CT Angiography and Doppler Sonography for Emergency Assessment in Acute Basilar Artery Ischemia

Tobias Brandt, MD; Michael Knauth, MD; Susanne Wildermuth, MD; Ralph Winter, MD; Rüdiger von Kummer, MD; Klaus Sartor, MD; Werner Hacke, MD

Background and Purpose—Both Doppler sonography (DS) and spiral CT angiography (CTA) are noninvasive vascular assessment tools with a high potential for application in acute cerebral ischemia. The usefulness of CTA for vascular diagnosis in acute basilar artery (BA) ischemia has not yet been studied.

Methods—We prospectively studied 19 patients (mean ± SD age, 58 ± 11 years) with clinically suspected acute BA occlusion by DS and CTA. Prior extracranial and transcranial DS was performed in all but 1 patient, with DS 4 hours after CTA. In 6 of 19 patients, we performed digital subtraction angiography.

Results—CTA was diagnostic in all but 1 patient. CTA revealed complete BA occlusion in 9 patients and incomplete BA occlusion with some residual flow in 2 patients. A patent BA was shown in 7 patients. Because of severe BA calcification, CTA results were inconclusive in 1 patient. DS was diagnostic in only 7 of 19 patients, indicating certain BA occlusion in 3 patients and BA patency in 4 patients. In an additional 9 patients, the results of DS were inconclusive. DS was false-negative in 2 patients with distal BA occlusion shown by CTA and digital subtraction angiography. In 1 patient with DS performed after CTA, recanalization was demonstrated. In addition to the diagnosis or exclusion of BA occlusion, CTA provided information on the exact site and length of BA occlusion and collateral pathways. In our series, CTA results prompted indication for intra-arterial thrombolysis in 5 patients.

Conclusions—CTA was superior to DS in the assessment of BA patency in patients with the syndrome of acute BA ischemia in terms of feasibility and conclusiveness, particularly in cases with distal BA occlusion. Our study confirmed the usefulness of combined extracranial and transcranial DS in the diagnosis and exclusion of proximal BA occlusion. (Stroke. 1999;30:606-612.)

Key Words: angiography, computed tomographic • basilar artery • brain stem • cerebral ischemia • ultrasonography, Doppler

Acute basilar artery occlusion (BAO) is a frequent but often difficult differential diagnosis in patients with rapid onset of decreasing consciousness and progressive brain stem dysfunction. Intra-arterial thrombolytic therapy is a potentially lifesaving procedure in selected cases of acute BAO.1,2 Therefore, reliable and widely available tools for the assessment of basilar artery (BA) patency are needed. Traditionally, intra-arterial digital subtraction angiography (DSA) has been used for diagnosis of clinically suspected BAO.3 However, DSA is a time-consuming, expensive, and invasive method that requires good cooperation of the patient or general anesthesia. Extracranial and intracranial Doppler sonography (DS) has been used to confirm or exclude suspected acute BAO.4-6 Unfortunately, considerable technical limitations of DS, especially in the diagnosis of BAO, could not be overcome until now.4-7 In particular, ultrasound penetration of the distal parts of the BA or in cases of adipose necks is most often insufficient, and identification of the BA may remain uncertain.8 The value of MR angiography for diagnosis of BAO has not been studied thus far in large series of patients. Generally, MR angiography is neither well suitable nor accessible for emergency workup of unstable or intubated patients with acute cerebral ischemia. Moreover, examination of patients with impaired consciousness is frequently hampered by motion artifacts.9,10

Spiral CT angiography (CTA) is a new noninvasive vascular imaging tool with a high potential for application in acute cerebral ischemia.9,11-15 The usefulness of CTA for diagnosis or exclusion of BAO has been shown in only a few cases.16 To our knowledge, studies comparing CTA with DS for this purpose have not been published.

Subjects and Methods

Among 20 patients prospectively included for CTA examination of suspected BAO, 19 also underwent DS. All patients were admitted consecutively to the emergency ward of our department from

Received May 26, 1998; final revision received December 2, 1998; accepted December 2, 1998.
From the Departments of Neurology (T.B., S.W., R.W., W.H.) and Neuroradiology (M.K., R. von K., K.S.), University of Heidelberg, Heidelberg, and the Technische Universität, Dresden (R. von K.), Germany.
Correspondence to Tobias Brandt, MD, Neurologische Universitätsklinik, Im Neuenheimer Feld 400, D 69120 Heidelberg, Germany. E-mail tobias_brandt@ukl.uni-heidelberg.de
© 1999 American Heart Association, Inc.

Stroke is available at http://www.strokeaha.org
Doppler Sonography, CT Angiography, and Digital Subtraction Angiography in Patients Suspected of Acute Basilar Artery Occlusion

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Coma</th>
<th>Intubated</th>
<th>DS</th>
<th>Site of Occlusion</th>
<th>Length</th>
<th>Collateralization</th>
<th>DSA</th>
<th>Therapy</th>
<th>Final Diagnosis, CCT or MRI on Follow-Up</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>+</td>
<td>+</td>
<td>Uncertain (TCD): BA diastolic flow reduction</td>
<td>Negative</td>
<td>...</td>
<td>...</td>
<td>ND</td>
<td>...</td>
<td>Postictal coma, old MCA infarct</td>
<td>Good</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>Uncertain (TCD): BA until 90 mm normal</td>
<td>Distal occlusion</td>
<td>Short</td>
<td>Good: PcoA</td>
<td>Short distal BAO; VA dissection; cerebellar collateralization</td>
<td>Thrombolysis; RFA</td>
<td>BA thrombosis, pontine and cerebellar infarcts</td>
<td>Moderate</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>-</td>
<td>Uncertain: VA occlusion (ECD), BA not identified (TCD)</td>
<td>Negative: no VA occlusion</td>
<td>...</td>
<td>...</td>
<td>ND</td>
<td>Heparin</td>
<td>Large MCA infarct</td>
<td>Poor</td>
</tr>
<tr>
<td>4</td>
<td>-</td>
<td>-</td>
<td>Uncertain (TCD): BA until 90 mm normal</td>
<td>Negative</td>
<td></td>
<td></td>
<td>ND</td>
<td>Heparin</td>
<td>Brain stem TIA, ND</td>
<td>Good</td>
</tr>
<tr>
<td>5</td>
<td>-</td>
<td>-</td>
<td>Uncertain: VA hypoplasia (ECD), BA flow reduction (TCD)</td>
<td>Incomplete thrombosis of terminus technicus BA</td>
<td>Short</td>
<td>...</td>
<td>ND</td>
<td>Ticlopidine</td>
<td>Partial BA thrombosis, small pontine infarct</td>
<td>Good</td>
</tr>
<tr>
<td>6</td>
<td>+</td>
<td>+</td>
<td>Positive: bilateral VA occlusion (ECD), BA high resistance (100 mm), PCA hypoperfusion (TCD)</td>
<td>Proximal vertebrobasilar occlusion; local calcification</td>
<td>Short</td>
<td>Poor</td>
<td>ND (prior infarcts)</td>
<td>Heparin</td>
<td>BA thrombosis, lower brain stem infarct</td>
<td>Died</td>
</tr>
<tr>
<td>7</td>
<td>-</td>
<td>-</td>
<td>Certain: negative</td>
<td>Negative</td>
<td>...</td>
<td>...</td>
<td>ND</td>
<td>Heparin</td>
<td>Lower brain stem infarct</td>
<td>Moderate</td>
</tr>
<tr>
<td>8</td>
<td>-</td>
<td>-</td>
<td>Bilateral VA occlusion (ECD); BA diastolic flow reduction (TCD)</td>
<td>Bilateral VA occlusion, distal BAO</td>
<td>Short</td>
<td>...</td>
<td>Bilateral VA occlusion distal short BAO</td>
<td>Heparin (decomarted infarcts)</td>
<td>BA thrombosis, thalamic and cerebellar infarcts</td>
<td>Moderate</td>
</tr>
<tr>
<td>9</td>
<td>-</td>
<td>-</td>
<td>Positive: bilateral VA resistance (ECD); BA high resistance (100 mm) (TCD)</td>
<td>Middle BAO</td>
<td>Short</td>
<td>Good: retrograde</td>
<td>ND (pontine infarct, minor deficits)</td>
<td>Heparin</td>
<td>BA thrombosis, pontine infarct</td>
<td>Moderate</td>
</tr>
<tr>
<td>10</td>
<td>+</td>
<td>+</td>
<td>Technically insufficient: no flow signals in bilateral VA and BA detectable</td>
<td>Middle and distal BAO</td>
<td>Long</td>
<td>Poor</td>
<td>Middle and distal BAO</td>
<td>Thrombolysis: RFA</td>
<td>BA thrombosis, multiple brain stem infarcts, ICB</td>
<td>Died</td>
</tr>
<tr>
<td>11</td>
<td>-</td>
<td>-</td>
<td>Negative: BA until 100 mm normal (TCD)</td>
<td>Incomplete distal BAO</td>
<td>Short</td>
<td>...</td>
<td>ND (PCA infarct)</td>
<td>Heparin</td>
<td>Partial BA thrombosis, bilateral PCA infarcts</td>
<td>Moderate</td>
</tr>
<tr>
<td>12</td>
<td>-</td>
<td>-</td>
<td>Certain: negative</td>
<td>Negative</td>
<td></td>
<td></td>
<td>ND</td>
<td>Heparin</td>
<td>Brain stem TIA</td>
<td>Good</td>
</tr>
<tr>
<td>13</td>
<td>-</td>
<td>-</td>
<td>Uncertain: VA hypoplasia (ECD); BA not identified, PCA hypoperfusion (TCD)</td>
<td>Middle and distal BAO</td>
<td>Long</td>
<td>Poor: intercerebellar</td>
<td>Middle BAO</td>
<td>Thrombolysis</td>
<td>BA thrombosis RFA, thalamic and cerebellar infarcts</td>
<td>Good</td>
</tr>
<tr>
<td>14</td>
<td>+</td>
<td>+</td>
<td>Uncertain: BA not identified</td>
<td>Distal BAO, proximal MCA occlusion</td>
<td>Short</td>
<td>Good retrograde: PcoA</td>
<td>Distal BA+proximal MCA occlusion</td>
<td>Thrombolysis: partial RFA</td>
<td>Embolic BA and MCA occlusions; cerebellar, PCA, and MCA infarcts</td>
<td>Died</td>
</tr>
<tr>
<td>15</td>
<td>+</td>
<td>+</td>
<td>Uncertain: BA not identified</td>
<td>Vertebrobasilar occlusion</td>
<td>Long</td>
<td>Good</td>
<td>Vertebrobasilar occlusion</td>
<td>Thrombolysis: RFA (on follow-up CTA)</td>
<td>BA thrombosis, cerebellar and pontine infarcts</td>
<td>Moderate</td>
</tr>
<tr>
<td>16</td>
<td>+</td>
<td>-</td>
<td>Certain: negative</td>
<td>Negative</td>
<td></td>
<td></td>
<td>ND</td>
<td>Heparin</td>
<td>Transient BA thrombosis, pontine and bilateral thalamic infarcts</td>
<td>Died</td>
</tr>
<tr>
<td>17</td>
<td>-</td>
<td>-</td>
<td>ND before CTA, 4 h after CTA; normal TCD indicating recanalization of BAO</td>
<td>Distal BAO and PCA occlusion</td>
<td>Short</td>
<td>...</td>
<td>ND (age, infarct)</td>
<td>Heparin</td>
<td>BA thrombosis, thalamic and PCA infarcts</td>
<td>Moderate</td>
</tr>
<tr>
<td>18</td>
<td>+</td>
<td>+</td>
<td>Certain: negative</td>
<td>Negative</td>
<td></td>
<td></td>
<td>ND</td>
<td></td>
<td>Intoxication, no infarct</td>
<td>Good</td>
</tr>
</tbody>
</table>
December 1995 until February 1997. CTA findings of 5 patients have been published earlier by Knauth et al.16 This study refers to those 19 patients, aged 23 to 86 years (mean 611 years), who underwent both CTA and DS. Clinical diagnosis was probable acute BAO in all cases. Inclusion criteria were variable clinical syndromes with sudden onset and progressive deterioration of consciousness, dizziness, double vision, dysarthria, oculomotor and other cranial nerve signs, or bilateral motor signs. Three patients were comatose. In 6 additional patients already ventilated on admission, clinical assessment was initially not possible.

Within a maximum of 30 minutes before CTA, we performed extracranial Doppler sonography (ECD) and transcranial Doppler sonography (TCD) in all but 1 patient according to standard procedures (Medasonics CDS; extracranial 4-MHz probe with continuous wave mode, transcranial 2-MHz probe with pulsed wave mode).8 One patient with BAO shown by CTA was excluded from the analysis because DS was not available at admission, and he was transferred to a secondary care center because of poor prognosis. In the remaining patient with BAO, DS was unavailable before CTA. This patient was treated by local thrombolysis and underwent DS 4 hours after CTA to assess recanalization. We classified the findings on ECD of both extracranial distal vertebral artery segments (V3 segments) and on TCD of the intracranial distal vertebral artery segments (V4 segments), BA, and both posterior cerebral arteries (PCA) using the suboccipital and temporal approaches into 3 categories: (1) no evidence of BAO: normal BA flow signal with well-preserved diastolic forward flow at a depth of 95 mm detectable by TCD; (2) uncertain signs of BAO: absent flow or high-resistance flow patterns in both vertebral artery V3 segments in ECD; no flow signal or flow signal without forward diastolic flow component detectable in the BA by TCD; diminished PCA flow without evidence of PCA stenosis; and (3) evidence of BAO: high-resistance to-and-fro flow patterns at depths of 85 to 95 mm in the BA (TCD), possibly combined with a sudden loss of flow signals on increasing the examination depth, or demonstration of retrograde flow in the distal BA.6,18

Immediately after conventional CT scanning (4-mm slice thickness for the posterior fossa), all patients had CTA of the vertebrobasilar circulation, after informed consent had been obtained from patients or their close relatives. Exclusion criteria for CTA were contrast medium allergy and renal failure.

For CTA of the main vertebrobasilar trunks from the foramen magnum to the top of the BA, including the circle of Willis, we used the method described by Knauth et al:16 130 mL of a nonionic contrast medium (Omnipaque, Schering) was infused into an antecubital vein (>18-gauge cannula) with an injection rate of 4 to 5 mL/s using an injection pump. After a delay of 20 seconds, spiral scanning was done (Picker PQ 2000 CT, Picker International). The following parameters were used in all patients: slice thickness 2.0 mm, index 1.5 mm, spiral pitch 1.25 mm, total scanning time 21 seconds with 1 second per revolution, 130 kV, and 125 mA. For vascular diagnoses we used the CTA source images and 3-dimensional reconstructions of the data sets (surface-shaded display; voxel Q workstation; Picker International). Data sets were

![Figure 1](http://stroke.ahajournals.org/)

**Figure 1.** Calcification as technical limitation of CTA in diagnosis of BAO. Precontrast admission CT (top) shows dense calcification of the BA. Differentiation of the massive calcification and any possible contrast enhancement in CTA (bottom) in the BA is not possible.
completed within 10 minutes after the start of scanning. Analysis of CTA data sets was performed by a neuroradiologist (M.K.) blinded to clinical data and prior DS results. The interrater reliability of the analysis of CTA data sets was assessed in a previous study and found to be high.13

For the description of the sites of segmental BAO, we used the definitions suggested by Archer and Horenstein19: (1) proximal BAO included the lowest BA segment from the vertebral artery junction up to the origin of the anterior inferior cerebellar arteries; (2) mid BAO included the segment from the origin of the anterior inferior cerebellar arteries to the origin of the superior cerebellar arteries; and (3) distal BAO included the top of the BA distally to the origin of the superior cerebellar arteries. On the basis of CTA findings, collateral supply of the distal BA via posterior communicating arteries and the circle of Willis was graded as “good” if it was clearly visible or “poor” if it was not detectable.20 Length of the occlusion was graded as “short” if only 1 BA segment was occluded and as “long” if ≥2 segments were affected. The latter definition corresponds to the category “extended” or “multilevel” occlusion given by Hacke and coworkers.1

In 6 patients without contraindications for intra-arterial thrombolytic therapy, we additionally performed conventional intra-arterial angiography (DSA), including internal carotid artery injections for visualization of collateral flow to the distal BA in 2 patients.

Results

In all 19 patients included in the study (Table), technical feasibility of CTA was good, with high-quality data sets. Site and length of BAO could be assessed by CTA in all but 1 patient. In this case, with evidence of BAO at DS, CTA was not diagnostic because of lack of differentiation of the massive calcification and any possible contrast enhancement in the BA (Figure 1).

CTA indicated complete BAO in 9 patients (Figure 2). CTA revealed short segmental BAO in 6 patients and multisegmental BAO in 3 patients. The proximal BA segment

Figure 2. Comparison of CTA and DSA in BAO. A, Source CT without contrast filling in the proximal and middle segments of the BA. B, CTA reconstruction demonstrating proximal to mid BAO (left) with corresponding DSA (right).
was occluded in 2 patients, the mid BA in 4, and the distal BA segment in 3 patients. Good collateral supply with retrograde filling of the distal BA from the anterior circulation via the posterior communicating arteries was visualized in 3 patients with proximal or middle BA segment occlusion, while collateral supply of the PCA was seen in another 2 patients with distal BAO. In another patient with long mid to distal BAO, CTA identified collateral SCA supply confirmed by DSA. In 1 patient with complete BAO, CTA additionally revealed left middle cerebral artery occlusion. In an additional 10 patients, CTA revealed extremely reduced BA contrast enhancement on 2 of the original slices, indicating incomplete occlusion. In 7 patients, normal CTA findings clearly excluded BAO (Figure 3). Direct and indirect Doppler findings indicating BAO are shown in Figure 4.

In 6 patients with CTA-based diagnosis of BAO and possible indication for thrombolytic therapy, DSA was performed. It confirmed BAO without exception. Important additional DSA findings not visualized by CTA, however, were extracranial vertebral artery dissection and anterograde cerebellar collaterals in 2 patients.

DS was diagnostic in 7 of 19 patients. Clear evidence of BAO, however, was demonstrated only in 3 patients with proximal or mid BAO according to CTA. DS correctly excluded BAO in 4 patients. In the remaining patients, signs of BAO were uncertain, with BA flow completely undetectable in 3 patients or absent in diastole, corresponding to a high-resistance flow pattern in 2 patients. Additional PCA hypoperfusion confirmed the assumption of BAO in 1 of these patients. DS was false-negative in 2 patients, 1 with complete and 1 with subtotal distal BAO on CTA and DSA. In 1 of these patients, follow-up DS revealed findings typical of BAO. DS findings indicated vertebral artery occlusion not confirmed by CTA in another patient. Results of DS were inconclusive in an additional 4 patients, 2 of whom were intubated and hyperventilated. In 1 of these patients, DS was technically insufficient, with no flow signal detectable in the vertebral arteries and the BA. One patient with BAO had no DS before CTA and DSA because of unavailability of DS but showed normal DS results 4 hours later, indicating recanalization after thrombolysis.

New infarcts in the posterior circulation on follow-up CT consistent with transient BAO were shown in 3 of 7 patients without BAO on CTA. Other final single diagnoses were transient brain stem ischemia of unknown origin, intoxication, large middle cerebral artery territory infarction, and postictal coma.

Intra-arterial thrombolytic therapy was performed in 5 patients with BAO. Three of 4 patients with successful recanalization survived. One patient without recanalization died.

Discussion

In patients with acute brain stem symptoms and negative cranial CT, vascular evaluation may provide important information concerning a possible occlusive ischemic etiology. Successful thrombolytic therapy with recanalization of BAO is associated with improved survival of progressive brain stem ischemia in selected cases.\(^1\)\(^2\) Therefore, rapid and reliable assessment of BA patency is mandatory in cases of clinically suspected BAO without contraindications for thrombolysis. While the validity of DSA for the diagnosis and exclusion of BAO is beyond question, attempts are made to confine it to patients undergoing intra-arterial thrombolytic therapy. The main reasons are its hazards, limited availability, high costs, and time consumption. To select patients for thrombolytic therapy as well as to assess patients who are not suitable for this invasive therapy, other methods of BA patency assessment are needed. In patients with contraindications for thrombolysis, DSA is not necessary in the acute stage because of lack of therapeutic consequences. Confirmation of the clinical diagnosis of BA thrombosis in these patients, however, is important for the assessment of further prognosis and for clinical decision making.
DS has become a standard vascular assessment tool. Unfortunately, technical problems limit the validity of DS, especially for the diagnosis and exclusion of BAO.\textsuperscript{5,7,8,18} Several studies confirmed the diagnostic value of some signs clearly indicating BAO, especially bilateral high-resistance flow patterns in the distal vertebral arteries and the proximal BA.\textsuperscript{5–7,18} In our series, these signs were identified by DS in only 3 of 9 patients with proximal complete BAO. DS correctly excluded BAO in 4 patients. In 2 patients with distal BA occlusions, however, DS findings were clearly false-negative. Therefore, the decision not to perform DSA is not to be based reliably on negative DS findings alone if BAO is clinically suspected. More recent technical developments, such as transcranial duplex sonography with application of contrast agents, have not been tested in large series of patients with BAO. Some case reports, however, indicate an improvement of visualization of the proximal and middle segments of the BA, whereas visualization of the distal parts still remained technically insufficient.\textsuperscript{21,22} The ability of DS to provide information on flow dynamics, however, and its usefulness for repeated flow monitoring are advantages that may be used for therapy and follow-up. Once the exact diagnosis has been defined, even uncertain DS signs of BAO may be useful for this purpose.

![Figure 4](http://stroke.ahajournals.org/)

**Figure 4.** Direct and indirect Doppler findings indicating BAO. a, High-resistance pattern in the distal extracranial vertebral arteries (V3 segments) (ECD). b, Orthograde high-resistance pattern in the proximal BA (95-mm depth) (TCD). c, Retrograde high-resistance pattern in the distal BA (105-mm depth) (TCD). d, Hypoperfusion in bilateral PCA (TCD).
CTA has been introduced as a new tool for emergency vascular assessment.\textsuperscript{9,11,15} It has been reported to more reliably depict the normal anatomy of the circle of Willis than MR angiography.\textsuperscript{8} In this series, CTA proved to be a feasible and rapid technique for evaluation of the posterior circulation, even in severely ill patients. Definite diagnosis or exclusion of BAO was possible in all but 1 patient. In all patients in whom DSA was performed, CTA-based diagnosis of BAO was confirmed without exception. CTA showed collateral supply of the distal BA in 6 patients with proximal BAO. The site and length of BAO and the collateral status have been shown to have an important impact on prognosis.\textsuperscript{2} This information was provided by CTA in all but 1 patient. With DSA, additional carotid artery injections are necessary to assess the length of BAO and the retrograde collateral flow.

The important drawbacks of CTA are its narrow scanning range, showing only selected parts of the vascular tree, its limited repeatability, and the low yield of dynamic flow information.\textsuperscript{9,14,23} The latter may be an explanation for the limited repeatability, and the low yield of dynamic flow range, showing only selected parts of the vascular tree, its assessment of the length of BAO and the retrograde collateral flow. DSA, additional carotid artery injections are necessary to completely occluded on CTA has not been determined thus far.\textsuperscript{11}

All 3 modes of vascular examination may be hampered by motion artifacts. In our series, only the CTA without diagnostic value was obscured by BA atherosclerosis with massive calcification. Flow-related examinations such as DSA and DS are probably influenced to a lesser extent by vessel wall calcification.

We conclude that CTA is a useful tool for the emergency evaluation of patients with suspected acute BAO. In our series it provided reliable information on BA patency, on the exact site and extent of BAO, and to some extent on collateral pathways. It appears reasonable to rely on CTA results in decisions concerning thrombolytic therapy in clinically suspected BAO. Although DSA was superior to CTA in depicting relevant vascular details such as collateral flow through cerebellar arteries. Whether high-grade BA stenoses may appear completely occluded on CTA has not been determined thus far.\textsuperscript{11}

References
CT Angiography and Doppler Sonography for Emergency Assessment in Acute Basilar Artery Ischemia

Tobias Brandt, Michael Knauth, Susanne Wildermuth, Ralph Winter, Rüdiger von Kummer, Klaus Sartor and Werner Hacke

Stroke. 1999;30:606-612
doi: 10.1161/01.STR.30.3.606

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1999 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/30/3/606

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/