Noninvasive Monitoring of Internal Carotid Artery Dissection

Wolfgang Steinke, MD; Wolfgang Rautenberg, MD; Andreas Schwartz, MD; Michael Hennerici, MD

Background and Purpose Internal carotid artery dissection (ICAD) is not known, it has increasingly been recognized as a cause of cerebrovascular events, particularly in young subjects. To date, angiography, which identifies characteristic features of ICAD, has been the keystone in establishing the diagnosis. However, because ICAD is a changing rather than a stable arterial disease, serial follow-up studies are required to assess the development of carotid dissection. In this respect, angiography is of limited value. In one of the largest series including 69 ICADs, the initial angiogram was performed after an average interval of 28 days and within a range of 1 to 247 days from symptom onset. Angiography was repeated in 50 dissections after a variable interval ranging from 6 to 896 days. Thus, scarce information was provided about the temporal profile of recanalization. In addition, very early and late resolution of ICAD were certainly not assessed.

Ultrasonographic findings in ICAD have recently been reported in several series. Most of these studies either included a relatively small number of patients or did not present a detailed analysis of the Doppler and duplex findings. In addition, common carotid artery (CCA) dissection, which has a different etiology and sonographic features than ICAD, was included in one study. Although some authors assessed the frequency of recanalization and occlusion by means of sonographic methods, only one report addressed the time course of ICAD in greater detail. Magnetic resonance imaging (MRI) may display the mural hematoma; however, it is inadequate to reliably assess recanalization, and experience with serial MR angiographic studies of ICAD is still limited.

Because patients with ICAD do not always present with the characteristic triad of neck or head pain, Horner's syndrome, and focal cerebral ischemic symptoms but with variable or minimal symptoms in many cases, the initial assessment of diagnostic Doppler and duplex features in all suspected cases is essential. We present the results of the largest currently reported prospective series of systematic serial sonographic investigations in 50 angiographically confirmed dissections with particular respect to the temporal development of the arterial disease.

Subjects and Methods Since 1982, we have consecutively examined 48 patients (18 men, 30 women; mean age, 44 years; range, 16 to 68 years) with 50 ICADs by serial prospective ultrasonographic studies. The initial duplex Doppler studies were performed because of ischemic cerebrovascular events, oculosympathetic paresis, and unilateral neck pain or headache. All patients had the first ultrasonographic examination within 3 days after onset of symptoms. Nineteen (39%) patients had a history of a minor head or neck trauma (n = 7) or other possibly provocative situations, such as a variety of sports activities, surgical procedures, chiropractic therapy, vomiting, or coughing (n = 12), that preceded the onset of symptoms for an average interval of 7 days. In 2 patients the dissection was bilateral; in 3 patients the carotid dissection was associated with a unilateral vertebral artery dissection.

Continuous-wave Doppler sonography (MDV; 4-MHz insonation frequency) or conventional duplex scanning (Diasonic) has increasingly been recognized as a cause of transient ischemic attack or stroke. However, scarce data exist on the natural history of the arterial lesions and the temporal profile of recanalization.
by guest on March 30, 2017 http://stroke.ahajournals.org/ Downloaded from

focal neurological symptoms, the clinical presentation patients presented with headache or neck pain without cerebrovascular events were not accompanied by a pain severe neurological deficits in 25%. Because 38% of all patients suffered a stroke, which initially produced focal cerebral symptoms (25%). Fifty-two percent of the cases, consisting of either amaurosis fugax attacks (4%) or transient symptoms occurred in 29% of the patients; 4% had Homer’s syndrome as the sole initial sign. A pulsatile tinnitus occurred in 12% on the affected side; 1 patient had a unilateral lingual paresis. The dissection. Oculosympathetic paresis (Homer’s syndrome) was associated with head or neck pain in 23% of the patients; 4% had Horner’s syndrome as the sole initial sign. A pulsatile tinnitus occurred in 12% on the affected side; 1 patient had a unilateral lingual paresis. Ischemic cerebrovascular events were frequent (81%). Transient symptoms occurred in 29% of the patients, consisting of either amaurosis fugax attacks (4%) or focal cerebral symptoms (25%). Fifty-two percent of the patients suffered a stroke, which initially produced severe neurological deficits in 25%. Because 38% of all cerebrovascular events were not accompanied by a pain syndrome or oculosympathetic paresis and 15% of the patients presented with headache or neck pain without focal neurological symptoms, the clinical presentation did not primarily suggest the diagnosis of ICAD in more than half of the cases. The stroke incidence was higher among patients with than those without prior trivial trauma (71% versus 48%).

**Doppler Sonography**

Continuous-wave Doppler sonography and spectrum frequency analysis from the pulsed-wave Doppler system incorporated in the duplex instrument demonstrated initial abnormal Doppler signals in all 50 dissections (Table 1). Until the present series, we had not seen a patient with normal Doppler sonographic results who had features of an ICAD on the angiogram (sensitivity, 100%). A characteristic high-resistance flow pattern with bidirectional signal components and absent diastolic flow was found in 68% (Fig 1). Typically, this Doppler signal could be traced along the extracranial carotid artery up to the submandibular region. During the study period a high-resistance Doppler signal was assessed along the course of the cervical ICA in 2 patients without angiographic evidence of ICAD: both had an acute embolic occlusion of the intracranial carotid artery bifurcation. Thus, the statistical analysis of the diagnostic accuracy of the high-resistance Doppler signal revealed a moderate sensitivity (68%), while the specificity (99%) as well as the positive (94%) and negative (99%) predictive values were high based on an estimated number of at least 3000 carotid artery angiograms and corresponding duplex Doppler studies in patients with cerebrovascular disease performed during the study period.

A markedly reduced systolic and diastolic flow velocity in the ICA, decreased flow velocity in the ipsilateral CCA on the affected side, and retrograde flow in most ophthalmic arteries indicated a hemodynamically relevant distal ICA obstruction in 14% of the dissections. A submandibular stenosis was assessed in 8% by means of the low-frequency pulsed-wave Doppler system, which was also used for transcranial insonation. Absence of any Doppler signal indicated complete occlusion of the ICA in 8%. Signs of a high-grade ICA stenosis at its origin from the bifurcation was found in only 2%.

Increasing diastolic flow velocity in the ICA indicated recanalization of the dissection, which occurred gradually over several weeks in most cases (Fig 2). According to sonographic criteria, 68% of the dissections recanalized. Of 34 ICADs with initial high-resistance pattern, 71% resolved (Table 1); however, resolution was incomplete in 2 cases with decreased flow velocity in the

<table>
<thead>
<tr>
<th>Initial Doppler Sonography</th>
<th>Recanalized</th>
<th>Occluded</th>
<th>Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-resistance flow pattern</td>
<td>34 (68)</td>
<td>24 (48)</td>
<td>6 (12)</td>
</tr>
<tr>
<td>Distal obstruction</td>
<td>7 (14)</td>
<td>6 (12)</td>
<td>0</td>
</tr>
<tr>
<td>Submandibular stenosis</td>
<td>4 (8)</td>
<td>2 (4)</td>
<td>0</td>
</tr>
<tr>
<td>Occlusion</td>
<td>4 (8)</td>
<td>2 (4)</td>
<td>0</td>
</tr>
<tr>
<td>Bifurcation stenosis</td>
<td>1 (2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>50 (100)</td>
<td>34 (68)</td>
<td>6 (12)</td>
</tr>
</tbody>
</table>

Values in parentheses are percent.

**TABLE 1. Initial Doppler Signals and Follow-up Results In 50 Internal Carotid Artery Dissections**

ics DRF 400; high-resolution 10-MHz B-mode system combined with a 5-MHz pulsed-wave spectrum analysis of the carotid and vertebral arteries was performed in 2- to 4-day intervals during the first weeks after the onset of symptoms and followed in 1- to 2-month intervals with a mean follow-up time of 7 months (range, 1 month to 24 months). Since 1989, Doppler color flow imaging (Acuson 128 with a 7.5-MHz transducer, Acuson GmbH, or QAD PV with a 10-MHz probe, Philips Medical System) replaced conventional duplex sonography in 14 patients. Recanalization of the dissection was defined as the reappearance and increase, respectively, of diastolic blood flow in the Doppler spectrum. Transcranial Doppler sonography (TCD) using a pulsed-wave system operating at 2-MHz insonation frequency (TC2-64, Eden Medizinische Electronic GmbH) was performed in 32 patients to assess collateral blood flow. Transcranial insonation was not possible in 1 patient because of a missing temporal bone window. Methods and techniques of extracranial and transcranial Doppler sonography, standard duplex scanning, and Doppler color flow imaging have been described elsewhere.18-23

All patients had a computed tomographic scan or MRI of the head. In addition, transverse MRI in conventional spin-echo technique and T2-weighted two-dimensional gradient-echo sequences with transversal spoiling was extended caudally in 9 patients to display the mural hematoma of the ophthalmic arteries indicated a hemodynamically relevant distal ICA obstruction in 14% of the dissections. A submandibular stenosis was assessed in 8% by means of the low-frequency pulsed-wave Doppler system, which was also used for transcranial insonation. Absence of any Doppler signal indicated complete occlusion of the ICA in 8%. Signs of a high-grade ICA stenosis at its origin from the bifurcation was found in only 2%.

Increasing diastolic flow velocity in the ICA indicated recanalization of the dissection, which occurred gradually over several weeks in most cases (Fig 2). According to sonographic criteria, 68% of the dissections recanalized. Of 34 ICADs with initial high-resistance pattern, 71% resolved (Table 1); however, resolution was incomplete in 2 cases with decreased flow velocity in the
affected carotid artery, and in 3 other dissections stenosis at the bifurcation and in the submandibular carotid segment, respectively, persisted. All ICADs with sonographic evidence of a distal carotid obstruction resolved completely except in 1 patient, who died 7 days after a complete middle cerebral artery (MCA) infarction. One half of the submandibular ICA stenoses and occlusions resolved.

The interval from the onset of symptoms to sonographic recanalization of the dissection ranged from 2 days to 1 year (mean period, 51 days). Analysis of the temporal profile of resolution revealed that most of the dissections recanalized during the first 2 months after the onset of symptoms with a cluster in the fourth week (Fig 3).

Initial TCD demonstrated collateral blood flow from the contralateral carotid artery via the anterior communicating artery in 62% of the patients. A combined collateral supply from both the anterior and posterior communicating artery was seen in 13%. Because of the absence of relevant cross-flow through the anterior or posterior communicating artery, increased flow velocity in the P1 and P2 segments of the posterior cerebral artery ipsilateral to the dissection indicated leptomeningeal collaterals from the posterior circulation in 9%. Collateral flow was not present in 5 patients (16%); in 3 of these patients Doppler spectra demonstrated reduced flow velocity in the ipsilateral MCA. The frequency of transient ischemic attack or stroke was not different among patients with or without collateral flow.
FIG 3. Intervals from symptom onset until Doppler sonographic recanalization of 34 internal carotid artery dissections.

from the contralateral or posterior circulation. Absent Doppler signals suggested MCA occlusion in 2 patients, which was confirmed by early angiography. Repeat TCD indicated recanalization of the MCA occlusions after 4 weeks in both cases, which again was confirmed by angiography. After extracranial Doppler sonography had assessed recanalization, transcranial follow-up studies reflected subsequent resolution of the hemodynamically relevant ICA obstruction.

Standard Duplex and Color Flow Imaging

High-resolution gray-scale (B-mode) echotomograms of 27 ICADs revealed no morphological abnormalities at the bifurcation or in the proximal ICA in 40% of the cases. The most frequent (29%) pathological finding was a tapering lumen of the ICA a short distance above the bifurcation (Fig 4A). A moving, thin, echogenic intravascular structure, which probably represented the dissected inner vessel wall ("intimal flap") or, less likely, a small, floating thrombus, was visualized in 15% (Figs 4B and 4C). Echolucent matrix caused by a large mural hematoma, which produced an increase of the arterial diameter by 3 mm compared with the contralateral unaffected ICA, was found in 1 (4%) patient. In another patient (4%), the ICA was not displayed adequately. Small nonstenotic atherosclerotic plaques were seen in 2 (8%) patients at the origin of the dissected ICA.

In dissections with normal B-mode scan as well as in those with tapering arterial lumen, Doppler color flow imaging demonstrated extremely reduced blood flow velocity in the ICA, which often produced only a few red pixels in 1 or 2 of the 18 frames-per-second real-time display (Fig 4A). A zone of blue-coded flow reversal, which was predominantly located at the origin of the ICA, was present in all dissections (Figs 1 and 4A). In cases showing an intimal flap on the B-mode scan, color flow signals did not demonstrate flow in the "false" lumen separated by the dissected arterial wall. Gradually increasing intensity and velocity of red-coded blood flow indicated recanalization of the dissection corresponding to the reappearance of diastolic flow in the Doppler spectrum analysis.

Angiography

Initial intra-arterial angiography essentially demonstrated four different features. A long segment dissection ("string sign") was the most frequent finding (36%) (Table 2). Pseudo-occlusion was present in 28% of dissections (Fig 2) and a distal subcranial ICA stenosis in 20%. Pseudoaneurysms proximal to the carotid canal were found in 10% and were commonly associated with significant ICA stenosis. Forty-five of the initial angiographic studies were performed before sonographic

**FIG 4.** Doppler color flow imaging of acute carotid dissection. A, Gray-scale echotomogram demonstrates pseudo-occlusion (open arrow) of internal carotid artery (ICA) a few centimeters distal to bifurcation. Complex hemodynamics are present, demonstrating segments of reduced orthograde flow in distal common carotid artery (red signals), reversed flow at origin of ICA (blue), and persistent flow distal to pseudo-occlusion (red). B, Floating sail-like intravascular structure on B-mode echotomogram of another ICA dissection probably represents an intimal flap (arrows). C, Corresponding cross-section reveals almost complete transient blockage of vessel lumen due to moving structure with reduced red-coded flow along the vascular wall (arrows).
TABLE 2. Angiographic Features of 50 Initial and 19 Repeat Intra-arterial Angiograms of Internal Carotid Artery Dissection and Mean Intervals Between Symptom Onset and Angiography

<table>
<thead>
<tr>
<th>Angiographic Feature</th>
<th>Initial Angiography</th>
<th>Repeat Angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean Interval</td>
<td>Mean Interval</td>
</tr>
<tr>
<td>Pseudo-occlusion</td>
<td>14 (28)</td>
<td>0</td>
</tr>
<tr>
<td>Distal stenosis</td>
<td>10 (20)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Long dissection</td>
<td>18 (36)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Pseudoaneurysm</td>
<td>5 (10)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Normal, irregular</td>
<td>2 (4)</td>
<td>12 (62)</td>
</tr>
<tr>
<td>Occlusion</td>
<td>1 (2)</td>
<td>2 (11)</td>
</tr>
</tbody>
</table>

Values in parentheses are percent.

Signs of recanalization were seen. The five angiograms performed after the reestablished diastolic flow was assessed by Doppler sonography demonstrated a moderate distal stenosis (n=2), long dissection (n=1), and normal carotid arteries (n=2).

The repeat angiograms revealed complete resolution of the dissection or minor residual vascular wall irregularities in almost two thirds of the cases (62%) (Fig 2, Table 2). None of the pseudo-occlusions and only a few long dissections (11%), pseudoaneurysms (11%), and distal stenoses (5%) persisted. In 1 case with unchanged Doppler results indicating a moderate submandibular stenosis, reangiography after 1 year revealed a marked decrease in the size of the pseudoaneurysm. If the intervals between the onset of symptoms and the time of angiography were evaluated (Table 2), it was evident that the angiographic features reflected mainly the process of recanalization rather than different morphological types of dissection. According to the mean intervals, ICAD started with angiographic pseudo-occlusion, followed by distal stenosis or long dissection, and finally resolved with residual minor wall irregularities or persistent pseudoaneurysm in some cases.

Magnetic Resonance Studies

Axial MRI of the neck in the subacute stage of the dissection displayed a mural hematoma of the cervical ICA at the base of the skull in 7 of 9 patients. Typically, the hematoma was semilunar with an eccentric residual vessel lumen (Fig 5, left). Visualization was best on T2-weighted images because of the contrast of hyperintensive blood and perivascular hypointensive tissue. However, MR images did not provide relevant information about the actual hemodynamics.

Four of 5 MR angiograms were performed before recanalization (Fig 5, right). The angiographic features of the MR studies corresponded to the intra-arterial angiography (2 long dissections, 1 pseudo-occlusion). However, in 1 case, MR angiography demonstrated a tapering pseudo-occlusion a few centimeters distal to the bifurcation, whereas the angiogram revealed a subtotal ICA stenosis immediately proximal to the carotid canal.
Pulsations probably contributed to the reversed low-systolic and diastolic blood flow velocity. Eljamel was not severe enough to produce the high-resistance waveform and spectrum analysis reflected the hemodynamic effects of the extremely increased vascular resistance. Hennerici provided relevant information about the diagnostic capacity of these techniques and the significance of serial noninvasive follow-up studies for the history of the arterial disease. Characteristic sonographic features of ICAD have been identified as follows.

## Prognosis

Resolution of the ICAD could not be predicted from the patients' initial neurological presentation. Analysis of the clinical syndrome and the vascular outcome revealed no significant differences between patients with or without transient or permanent cerebral ischemic deficits (Table 3). The frequency of resolution ranged from 78% in patients with transient ischemic attack to 62% in stroke patients. Furthermore, recanalization apparently had no impact on the neurological prognosis. At the end of follow-up, 12 of 25 stroke patients had a disabling deficit, and 13 patients had a good or complete recovery. However, the frequency of recanalization was not different between both subgroups.

## Discussion

The systematic investigation of ICAD over more than 10 years using extracranial and transcranial Doppler sonography and ultrasound imaging techniques has provided relevant information about the diagnostic capacity of these techniques and the significance of serial noninvasive follow-up studies for the history of the arterial disease. Characteristic sonographic features of ICAD have been identified as follows.

1. A high-resistance Doppler signal with bidirectional low-amplitude flow components and absent diastolic flow was found in two thirds of the cases. Typically, the signal could be traced along the extracranial course of the ICA, thus allowing the distinction from stump flow in atherosclerotic carotid occlusion. Doppler waveform and spectrum analysis reflected the hemodynamic effects of the extremely increased vascular resistance caused by the mural hematoma, which produced a long segment luminal narrowing of the cervical carotid artery and frequently a circumscribed high-grade stenosis at the base of the skull. In addition, abnormal wall pulsations probably contributed to the reversed low-frequency components of the Doppler signal. Hennerici et al.17 found the high-resistance pattern in 76% of 22 ICADs, whereas the frequency of this characteristic Doppler signal in other series, in which ultrasound methods were used, is unknown because the extracranial Doppler findings were not described in detail.8,11,12

2. In some of our patients, the carotid obstruction was not severe enough to produce the high-resistance Doppler pattern but resulted only in a marked reduction of systolic and diastolic blood flow velocity. Eljamel et al.10 consistently found reduced flow velocity by Doppler in the CCA and ICA in all 10 dissections; however, it was not stated whether all the Doppler examinations were performed in the acute stage of the disease.

(3) In a few cases, a significantly increased Doppler shift frequency and turbulence in the upper cervical segment of the ICA indicating distal stenosis was best assessed by means of the 2-MHz pulsed-wave Doppler system of the TCD instrument. In studies in which the ultrasound examination was limited to conventional duplex scanning, distal segment stenoses were not detected.10,11

The high-resistance bidirectional flow pattern, significantly reduced flow velocities, and submandibular stenosis accounted for 90% of the abnormal Doppler results that suggested ICAD. In 10% of our cases, complete ICA occlusions and a high-grade stenosis at the level of the bifurcation caused by dissection could not be differentiated from obstructive atherosclerotic lesions based on the Doppler findings alone. In these patients, typical features of conventional duplex scanning or color flow imaging, as well as young age and clinical presentation with neck pain or oculosympathetic paresis, suggested the diagnosis of ICAD. However, there was no significant correlation between the initial Doppler sonographic features and the clinical presentation.

High-resolution gray-scale (B-mode) echotomography without Doppler spectrum analysis or superimposed color-coded Doppler signals had limited diagnostic value. Insolation of the bifurcation and the proximal ICA revealed no structural abnormalities in half of the cases, probably because the dissection frequently began in a more distal arterial segment that was difficult to visualize by ultrasound imaging techniques. DeBray et al.9 found various abnormalities in 78% of the echotomograms, whereas, corresponding to our experience, Sturzenegger13 emphasized that only typical abnormalities of the Doppler signal suggest the correct diagnosis in ICAD with normal B-mode scans.

In ICAD with abnormal gray-scale echotomogram, we found three characteristic features that have consistently been described in most previous studies:5,11,12,16 tapering vessel lumen in many cases, infrequent visualization of a dissection membrane, and absence of relevant atherosclerotic lesions. Pathologically, the different sonographic appearance of tapering lumen and dissec-

### Table 3. Initial Clinical Presentation of 48 Patients With Internal Carotid Artery Dissection and Neurological Outcome in 25 Stroke Patients Stratified for Sonographic Recanalization of the Dissection

<table>
<thead>
<tr>
<th>Clinical presentation</th>
<th>n</th>
<th>Recanalization, %</th>
<th>No Recanalization, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache/oculosympathetic paresis</td>
<td>9</td>
<td>67</td>
<td>33</td>
</tr>
<tr>
<td>Transient ischemic attack</td>
<td>14</td>
<td>78</td>
<td>22</td>
</tr>
<tr>
<td>Stroke</td>
<td>25</td>
<td>64</td>
<td>36†</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neurological outcome</th>
<th>n</th>
<th>Recanalization, %</th>
<th>No Recanalization, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>No/minor deficit</td>
<td>13</td>
<td>62</td>
<td>38*</td>
</tr>
<tr>
<td>Disabling deficit</td>
<td>12</td>
<td>67</td>
<td>33†</td>
</tr>
</tbody>
</table>

*Two patients included with short follow-up time caused by operation of carotid pseudoaneurysm.
†One patient included who died after 1 week from a large hemispheric infarct.
tion membrane probably depended on the variable proximal origin of the dissection and on the extension of the mural hematoma.

Recanalization of the dissection was assessed by Doppler sonography in more than two thirds of our cases. In Bogousslavsky's series, sequential Doppler tests demonstrated recanalization in 57% of the patients who survived the acute stroke. Partial or complete sonographic resolution was reported in 50% of the dissections in a French study. Surprisingly, the frequency of recanalization was higher (85% and 78%) in studies with angiographic follow-up, which may be due to selection bias for the repeat angiogram.

Because it is not possible to monitor the dynamic development of the dissection by means of intra-arterial angiography or MRI, Doppler sonography plays an important role in monitoring the disease until recanalization or final occlusion. The major impact of serial sonographic studies in ICAD is on diagnosis, but it may also be helpful in guiding patients' management decisions. On an empirical basis and according to suggestions in the literature, we have routinely treated patients with acute carotid dissection with full-dose intravenous heparin. If recanalization did not occur during hospitalization, warfarin (Coumadin) was started before discharge and continued for up to 6 months. After resolution of the dissection assessed by close-meshed Doppler studies, anticoagulation therapy was changed to aspirin. Because ICAD resolves after a variable period from symptom onset and anticoagulation has well-known potential risks, we suggest that clinical decisions be guided by Doppler follow-up examinations.

As we have shown in the present study, characteristic features of the Doppler spectrum, the gray-scale echotomogram, and color Doppler flow imaging provide diagnostic clues in the majority of patients. Because we have not seen a patient with acute onset of cerebrovascular or oculosympathetic symptoms and normal sonographic results who had features of ICAD on the angiogram, it is likely that most ICADs initially produce a hemodynamically significant obstruction. In a number of reports with angiographically documented ICADs that were either not stenotic or only moderately stenotic, the angiograms were not performed in the first days after symptom onset or the timing of the study was not given. Thus, initial pseudo-occlusion may have been missed. This corresponds to our finding that angiograms that demonstrated pseudo-occlusion were performed early within a mean interval of 8 days from symptom onset, whereas pseudoaneurysms without relevant stenosis on the angiogram were found after a mean interval of 22 days.

Visualization of the mural hematoma on axial MRI of the cervical carotid artery below the base of the skull further increases the reliability of the diagnosis based on noninvasive methods. Finally, in the near future, the combination of MR angiography of the carotid artery including the distal cervical and intracranial segments with the assessment of characteristic flow dynamics by duplex Doppler and the display of the vascular wall hemorrhage on MR scans will probably limit the use of conventional angiography to a few selected cases.

The neurological prognosis of patients with ICAD is controversial. The frequency of severe persistent deficits among our patients (25%) corresponds well to that in the patients (18%) with poor outcome in the French study and to the excellent or complete recovery in 85% of the patients studied by Mokri et al. In contrast, in Bogousslavsky's series, 7 of 30 patients died from large hemispheric infarcts; among the 23 survivors only 12 had a good recovery. This obvious discrepancy may be explained partially by selection bias because all patients from the Lausanne study had a stroke while patients with pure oculosympathetic paresis and transient ischemic attack were not included. In addition, the authors found that recanalization of the dissection was infrequent among patients with severe neurological deficits but was regularly assessed in patients with good outcome. In contrast, our data demonstrate that the reopening of the dissected artery apparently had no impact on the severity of residual neurological deficits.

References


Noninvasive monitoring of internal carotid artery dissection.
W Steinke, W Rautenberg, A Schwartz and M Hennerici

Stroke. 1994;25:998-1005
doi: 10.1161/01.STR.25.5.998

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 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/25/5/998

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/