Transcranial Color-Coded Duplex Sonography in Cerebral Aneurysms

Ralf W. Baumgartner, MD; Heinrich P. Mattle, MD; Karl Kothbauer, MD; Gerhard Schroth, MD

Background and Purpose Diagnosis and successful therapy before rupture of cerebral aneurysms would be most desirable in view of the high mortality and morbidity rates of aneurysmal subarachnoid hemorrhage. Using transcranial color-coded duplex sonography, we studied radiologically proven cerebral aneurysms to define ultrasonographic criteria and sensitivity for their diagnosis and detection.

Methods Twenty-nine consecutive patients with 30 radiologically proven cerebral aneurysms (16 women and 13 men; mean age±SD, 52±16 years; range, 15 to 76 years) with 30 CAs identified by cerebral angiography (CT), magnetic resonance imaging (MRI), or both that were seen in our neurovascular laboratory, traditionally blue is assigned for flow toward the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.

All TCCD examinations were performed by the same examiner (R.W.B.). The sonographer knew cerebral CT and MRI findings but was blinded to the results of cerebral angiography. The intraluminal size of CAs was determined in two-dimensional transsections by measuring the maximal extent of the color-coded blood flow information ("flow lumen").9-11 In addition, the sonographer tried to identify the neck, the wall, thrombosed parts, and the parent artery.

For arterial vasospasm and stenosis, the fastest maximal systolic (V1) and maximal end-diastolic (V2) blood velocities were measured. The energy output had a maximal in situ intensity of 262 mW/cm² I-SPTA (spatial peak time average intensity), corresponding to 120 W/cm² I-SPPA (spatial peak pulse average intensity). In our laboratory, traditionally blue is assigned for flow toward and red for flow away from the transducer. Deep shades indicate slow mean blood velocities; lighter shades or a change from blue to green (for flow toward the transducer) and from red to yellow (for flow away from the transducer) indicate fast mean blood velocities. The axial extension of the sample volume for the measurement of blood velocities was as small as possible and ranged between 1.5 and 2.5 mm. With the patient in a supine position, the internal carotid and anterior, middle, and posterior cerebral arteries (ICA, ACA, MCA, and PCA, respectively) and the tip of the basilar artery (BA) were visualized through the temporal window. Axial and sagittal cross sections were acquired from both sides. With the patient in a sitting position, the vertebral artery (VA) and lower parts of the BA were imaged through the occipital foramen. Axial and sagittal cross sections were performed for all foramen positions.
TABLE 1. Neurological Symptoms and Signs, Cerebral CT, MRI, and Angiographic Findings, and TCCD Diameters of “Flow Lumen” in 30 Cerebral Aneurysms

<table>
<thead>
<tr>
<th>Patient</th>
<th>Aneurysm</th>
<th>Asymptomatic</th>
<th>CN Deficit</th>
<th>SAH</th>
<th>Hematoma</th>
<th>Thrombosis</th>
<th>Diameter, mm</th>
<th>Diameter, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ICA C4</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>16.1</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>ICA OphA</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
<td>25.3</td>
<td>26.9</td>
</tr>
<tr>
<td>3</td>
<td>ICA OphA</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>8.1</td>
<td>9.2</td>
</tr>
<tr>
<td>4</td>
<td>ICA PCoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i,c</td>
<td></td>
<td>15.5</td>
<td>14.8</td>
</tr>
<tr>
<td>5</td>
<td>ICA PCoA</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
<td>7.3</td>
<td>6.5</td>
</tr>
<tr>
<td>6</td>
<td>ICA PCoA</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
<td>20.5</td>
<td>10.9</td>
</tr>
<tr>
<td>7</td>
<td>ICA PCoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>10.0</td>
<td>9.0</td>
</tr>
<tr>
<td>8</td>
<td>ICA PCoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i</td>
<td>+</td>
<td>18.8</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>ICA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,c</td>
<td>-</td>
<td>6.0</td>
<td>6.3</td>
</tr>
<tr>
<td>10</td>
<td>ICA Bif</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>8.3</td>
<td>8.7</td>
</tr>
<tr>
<td>11</td>
<td>ICA Bif</td>
<td>-</td>
<td>a</td>
<td>-</td>
<td>-</td>
<td></td>
<td>9.8</td>
<td>9.5</td>
</tr>
<tr>
<td>12</td>
<td>ICA Bif</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>16.6</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>ICA Bif</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>7.2</td>
<td>7.7</td>
</tr>
<tr>
<td>14</td>
<td>ACoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i,c</td>
<td>-</td>
<td>8.4</td>
<td>8.9</td>
</tr>
<tr>
<td>15</td>
<td>ACoA</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>20.0</td>
<td>18.2</td>
</tr>
<tr>
<td>16</td>
<td>ACoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>4.1</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>ACoA</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>6.4</td>
<td>6.0</td>
</tr>
<tr>
<td>18</td>
<td>ACoA</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>4.4</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,c</td>
<td>+*</td>
<td>10.2</td>
<td>11.9</td>
</tr>
<tr>
<td>20</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i</td>
<td>+*</td>
<td>14.7</td>
<td>15.2</td>
</tr>
<tr>
<td>21</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>8.3</td>
<td>7.7</td>
</tr>
<tr>
<td>22</td>
<td>MCA Bif</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>10.1</td>
<td>9.5</td>
</tr>
<tr>
<td>23</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i,c</td>
<td>-</td>
<td>13.0</td>
<td>12.4</td>
</tr>
<tr>
<td>24</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>6.5</td>
<td>7.1</td>
</tr>
<tr>
<td>25</td>
<td>PCA (P3)</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i,c</td>
<td>-</td>
<td>5.1</td>
<td>-</td>
</tr>
<tr>
<td>26</td>
<td>BA Tip</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7.0</td>
<td>-</td>
</tr>
<tr>
<td>27</td>
<td>BA Tip</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s,i</td>
<td>-</td>
<td>10.3</td>
<td>10.9</td>
</tr>
<tr>
<td>28</td>
<td>BA Tip</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s</td>
<td>-</td>
<td>11.8</td>
<td>12.8</td>
</tr>
<tr>
<td>29</td>
<td>BA Proximal</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>12.6</td>
<td>13.4</td>
</tr>
</tbody>
</table>

CT indicates computed tomography; MRI, magnetic resonance imaging; TCCD, transcranial color-coded duplex sonography; CN, cranial nerve; SAH, subarachnoid hemorrhage; ICA, internal carotid artery; C4, juxtasellar segment of ICA; OphA, ophthalmic artery; PCoA, posterior communicating artery; Bif, bifurcation; ACoA, anterior communicating artery; PCA, posterior cerebral artery; BA, basilar artery; +, yes; -, no; s, subarachnoid; i, intraventricular hemorrhage; c, intracerebral hemorrhage; and a, aneurysm embolism with transient ischemic attack.

*Partially thrombosed.

were determined in the ACA, MCA, PCA, BA, and VA to detect vasospasm and stenoses that might influence aneurysmal hemodynamics. Vasospasm was diagnosed when \( V_r \) and \( V_d \) in patients with SAH were >2 SD above the reference values. Stenosis was diagnosed when \( V_r \) and \( V_d \) in patients without SAH were >2 SD above the reference values.

Reference values were obtained from 104 healthy volunteers (57 women, 47 men) with a mean age±SD of 45±18 years (range, 18 to 86 years). The volunteers had no cerebrovascular risk factors and no history of neurological disease.

All patients underwent cerebral CT; in addition, 13 patients underwent cerebral MRI. All patients, excepting patient 4, were examined with cerebral intra-arterial digital subtraction angiography. CT was used as well as MRI, if available, to determine aneurysm size. Maximal intraluminal diameters were assessed in patent CAs. In thrombosed CAs, maximal aneurysm diameters were determined.

**Results**

Table 1 summarizes CT, MRI, angiographic, and TCCD findings in all CAs studied.

TCCD detected 23 of 27 (85%) nonthrombosed CAs with a diameter ranging from 6 to 25 mm. Four nonthrombosed CAs with a mean intraluminal diameter of 5.2 mm were not visualized. In addition, three thrombosed CAs were missed; in one of these patients, TCCD study could not be performed because of a thick...
Fig 1. Aneurysm of the upper basilar artery bifurcation (patient 27) is shown in serial axial computed tomographic scans with intravenous contrast medium infusion (A). B through D, Transtemporal sonations with axial (B left, C left) and coronal (D) scanning planes through the base (B left) and the middle of the aneurysm (C left, D). B right and C right, schemes corresponding to B left and C left, respectively.
temporal bone. Ultrasonographic visualization of the intracranial arteries was good in 20 patients (69%), average in 8 patients (28%), and poor in 1 patient (3%).

The measured TCCD values for the intraluminal diameter were $-0.1\pm 12.7\%$ (mean $\pm$ SD; range, $-46.8\%$ to $16.7\%$) lower than the values obtained by CT and MRI. The neck was identified in three of 27 (11%) nonthrombosed CAs (Fig 2). The walls and thrombosed parts were missed.

Typical TCCD findings in CAs (Figs 1 and 2) were as follows: (1) Scanning planes that transected approximately at the middle of CAs always delineated a round or oval mass that was divided by a “separation zone” into blue and red areas. (2) The “separation zone” was medially or paramedially located, was straight or oval, and contained dark and/or no colors. Its location and orientation depended on the scanning plane. (3) Peripheral scanning planes showed a monochromatic picture. (4) No turbulence was detected. (5) Flow patterns showed no spontaneous fluctuations.

For vasospasm/stenosis, the delay of the TCCD examination in patients with SAH after the beginning of symptoms was $51\pm 44$ hours (mean $\pm$ SD). The delay between TCCD and angiographic studies was $10\pm 8$
rysmal blood flow depends on different anatomic fac-

centrally or paracentrally located and was characterized
the aneurysm and the axis of the parent artery, the area
zones, indicating blood flow toward and away from the

patients with unruptured CAs remains controversial. 6-23

vasospasm in the ipsilateral ACA (V_s/V_d= 148/56 cm/s)

transducer, respectively. (2) The separation zone was
delineated using TCCD. An alteration of the dimension
of insonation by 90° changed the orientation of the
separation zone. Therefore, its peripheral parts must be
artifacts resulting from an insonation angle reaching
90°, and only the central meeting point of two separa-
tion zones corresponds to an area with slow or unde-
tectable blood flow. This observation confirms the find-

hours (mean±SD). Patient 9 showed signs of mild
vasospasm in the ipsilateral ACA (V_s/V_d=148/56 cm/s)
and MCA (V_s/V_d=181/94 cm/s). Patient 22 showed
signs of a mild stenosis in the ipsilateral ACA (V_s/
V_d=136/44 cm/s) and MCA (V_s/V_d=185/78 cm/s).
These TCCD findings were confirmed by angiography.
The reference values for V_s and V_d in the ACA,
MCA, PCA, BA, and VA are given in Table 2.

Fourteen CAs (patients 5, 9, 10, 13, 14, 16, 17, 19
through 24, and 26) were surgically treated (13 clippings,
1 wrapping), five CAs were embolized with
Guglielmi detachable coils (patients 3, 7, 25, 27, and
28), and one CA (patient 2) was occluded with
a balloon. Ten CAs (patients 1, 4, 6, 8, 11, 12, 15, 18,
and 29) were left untreated. Patients 4 and 8 died because of
the SAH.

Discussion

CAs occur in about 1% of the general population,
although their frequency ranges in various postmortem
series from 0.2% to 9.0%.12,13 The annual rupture rate
of CAs is 1% to 2% per year and seems to remain
almost constant from decade to decade.24-27 Only about
50% of patients with aneurysmal SAH have a favorable
outcome.14-17 In most surgical series, clipping of unrup-
tured CAs is associated with 0% mortality,18-21 and the
morbidity rate does not exceed 5%.22,23 Although it is
likely that these figures represent an improvement over
the natural history of these lesions, the management of
patients with unruptured CAs remains controversial.6,23
At the present time, there is no cost-effective method to
screen the general population for unruptured CAs.23
TCCD is a new ultrasonographic method that might
refine and ultimately replace transcranial Doppler
sonography for the study of the intracranial circulation.
Therefore, additional examination for unruptured CAs
during routine TCCD studies might prove useful.
In our search for reliable criteria for ultrasonographic
diagnosis, the following five TCCD findings were deline-
ated: (1) In central scanning planes, a circular or oval
mass was divided by a separation zone into blue and red
zones, indicating blood flow toward and away from the
transducer, respectively. (2) The separation zone was
centrally or paracentrally located and was characterized
by dark or no color-coded Doppler signals. Intra-aneu-
rysmal blood flow depends on different anatomic fac-
tors, such as the angle between the downstream wall of
the aneurysm and the axis of the parent artery, the area
and location of the orifice, the configuration and axis of
the aneurysm, and the diameter of the fundus.24-27
Thus, the direction of intra-aneurysmal blood flow is
not predictable using TCCD because anatomic details
of CAs are not visualized in the B-mode image. In
Doppler sonography, the measured blood velocity (V_d)
depends on the cosine value of the insonation angle β
according to the formula V_d=V_s*cos β, where V_s
represents the real blood velocity.28 Since the direction
of blood flow within CAs and thus the angle of in-
sonation are not known, intra-aneurysmal V_s cannot be
delineated using TCCD. An alteration of the dimension
of insonation by 90° changed the orientation of the
separation zone. Therefore, its peripheral parts must be
artifacts resulting from an insonation angle reaching
90°, and only the central meeting point of two separa-
tion zones corresponds to an area with slow or unde-
tectable blood flow. This observation confirms the find-

V_s indicates systolic blood velocity; V_d, end-diastolic
blood velocity; ACA, anterior cerebral artery; MCA, middle
cerebral artery; PCA, posterior cerebral artery; VA, vertebral artery; and
BA, basilar artery. Volunteers included 57 women and 47 men
with a mean age±SD of 45±18 years.

Discussion

CAs occur in about 1% of the general population,
although their frequency ranges in various postmortem
series from 0.2% to 9.0%.12,13 The annual rupture rate
of CAs is 1% to 2% per year and seems to remain
almost constant from decade to decade.24-27 Only about
50% of patients with aneurysmal SAH have a favorable
outcome.14-17 In most surgical series, clipping of unrup-
tured CAs is associated with 0% mortality,18-21 and the
morbidity rate does not exceed 5%.22,23 Although it is
likely that these figures represent an improvement over
the natural history of these lesions, the management of
patients with unruptured CAs remains controversial.6,23
At the present time, there is no cost-effective method to
screen the general population for unruptured CAs.23
TCCD is a new ultrasonographic method that might
refine and ultimately replace transcranial Doppler
sonography for the study of the intracranial circulation.
Therefore, additional examination for unruptured CAs
during routine TCCD studies might prove useful.
In our search for reliable criteria for ultrasonographic
diagnosis, the following five TCCD findings were deline-
ated: (1) In central scanning planes, a circular or oval
mass was divided by a separation zone into blue and red
zones, indicating blood flow toward and away from the
transducer, respectively. (2) The separation zone was
centrally or paracentrally located and was characterized
by dark or no color-coded Doppler signals. Intra-aneu-
rysmal blood flow depends on different anatomic fac-
tors, such as the angle between the downstream wall of
the aneurysm and the axis of the parent artery, the area


visual inspection of the intracranial arteries should be
12. Jellinger K. Pathology and aetiology of intracranial aneurysms. In:
done during all routine TCCD studies to detect
screening for CAs. However, we suggest that careful
visual inspection of the intracranial arteries should be
done during all routine TCCD studies to detect
nonthrombosed CAs.

In summary, TCCD can provide the diagnosis of
nonthrombosed CAs using the above-cited hemody-
amic criteria. A definite limitation is its inability to
detect small CAs and thrombosis. Another limitation is
that aneurysm size cannot be adequately detected.
Finally, it is evident that without previous knowledge of
aneurysm location the rate of ultrasonographic detect-
ion will decrease, especially for inexperienced sonog-
rappers. Therefore, TCCD should not be used for
screening for CAs. However, we suggest that careful

References
1. Rosenorn J, Eskesen V, Schmidt K, Espersen JO, Haase J,
Harmesen A, Hein O, Knudsen V, Midholm S, Marcusen E.
Clinical features and outcome in 1076 patients with ruptured intra-
cranial vascular aneurysms: a prospective consecutive study. Br J
2. Saveland H, Soneson B, Ljunggren B, Brandt L, Uski T, Zygmont
S, Hindfelt B. Outcome evaluation following subarachnoid hem-
3. Heiskanen O. Risks of surgery for unruptured intracranial aneurysms.
4. Jane JA, Kassell NF, Tomer JC, Winn MR. The natural history of
5. Juvela S, Porras M, Heiskanen O. Natural history of intracranial aneurysms:
6. Wiebers DO, Whisnant JP, Sundt TM Jr, O’Fallon M. The signif-
7. Winn HR, Almaani WS, Berga SL, Jane JA, Richardson AE. The long-term
outcome in patients with multiple aneurysms: incidence of late hemorrhage and impli-
1983;59:642-651.
Warmuth-Metz M, Bogdahn U. Diagnosis and monitoring of sub-
arachnoid hemorrhage by transcranial color-coded real-time
WD, Quiroz FA, Macrander SJ, Lipichk EO. Stenosis of the
internal carotid artery: assessment using color Doppler imaging
10. Steinke W, Hennerici M, Rautenberg W, Mohr JP. Symptomatic
and asymptomatic high-grade carotid stenoses in Doppler
A, Kahn T, Steinmetz H. Between-method correlation in quan-
12. Jellinger K. Pathology and aetiology of intracranial aneurysms. In:
Pia HW, Langmaid C, Zierski J. Cerebral Aneurysms. Advances in
Diagnosis and Therapy. Berlin, Germany: Springer; 1979:5-19.
Transcranial color-coded duplex sonography in cerebral aneurysms.
R W Baumgartner, H P Mattle, K Kothbauer and G Schroth

Stroke. 1994;25:2429-2434
doi: 10.1161/01.STR.25.12.2429

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/25/12/2429

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/