia, clinical experiences, and a systematic meta-analysis about antithrombotic agents in these patients.7 There is, however, no consensus about the duration of antithrombotic treatment. Many clinicians feel that antithrombotic therapy is no longer necessary once the flow in the dissected artery is reestablished.8

Combined extracranial and transcranial ultrasound is useful, particularly in patients presenting with carotid territory ischemia for assessing carotid stenosis and occlusion in patients with SICAD, both in the acute phase and during recanalization.9,10

We undertook this study to investigate the predictors and the time course of recanalization of the cervical internal carotid artery using ultrasound in patients with SICAD.

Key Words: carotid ultrasound ■ dissection ■ outcome ■ recanalization

Subjects and Methods

Consecutive patients who presented with SICAD at 2 university-based stroke centers (Zurich and Bern) were prospectively included in a database from October 1992 until October 2005. The diagnosis of SICAD was based on imaging findings (cervical MRI and MRA, digital subtraction angiography, or both). Carotid dissection was considered proven if the cervical internal carotid artery (ICA) showed a string sign, an intimal flap, or an aneurysm at angiography, or a wall hematoma at cervical MRI, or both.11 Patients with a narrowed or occluded cervical ICA on the initial ultrasound were included. Patients were excluded if they underwent thrombolytic therapy for acute stroke or surgical or endovascular treatments for SICAD. Furthermore, SICAD recurring in a previously dissected carotid artery was not analyzed.

Baseline investigations have been reported before1 and include neurological and physical examinations, routine blood analysis, 12-lead ECG, ultrasound studies of the cerebral arteries, and cerebral CT, MRI, or both in patients with stroke or TIA. The following stroke risk factors were assessed: age, sex, hypertension (defined as a history of hypertension [systolic blood pressure >160 mm Hg, diastolic blood pressure >90 mm Hg] until September 2000, and systolic blood pressure >140 mm Hg, diastolic blood pressure >85 mm Hg since October 200012) measured on 2 separate occasions, or antihypertensive treatment, or both), diabetes mellitus (defined by preadmission history or venous plasma glucose concentration of ≥7.0 mmol/L after an overnight fast on at least 2 separate occasions, or antihypertensive treatment, or both), diabetes mellitus (defined by preadmission history or venous plasma glucose concentration of ≥7.0 mmol/L after an overnight fast on at least 2 separate occasions, or antihypertensive treatment, or both), diabetes mellitus (defined by preadmission history or venous plasma glucose concentration of ≥7.0 mmol/L after an overnight fast on at least 2 separate occasions, or antihypertensive treatment, or both).
occasions, or ≥11.1 mmol/L 2 hours after oral ingestion of 75 grams of glucose and on 1 other occasion during the 2-hour test), current cigarette smoking, hypercholesterolemia (defined by prediagnosis history or total cholesterol concentration >5 mmol/L, low-density lipoprotein cholesterol concentration >3 mmol/L, total cholesterol/high-density lipoprotein ratio >5), increased high-density lipoprotein cholesterol levels (defined by high-density lipoprotein cholesterol concentration <1 mmol/L), coronary artery disease, and migraine.

Patients were categorized according to the presenting signs and symptoms into 5 groups: (1) stroke (ischemic deficit lasting >24 hours); (2) TIA (ischemic deficit lasting <24 hours); (3) amaurosis fugax or retinal infarction; (4) 1 or several of the following local symptoms and signs: headache, neck pain, pulsatile tinnitus, Horner syndrome, or cranial nerve palsy on the side of SICAD; and (5) asymptomatic. Stroke severity on admission was assessed with the National Institute of Health Stroke Scale.13

The ultrasound studies were performed with the same equipment (for extracranial and transcranial color duplex studies, Acuson XP 10 or Sequoia; for transorbital Doppler studies, EME; and since October 1997, Acuson XP 10 or Sequoia). Extracranial color duplex sonography of the ICA at the origin, the external carotid artery, and common carotid artery was performed with linear probes (2.0–3.5 MHz), and of the cervical ICA with sector probes (2.0–3.5 MHz). Transorbital insolation of the ophtalmic arteries and the carotid siphon was performed with pulsed-wave Doppler probes (2 MHz) or color Doppler sector probes (2.0–3.5 MHz). Transcranial color duplex sonography studies of the basal cerebral arteries were performed with sector probes (2.0–3.5 MHz) as reported previously.14 In brief, the transtemporal approach was used for insolation of the terminal (C1) ICA, middle, anterior, precommunicating (P1), and postcommunicating (P2) posterior cerebral artery, and anterior communicating arteries. Intracranial arteries were investigated for the presence of stenoses, occlusions, and cross-flow through the anterior communicating artery according to previously published criteria.14,16 Since 1996, all patients with insufficient temporal bony windows were also investigated with the echocontrast agent Levovist using concentrations of 400 mg/mL,17 and since July 2005 with the echocontrast agent Sonovue.18

Stenoses of the cervical ICA were quantified according to the following criteria11 (Table 1): ≤50% stenosis was diagnosed when intrastenotic peak systolic velocity (PSV) was >90 cm/sec in women and >80 cm/sec in men, and the PSV quotient intrastenotic ICA/contralateral cervical ICA was >1.12 (each reference value was higher than the PSV mean value plus 3 SD of 78 unpublished own healthy volunteers). A >50% stenosis was diagnosed when intrastenotic PSV was >120 cm/sec and the PSV quotient intrastenotic ICA/contralateral common carotid artery on the side of ICAD (ipsilateral) was >1.5. Intrastenotic velocities are frequently decreased in SICAD, causing high-grade stenosis.18 To avoid falsely negative findings, only >80% stenoses were diagnosed using prestenotic and poststenotic hemodynamic criteria, and at least 2 of the following 3 had to be present: (1) the quotient of the resistance index (PSV/peak end-diastolic velocity/PSV) ipsilateral common carotid artery/resistance index contralateral common carotid artery >0.15; (2) reversed flow in the ipsilateral ophthalmic artery; and (3) cross-flow through the anterior communicating artery. The criteria for 51% to 80% and 81% to 99% ICA stenoses have been published in peer-reviewed journals.9,19–21 developed in studies using catheter angiography and the North American Symptomatic Carotid Endarterectomy Trial criteria as standard of reference, and elaborated with the same ultrasound equipment used in the present study. The last criterion was used because different ultrasound machines have been shown to measure different flow velocities under identical conditions of examination in both flow phantoms and patients.22 ICA occlusion was assessed as reported before (Table 1).

Ultrasound follow-up examinations to assess recanalization were performed within the first month after the onset of SICAD symptoms, and after 3, 6, and 12 months. If an ultrasound follow-up showed normal findings (ie, complete recanalization), then no further examinations were performed. Complete recanalization was diagnosed when ultrasound showed normal findings, and partial recanalization was diagnosed when ultrasound revealed a reduction in the degree of stenosis (from 81%–99% to 51%–80% or from 51%–80% to ≤50%) or a change from occlusion to a stenosis. The time interval (days) from SICAD diagnosis to complete recanalization (Δt) was calculated according to the formula: Δt = (ts – td)/2, where ts is the time point of the first ultrasound examination showing normal findings, td is the time point of the last ultrasound examination with pathological findings, and ts is the time point of the ultrasound examination at presentation. All ultrasound studies were performed or supervised by an experienced sonographer.

Choice and duration of antithrombotic treatment was at the discretion of the treating neurologist. No selection criteria toward aspirin or anticoagulation were applied. Before the publication of the International Stroke Trial in 1997,24 most SICAD patients were treated with anticoagulation. After the publication of the International Stroke Trial results, some neurologists started to treat acute strokes caused by SICAD with aspirin. Clinical follow-ups were performed after 3, 6, and 12 months. In patients with a stroke, 3-month functional outcome was assessed using the modified Rankin scale.25 The modified Rankin scale scores of 0 to 1 were defined as “favorable,” and modified Rankin scale scores of 2 to 6 were defined as “unfavorable” outcome. In patients with suspicion of stroke, TIA, retinal ischemia, recurrent dissection, and the occurrence of new local symptoms or signs, the diagnostic work-up mentioned was repeated. During 3-month follow-up, 1 patient had a stroke and 8 patients had a TIA. Neurologists or physicians in neurology training certified for National Institute of Health Stroke Scale and modified Rankin scale assessment performed the baseline and the follow-up clinical examinations. The study was approved by the local ethics committee.

### Statistical Analysis

Data are reported in frequency tables. The effects of patient characteristics on recanalization were assessed using the χ² test (for
comparison of proportions), Student t test (for comparison of continuous variables), and Mann–Whitney U test (for comparison of ordinal variables). A multivariate logistic regression analysis, which included variables that showed statistical difference $P \leq 0.1$ on univariate comparison, was performed. The Kaplan-Meier method was used to determine the likelihood of recanalization at 3, 6, and 12 months. $P < 0.05$ was considered statistically significant.

## Results

### Presenting Findings

We evaluated 321 consecutive patients (189 [59%] men; mean age, 46±10 years) with 354 SICAD. Of the 321 patients with SICAD, 241 (75.1%) presented with symptoms of ischemia from the carotid territory, 79 (24.6%) presented with local symptoms only, and 1 patient (0.3%) had vertigo vice versa in 22 (8%) patients.

Of 321 patients with SICAD, 72 patients with 86 SICAD were not included because they met ≥1 exclusion criterion (Table 2). Thus, 249 patients (142 [57%] men; mean age, 45±11 years) with 268 SICAD were included in the study.

The presenting clinical characteristics of 160 SICAD (60%) that recanalized completely and 108 SICAD (40%) that did not recanalize completely during follow-up are shown in Table 3. Clinical presentation with local symptoms and signs was more frequent in the group of SICAD with complete recanalization ($P = 0.048$). The other presenting clinical characteristics did not differ between the 2 groups.

The presenting ultrasound findings were assessed after a median delay of 2 days (range, 0–58 days) after the onset of SICAD symptoms, and are shown in Table 4. Of 268 SICAD, 20 (7.5%) showed ≤50% stenosis, 31 (11.6%) showed 51% to 80% stenosis, 92 (34.3%) showed 81% to 99% stenosis, and 125 (46.6%) showed an occlusion. A ≤50% stenosis at presentation was more frequent in the group of SICAD with complete recanalization at follow-up ($P = 0.004$). On the contrary, 81% to 99% stenoses or total occlusions at presentation were more frequent in the group of SICAD without complete recanalization. The other ultrasound findings did not differ between the 2 groups. Antithrombotic treatment included anticoagulation in 174 (67%) patients, aspirin in 64 (24%) patients, and aspirin followed by anticoagulation or vice versa in 22 (8%) patients.

### Clinical Follow-Up

The functional outcome at 3 months was assessed in 154 (98%) of 157 patients with stroke. A favorable outcome

### Table 2. Reasons for Excluding 72 Patients With Spontaneous Carotid Dissection

<table>
<thead>
<tr>
<th>Excluded Patients, n (%)</th>
<th>Excluded ICA Dissections, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal baseline ultrasound findings</td>
<td>33</td>
</tr>
<tr>
<td>Intra-arterial thrombolysis</td>
<td>4</td>
</tr>
<tr>
<td>Carotid surgery</td>
<td>4</td>
</tr>
<tr>
<td>No ultrasound follow-up</td>
<td>15</td>
</tr>
<tr>
<td>Death from stroke caused by carotid dissection</td>
<td>8</td>
</tr>
<tr>
<td>Recurrent dissection in a carotid artery</td>
<td>0</td>
</tr>
<tr>
<td>All</td>
<td>72*</td>
</tr>
</tbody>
</table>

*Some patients met ≥1 exclusion criterion.

### Table 3. Presenting Clinical Characteristics in 268 Spontaneous Carotid Dissections With and Without Complete Recanalization Occurring in 249 Patients

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Complete Recanalization, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n=160)</td>
</tr>
<tr>
<td>Mean age±SD, yr</td>
<td>45±10</td>
</tr>
<tr>
<td>Male</td>
<td>90 (56)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>43 (27)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Current smoking</td>
<td>42 (26)</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>58 (36)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Migraine</td>
<td>15 (9)</td>
</tr>
<tr>
<td>Minor trauma</td>
<td>34 (21)</td>
</tr>
<tr>
<td>Stroke</td>
<td>86 (54)</td>
</tr>
<tr>
<td>TIA</td>
<td>36 (23)</td>
</tr>
<tr>
<td>Retinal ischemia</td>
<td>21 (13)</td>
</tr>
<tr>
<td>Local symptoms and signs only</td>
<td>128 (80)</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>10 (6)</td>
</tr>
<tr>
<td>Median NIHSS in stroke patients (range)</td>
<td>10 (1–28)</td>
</tr>
</tbody>
</table>

Local symptoms and signs include: headache, neck pain, and pulsatile tinnitus, and Horner syndrome or cranial nerve palsy on the side of carotid dissection.

NIHSS indicate National Institutes of Health Stroke Scale.

P = difference between subgroups by $\chi^2$ test, t test, or Mann–Whitney U test.

### Table 4. Presenting Ultrasound Findings in 268 Spontaneous Carotid Dissections With and Without Complete Recanalization Occurring in 249 Patients

<table>
<thead>
<tr>
<th>Presenting Characteristic</th>
<th>Complete Recanalization, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n=160)</td>
</tr>
<tr>
<td>Left</td>
<td>89 (56)</td>
</tr>
<tr>
<td>Unilateral</td>
<td>144 (90)</td>
</tr>
<tr>
<td>Cervical carotid artery</td>
<td></td>
</tr>
<tr>
<td>Stenosis ≤50%</td>
<td>18 (11)</td>
</tr>
<tr>
<td>51%–80%</td>
<td>19 (12)</td>
</tr>
<tr>
<td>81%–90%</td>
<td>67 (42)</td>
</tr>
<tr>
<td>Occlusion</td>
<td>56 (35)</td>
</tr>
</tbody>
</table>

P = difference between subgroups by $\chi^2$ test with regard to occlusive spontaneous carotid dissection vs nonocclusive carotid dissection at presentation.
Recanalization of SICAD

All patients underwent a total of 1190 ultrasound studies. At 3-month follow-up, complete recanalization was observed in 133 (50%) of 268 SICAD. At 6- and 12-month follow-ups, complete recanalization was observed in 160 (60%) of 268 SICAD; (2) complete recanalization occurred within the first 6 months of follow-up (Figure). The estimated rate of complete recanalization was 15.7% (95% CI, 13.7%–17.7%) at 1 month, 50.4% (95% CI, 47.3%–53.5%) at 3 months, and 59.7% (95% CI, 56.7%–62.7%) at 6 and 12 months.

Predictors of Complete Recanalization of SICAD

The probability values for the difference of presenting clinical characteristics between SICAD with and without complete recanalization are shown in Table 3. This univariate analysis showed that SICADs with only local symptoms and signs were associated with complete recanalization (P=0.048), whereas occluded SICADs were less likely to recanalize (P<0.001). There was a trend toward a higher prevalence of complete recanalization in patients treated with anticoagulants, although the association did not reach statistical significance (P=0.064).

Multivariate logistic regression analysis included the variables that showed statistical difference P≤0.1 on univariate comparison, ie, SICAD with stroke (P=0.051), SICAD with TIA (P=0.10), SICAD with local symptoms and signs only (P=0.048), occlusive SICAD (P<0.001), and anticoagulation (P=0.064). SICAD with local symptoms and signs only was independently associated with complete recanalization (OR, 0.4; 95% CI, 0.2–0.8, P=0.014), whereas occlusive SICAD reduced the odds of complete recanalization (OR, 4.0; 95% CI, 2.2–7.3).

Discussion

In this prospective observational study, we found: (1) complete recanalization occurred in 160 (60%) of 268 SICAD; (2) complete recanalization occurred within the first 6 months but not thereafter; and (3) occlusive SICAD was less likely to recanalize, whereas SICAD presenting with local symptoms and signs only was independently associated with recanalization.

The 60% complete recanalization rate of SICAD observed in this investigation is similar to the results of previous smaller ultrasound studies.\textsuperscript{10,23} Steinke et al\textsuperscript{10} observed 48 patients with 50 SICAD who were treated with anticoagulation for an average period of 51 days. Complete recanalization was found in 34 (68%) patients. Sturzenegger et al\textsuperscript{23} followed-up 43 patients with 44 SICAD during a mean interval of 15 months and reported complete recanalization in 25 (63%) of 40 patients. Desfontaines et al\textsuperscript{26} surveyed 60 patients with SICAD (spontaneous, n=50; traumatic, n=10) who were using anticoagulation in 34 cases for a mean time period of 37.5 months using ultrasound. Complete recanalization of SICAD was observed in 68%. In a catheter angiography study, 23 patients with 23 stenotic and 4 occlusive SICAD were restudied angiographically after a mean delay of 58.5 months (range, 14 to 140).\textsuperscript{27} Recanalization was

\begin{table}
\centering
\caption{Ultrasound Findings Observed at Presentation and at the Last Follow-Up in 268 Dissected Carotid Arteries Occurring in 249 Patients}
\begin{tabular}{|c|c|c|c|c|c|}
\hline
\textbf{Ultrasound Findings at Presentation} & \textbf{Normal, n (%)} & \textbf{Stenosis \leq 50\%, n (%)} & \textbf{Stenosis 51\%–80\%, n (%)} & \textbf{Stenosis 81\%–99\%, n (%)} & \textbf{Occlusion, n (%)} \\
\hline
\textbf{Stenosis} & & & & & \\
\hline
\textless{}50\% (n=20) & 18 (90) & 2 (10) & \ldots & \ldots & \ldots \\
51\%–80\% (n=31) & 19 (61) & 10 (32) & 1 (3) & \ldots & 1 (3) \\
81\%–99\% (n=92) & 67 (73) & 9 (10) & 1 (1) & 14 (15) & 1 (1) \\
Total occlusion (n=125) & 56 (45) & 6 (5) & 2 (2) & 12 (10) & 49 (39) \\
All (n=268) & 160 (60) & 27 (10) & 4 (1) & 26 (10) & 51 (19) \\
\hline
\end{tabular}
\end{table}
Complete recanalization was observed in the first 6 months after the onset of symptoms but not on later follow-up in our series. These findings are in accordance with the results of previous studies reporting 92% to 100% vessel recanalization occurring within 6 months.

The time course of SICAD recanalization observed in this study supports the current clinical practice of many centers, including our own. Vascular studies are performed 3 and 6 months after the onset of SICAD symptoms, and antithrombotic treatment is administered at least until complete recanalization is observed. Many vascular neurologists administer long-term low-dose oral aspirin in SICAD patients with aneurysm to prevent thromboembolic events. Under the unproven assumption that aspirin is mandatory for stroke prevention in patients with SICAD causing an aneurysm, the absence of follow-up MRA for detecting such an aneurysm that develops during recanalization may be a limitation of this study. The present study does not address the question of whether patients with and without persistent carotid stenosis or occlusion caused by SICAD should undergo antithrombotic treatment for stroke prevention beyond 6 months after symptom onset. An occlusive SICAD was an independent negative predictor of complete recanalization, which occurred in 45% of occlusive dissections in this study. These findings are in accordance with the results of 2 previous ultrasound studies. Sturzenegger et al observed a complete recanalization in 18 (60%) of 30 occlusive SICAD. Another study assessed recanalization in 38 patients with acute stroke attributable to occlusive forms of SICAD and spontaneous vertebral artery dissection. Of 38 vessels, 16 (42%) recanalized completely within 2 days and 6 weeks after the onset of stroke. The recanalization rate of SICAD alone was not reported.

SICAD causing only local symptoms and signs were independently associated with recanalization in our series. In a previous study, SICADs causing carotid territory ischemia showed a lower prevalence of Horner syndrome and caudal cranial nerve palsies than SICADs causing no ischemic events. Furthermore, SICADs with severe stenosis and occlusions more often caused carotid territory ischemia than SICADs with less severe stenosis or without stenosis. One may speculate that SICAD with carotid territory symptoms is more often an inward expansion of the wall hematoma, which causes more severe stenosis and occlusion and is less likely to recanalize. On the contrary, outward expanding mural hematomas may cause less severe stenoses, occlusions, and ischemia, but more frequent local symptoms and signs only. They are thus associated with higher rates of complete recanalization.

We did not observe any association between hypertension and the recanalization rate, as was reported by Caso et al. However, the hypothesis that hypertension damages the elasticity and the permeability of the arterial wall is intriguing and deserves further research.

Previous case reports and small series suggested that anticoagulation might impair recanalization of SICAD by increasing the extent of the intramural hematoma. In this series there was no indication that anticoagulation had a negative impact on carotid recanalization. On the contrary, there was a trend toward better recanalization on anticoagulants. A recent overview about antithrombotic treatment of patients with SICAD has been provided by the CADISP group.

In the present study, complete recanalization did not influence the functional outcome in patients with stroke, which is in keeping with the findings of acute stroke treatment trials.

The present study has some limitations. The choice and duration of antithrombotic therapy was not identical for all patients, but was at the discretion of the treating physician. The reason is that no controlled randomized trial has compared different antithrombotic treatments. Another drawback is the heterogeneity with respect to the timing of initial and follow-up ultrasound investigations. This limitation, however, was not avoidable, because several predictable facts prevented the establishment of a defined timetable for ultrasound studies: It is not possible to determine precisely the time of SICAD onset, particularly when diagnosis is not established by cervical MRI. SICAD patients will be outpatients or inpatients with different durations of hospital stay, depending on clinical presentation and the severity of the stroke. Headache or neck pain, which is the most frequent local symptoms, may show the same location, quality, and intensity compared to previous head or neck pain, and thus prevent the determination of the onset of SICAD symptoms. The time interval from symptom onset to arrival in our hospitals is significantly longer in patients presenting with local symptom and signs compared to patients with ischemia. Finally, the time interval from symptom onset to admission has decreased in patients with stroke and TIA in the past years, which would have urged us to readapt the timing of the ultrasound studies.

In conclusion, when complete recanalization of SICAD was achieved, it occurred within 6 months after the onset of symptoms but not thereafter. In addition, nonocclusive SICAD as well as SICAD causing local symptoms and signs only were independent predictors of recanalization.

Acknowledgments

The authors thank Andros Tofiel for his thoughtful linguistic review of the manuscript.

Sources of Funding

This study was supported by a grant from The Olga-Mayenfisch Foundation, Zurich and The Novartis Foundation, Basel.
References


Recanalization of Spontaneous Carotid Artery Dissection
Krassen Nedeltchev, Stefan Bickel, Marcel Arnold, Hakan Sarikaya, Dimitrios Georgiadis, Matthias Sturzenegger, Heinrich P. Mattle and Ralf W. Baumgartner

*Stroke*. 2009;40:499-504; originally published online December 24, 2008;
doi: 10.1161/STROKEAHA.108.519694

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 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/40/2/499

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/