Transcranial Color-Coded Duplex Sonography in Cerebral Aneurysms

Ralf W. Baumgartner, MD; Heinrich P. Matti, 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 were prospectively examined using transcranial color-coded duplex sonography. The sonographer was aware of cerebral computed tomographic and magnetic resonance imaging findings but was blinded to the results of cerebral angiography.

Results Ultrasonographic findings for aneurysms studied were as follows: (1) Scanning planes that transsected approximately mid-aneurysm showed a round or oval mass that was divided by a "separation zone" into red and blue areas. (2) The "separation zone" was characterized by dark or no colors. (3) Peripheral scanning planes showed monochromatic images. (4) No turbulence was found. (5) No spontaneous fluctuations were detected. Twenty-three of 27 (85%) nonthrombosed aneurysms with a diameter of 6 to 25 mm were identified. The walls and three thrombosed and four nonthrombosed aneurysms (mean diameter, 5 mm) were missed.

Conclusions Transcranial color-coded duplex sonography can provide the diagnosis of nonthrombosed aneurysm using the above-cited criteria because of its capacity to reveal flow phenomena. It is not the method of choice in the search for aneurysms because small and thrombosed aneurysms are missed. Careful visual inspection of the intracranial arteries to permit incidental detection of cerebral aneurysms should be part of every transcranial color-coded duplex examination.

Key Words • cerebral aneurysms • subarachnoid hemorrhage • ultrasonics
<table>
<thead>
<tr>
<th>Patient</th>
<th>Aneurysm</th>
<th>Symptom</th>
<th>CN Deficit</th>
<th>SAH</th>
<th>Hematoma</th>
<th>Thrombosis</th>
<th>Diameter, mm</th>
<th>TCCD Diameter, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ICA C4</td>
<td>Asymptotic</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>-</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>s</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</td>
<td>-</td>
<td>8.2</td>
<td>9.0</td>
</tr>
<tr>
<td>21</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>22</td>
<td>MCA Bif</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>8.3</td>
<td>7.7</td>
</tr>
<tr>
<td>23</td>
<td>MCA Bif</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>s, 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, c</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>-</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.

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 insonations 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 \(-0.1\pm12.7\%\) (mean±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±44 hours (mean±SD). The delay between TCCD and angiographic studies was 10±8
rysmal blood flow depends on different anatomic fac-
by dark or no color-coded Doppler signals. Intra-aneu-
centrally or paracentrally located and was characterized
the aneurysm and the axis of the parent artery, the area
ctors, such as the angle between the downstream wall of
zones, indicating blood flow toward and away from the
morbidity rate does not exceed 5%. Although it is
the natural history of these lesions, the management of
series from 0.2% to 9.0%. The annual rupture rate
of patients with aneurysmal SAH have a favorable
patients with unruptured CAs remains controversial. Only 50%
and thus the angle of in-
uated CAs are not visualized in the B-mode image. In
Doppler sonography, the measured blood velocity ($V_a$) depends on the cosine value of the insonation angle $\beta$
according to the formula $V_a = V_m \times \cos \beta$, where $V_m$
represents the real blood velocity. Since the direction of
blood flow within CAs and thus the angle of in-
ulation zones corresponds to an area with slow or unde-
tectable blood flow. This observation confirms the find-
ings of previous work that the slowest velocities are
registered in the center of an aneurysm.3-24-26 (3) In
peripheral scanning planes, monodirectional flow pat-
tterns were found. This finding corroborates results of
previous studies using Doppler sonography, angiogra-
phy, and MRI.26-27-29 (4)Unlike Ferguson30 but in
accordance with other authors,26-27-31 we did not find
intra-aneurysmal circulation was stab.
On the basis of the above-cited five criteria, TCCD
identified 23 of 27 (85%) radiologically proven and
nonthrombosed CAs with an intraluminal diameter of 6
to 25 mm. These results are slightly better than the
findings of Becker et al, who identified 76% of all
examined and nonthrombosed CAs. Nonidentification
of four CAs with a mean intraluminal diameter of 5 mm
probably relates to their small size. This caused lower
intensity of the Doppler signal and impeded the use of
more accurate insonation angles and the distinction
from neighboring vessels. In addition, these CAs were
located in the paramedian anterior and posterior border
zones of the TCCD image, increasing the travel path
and thus the attenuation of the ultrasound beam. A
thick temporal bone prevented the performance of
TCCD study in a patient with a thrombosed CA. These
figures correspond to the reported data that cerebral
arteries were identified in four TCCD studies in more
than 90% of 191 patients.32-35 However, further studies
are necessary to evaluate the positive and negative
predictive values of TCCD to detect CAs with an
intraluminal diameter larger than 5 mm.

The patent lumen of CAs was delineated by measur-
ing the maximal extent of the color-coded Doppler
signal, since the walls and thromboses including three
completely thrombosed CAs were not detected in
B-mode images. The determination of intraluminal di-
ameter by measuring the “flow lumen” has already been
used in extracranial color-coded duplex sonography
with high accuracy.3-11 Mean TCCD values for intralu-
minal diameter were quasi-identical with those obtained
by CT. However, the standard deviation and range of
the TCCD values were unacceptably high. “Over gain-
ing” of color settings leading to bleeding of the color
signal over the boundaries, attenuation of Dopplar

### TABLE 2. Maximal Systolic and End-Diastolic Blood Velocities for the ACA, MCA, PCA, VA, and BA in 104 Healthy Volunteers

<table>
<thead>
<tr>
<th>Vessel</th>
<th>$V_s \pm 2$ SD, cm/s</th>
<th>$V_d \pm 2$ SD, cm/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACA</td>
<td>82 ± 58</td>
<td>34 ± 19</td>
</tr>
<tr>
<td>MCA</td>
<td>106 ± 39</td>
<td>46 ± 20</td>
</tr>
<tr>
<td>PCA</td>
<td>65 ± 25</td>
<td>28 ± 14</td>
</tr>
<tr>
<td>VA</td>
<td>51 ± 27</td>
<td>22 ± 14</td>
</tr>
<tr>
<td>BA</td>
<td>64 ± 31</td>
<td>27 ± 16</td>
</tr>
</tbody>
</table>

$V_s$ 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
40% of patients with aneurysmal SAH have a favorable
outcome.14-17 In most surgical series, clipping of unru-
 spurred CAs is associated with 0% mortality,18-21 and the
morbidity rate does not exceed 5%.2-22 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.2-23

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 delin-
eated: (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-

Baumgartner et al TCCD in Cerebral Aneurysms 2433

Downloaded from http://stroke.ahajournals.org/ by guest on August 28, 2017
signals, and the fact that resolution for color is less than for B-mode imaging are probably the most important explanations. Therefore, TCCD measurement of the "flow lumen" is not accurate for the determination of intraluminal diameter of CAs.

In summary, TCCD can provide the diagnosis of nonthrombosed CAs using the above-cited hemodynamic 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 detection will decrease, especially for inexperienced sonographers. Therefore, TCCD should not be used for 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.

References


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

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/