Transcranial Color-Coded Duplex Sonography of Intracerebral Hematomas in Adults

G. Seidel, MD; M. Kaps, MD; W. Dorndorf, MD

Background and Purpose: It is well established from pediatric experience and animal experiments that intracerebral blood can be demonstrated by B-mode real-time duplex scanning. This has recently become feasible in adults as well. The present study investigated the changes in the sonographic appearance of intracerebral hematomas over the course of time.

Methods: Starting in May 1991, 23 consecutive patients with intracerebral hematoma confirmed by computed tomography (21 spontaneous and 2 traumatic hematomas) were investigated within 1 year. They were monitored by repeated ultrasound scanning via the transtemporal approach. The sonographic appearance of the hematomas on B-mode scans and the angle-corrected blood flow velocity in the basal cerebral arteries were assessed.

Results: There was unequivocal localization of the hematoma in 18 patients (78%). In 3 cases (13%), an adequate acoustic window could not be found. One small intracerebral hemorrhage was overlooked, and one extensive hemorrhage in the basal ganglia was misdiagnosed as a lobar hematoma. There was an alteration of the appearance of the hematoma with time. This was divided into three sonographic stages (initial stage, days 1 to 5; intermediate stage, days 6 to 10; and capsular stage, from day 10). In 14 of the 20 patients with an appropriate acoustic bony echo window, the blood flow velocity in the middle cerebral artery could be measured; in 1 of these patients, the signs of increasing intracranial pressure were apparent from Doppler frequency spectrum. In 5 patients, the intracerebral hematoma could be imaged but not the ipsilateral middle cerebral artery. One female patient showed cerebral circulatory arrest at the time of examination, which took place within 24 hours after the onset of clinical symptoms.

Conclusions: Most intracerebral hematomas in adults can be imaged in B-mode. Their sonographic appearance changes over the course of the disease. The advantages of this noninvasive method are its easy bedside operation and its suitability for follow-up; it is also less stressful than other imaging procedures. It yields a combination of structural and functional diagnostic information. In approximately 13% of the cases, the investigation was not feasible because of inadequate ultrasonic penetration of the intact skull. (Stroke. 1993;24:1519-1527.)

Key Words • cerebrovascular disorders • hematoma • ultrasonics

Transcranial sonography has a long tradition, beginning in the early 1940s,1,2 with one-dimensional echoencephalography and the detection of intracranial brain tumors and hematomas.3,4 With the two-dimensional static B-scanning of the brain, it was possible to get a laminographic presentation of the cerebral anatomy.5,6 This method had limitations because more patient cooperation was needed than for A-mode, with a complete examination taking approximately 1 hour. Interpretation of B-mode echoencephalograms was difficult, so other imaging systems, like computed tomography, with higher spatial resolution and sensitivity took their place.

Investigations in pediatric patients through the nonossified fontanel7,8 and animal studies through the trepanned skull9 have shown that intracerebral hematomas can be demonstrated with sufficient spatial resolution by transcranial real-time sonography, a new computer-imaging technology.10

By use of the new duplex systems, it is now easily possible via a transtemporal approach to recognize intracerebral structures in adults using B-mode real-time sonography11 and to record the blood flow velocity in the basal cerebral arteries.12

Compared with conventional “blind” transcranial Doppler sonography, this technique offers the advantage of reproducible and comparable measurements of blood flow velocity in the arteries at the base of the brain, even in the presence of large cerebral hemorrhages and independent of any potential vessel displacement and consequent alterations in the scanning angle.

The present study examines to what extent intracerebral hematomas in the adult can be imaged and how the sonographic appearance changes over time. In addition, possible hematoma complications are demonstrated, such as ventricular hemorrhage, indirect signs of cerebral edema with midline shift, and alterations of the blood flow velocity in the middle cerebral artery (MCA).

Subjects and Methods

Within a year beginning May 1991, a series of 23 consecutive patients from our clinic with intracerebral

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FIG 1. Hemorrhage into the basal ganglia. Serial scanning by computed tomography (A, D, and G) and sonography (B, C, E, F, H, and I) in comparable axial planes and at similar intervals is shown as follows day 1, A and B; day 4, C; day 7, D and E; day 9, F; day 13, G and H; and day 21, I.

hematomas, confirmed by computed tomography, were enrolled (age, 66.4±15.5 years; 9 female and 14 male; 21 spontaneous and 2 posttraumatic hematomas [clinically, comotio cerebri]).

The change in the sonographic appearance of the hematomas over time (Table 1) and the angle-corrected blood flow velocity in the M1 segment of the MCA were assessed bilaterally. On the basis of animal experimental data involving the sonographic appearance of intracerebral hematomas,\textsuperscript{9} it seemed appropriate to limit the period of investigation to 21 days from the onset of neurological symptoms and the presence of an intracerebral hematoma confirmed by computed tomography.

Color-coded duplex sonography was performed transtemporally in projection to the so-called “orbitomeatal line” using a 2.5-MHz 90° sector transducer (HP SONOS 1000, Hewlett-Packard Co, Palo Alto, Calif). With a scanning depth of 16 cm and progressing from the side opposite the hematoma, imaging of the brain stem was necessary first to obtain a “landmark” for orientation.

By tilting the duplex probe, the maximum extent of the hematoma could then be imaged in the opposite hemisphere in both axial and coronal planes. In a second step, the angle-corrected blood flow velocity in the M1 segment of the MCA was assessed from the ipsilateral side.
First, the brain stem had to be identified unequivocally at a scanning depth of 16 cm. After reducing the scanning depth to 10 cm, the course of the ipsilateral MCA was found by rotating and tilting the ultrasound probe; the artery is distinguished from the surrounding brain tissue by its echogenicity on B-mode scans. By switching to the color-coded representation of the flow velocity, the course of the artery could be identified clearly, and the angle-corrected measurement of blood flow velocity was carried out at a scanning depth of 40 to 65 mm by placing the probe parallel to the course of the artery. The diameter of the sample volume was 1.2 mm. By means of fast Fourier analysis, the frequency range of the flow velocity could be calculated and graphically displayed (maximum, 154 cm/s). During the flow velocity measurements, the B-mode image was refreshed every 3 seconds to verify correct positioning of the probe.

The color coding depends on the direction of flow. By convention, flow toward the probe is represented in red, and flow away from it is represented in blue. Medium packet size and a cutter filter were selected for fast imaging rates and to minimize high-intensity low-velocity artificial signals.

At least one computed tomographic control scan was recorded during the study (5- to 8-mm sections parallel to the base of the skull, Siemens Somatom HiQ).

Results

By use of transcranial duplex sonography, the intracerebral hematoma could be clearly localized in 18 of the 23 patients investigated. The relation to the ventricular system was also demonstrable (Table 1). In three female patients (aged 66, 68, and 69 years), no adequate bony echo window for the assessment of intracerebral structures could be found. In one woman, a small intracortical hemorrhage was not recognized; in another woman, extensive hemorrhage into the basal ganglia was misinterpreted as a lobar hematoma.

The size of the hematomas diagnosed by transcranial duplex scanning varied from 6.8×4.8×3.2 cm^3 (approximately 55 mL) to 1.2×1.0×1.5 cm^3 (approximately 1 mL). The size of the undiagnosed intracerebral hemorrhage was 1.2×0.8 cm^3 (approximately 0.5 mL) on the computed tomographic scan and could not be clearly demarcated sonographically from the cortical folds.

Alterations over time in the sonographic appearance of the intracerebral hematomas (Fig 1) can be divided into three phases (Fig 2): the initial phase, the intermediate phase, and the capsular phase.

During the initial phase, extending from day 1 to day 5 after the onset of the hemorrhage, the hematoma is more echogenic than the surrounding brain tissue and at first appears sharply demarcated, but later the margins become blurred. In a few patients, patches of lesser echogenicity could be observed within the sharply demarcated echo-dense hematoma on the first day (Fig 2A), before the hematoma became uniformly echo dense during the following day (Fig 1B and 1C). We couldn't see any direct duplex signs of perihematoma edema.

During the subsequent intermediate phase (days 6 to 10), there was a gradual decline in echogenicity in the center of the hematoma (Figs 1E and 2D).

The period after day 10 can be referred to as the capsular phase. At this stage, the center of the hematoma is of lesser echogenicity than the surrounding brain tissue and is lined by a fine, distinct, echo-dense seam (Figs 1H, 1I, and 2F).

In assessing intracerebral hematomas, the question of ventricular involvement is of clinical interest. In Fig 2, an echo-dense structure is clearly visible in the anterior horn of the lateral ventricle, indicating intraventricular
blood. When blood enters the posterior horn of the lateral ventricle, distinguishing it morphologically from the echo-dense choroid plexus may be difficult. Sometimes a decision can only be made on the basis of serial duplex sonographies. In the remaining portions of the ventricles, blood can be recognized by its typical echo-dense appearance within the cerebral parenchyma.

In 14 of the 20 patients with bony echo windows, the MCA could be demonstrated (Table 2). In four women and one man, because of compromised sonographic conditions, only the hematoma could be imaged but not the flow velocity in the basal cerebral arteries. In one 36-year-old man with a left-sided temporoparietal lobar hematoma, the typical Doppler sonographic signs of increasing intracranial pressure appeared. Fig 3A shows the midline shift caused by the space-occupying lobar hematoma on the left side. In the course of increased intracranial pressure, an elevated pulsatility index (Fig 3B) and, in the final stage, a swelling of the basal cisternae with brain stem compression (Fig 3C) could be detected. This patient eventually died of cerebral circulatory arrest.

Cerebrospinal fluid circulation disturbance is a severe complication of intracerebral hematoma. Fig 4 shows duplex scans of a 58-year-old man with a thalamus hematoma and blood in the third and lateral ventricle. From day 2 to day 4 the diameter of the lateral ventricle contralateral to the hematoma increased, indicating hydrocephalus. A ventricular shunt was applied 4 hours after the last duplex scan on day 4, and the intraventricular pressure was elevated up to 32 mm Hg.

**Discussion**

The present study shows that in 19 of 23 (83%) adult patients with intracerebral hematomas confirmed by computed tomography, the diagnosis was made by transcranial duplex sonography. The size and localization of the hematomas are important. Small cortical hemorrhages cannot be clearly distinguished from the echogenic appearance of the border zone between the cortex and subarachnoid space.

In adults, there is a characteristic change in the sonographic appearance of the hematomas over time, which hitherto could only be investigated in experimental animals after performing trepanation and in new-

**TABLE 1. Comparison of Hematoma Localization by Computed Tomography and Duplex Sonography**

<table>
<thead>
<tr>
<th>Initials</th>
<th>Sex</th>
<th>Age, y</th>
<th>Computed Tomography</th>
<th>Duplex B-Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>RH</td>
<td>M</td>
<td>59</td>
<td>BG (total)+VH</td>
<td>BG (total)+VH</td>
</tr>
<tr>
<td>LL</td>
<td>M</td>
<td>84</td>
<td>Putamen</td>
<td>Putamen</td>
</tr>
<tr>
<td>SH</td>
<td>F</td>
<td>87</td>
<td>Putamen</td>
<td>Putamen</td>
</tr>
<tr>
<td>MJ</td>
<td>M</td>
<td>64</td>
<td>Putamen</td>
<td>Putamen</td>
</tr>
<tr>
<td>OW</td>
<td>M</td>
<td>80</td>
<td>Putamen</td>
<td>Putamen</td>
</tr>
<tr>
<td>NR</td>
<td>M</td>
<td>58</td>
<td>Thalamus+VH</td>
<td>Thalamus+VH</td>
</tr>
<tr>
<td>IP</td>
<td>M</td>
<td>62</td>
<td>Thalamus+VH</td>
<td>Thalamus+VH</td>
</tr>
<tr>
<td>PK</td>
<td>M</td>
<td>80</td>
<td>Thalamus+VH</td>
<td>Thalamus+VH</td>
</tr>
<tr>
<td>SH</td>
<td>F</td>
<td>79</td>
<td>Thalamus+VH</td>
<td>Thalamus+VH</td>
</tr>
<tr>
<td>HE</td>
<td>M</td>
<td>87</td>
<td>Thalamus</td>
<td>Thalamus</td>
</tr>
<tr>
<td>MM</td>
<td>F</td>
<td>63</td>
<td>Thalamus</td>
<td>Thalamus</td>
</tr>
<tr>
<td>SR</td>
<td>M</td>
<td>36</td>
<td>Lobar/HS</td>
<td>Lobar/HS</td>
</tr>
<tr>
<td>SW</td>
<td>M</td>
<td>74</td>
<td>Lobar/SC+VH</td>
<td>Lobar/SC+VH</td>
</tr>
<tr>
<td>BJ</td>
<td>M</td>
<td>55</td>
<td>Lobar/SC+VH</td>
<td>Lobar/SC+VH</td>
</tr>
<tr>
<td>NT</td>
<td>M</td>
<td>35</td>
<td>Lobar/SC</td>
<td>Lobar/SC</td>
</tr>
<tr>
<td>RK</td>
<td>M</td>
<td>72</td>
<td>Lobar/cortical</td>
<td>Lobar/cortical</td>
</tr>
<tr>
<td>NK</td>
<td>M</td>
<td>58</td>
<td>Lobar/cortical</td>
<td>Lobar/cortical</td>
</tr>
<tr>
<td>SA</td>
<td>F</td>
<td>59</td>
<td>Cerebellum, lobar/SC</td>
<td>Cerebellum, lobar/SC</td>
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<tr>
<td>SG</td>
<td>F</td>
<td>69</td>
<td>Thalamus</td>
<td>0</td>
</tr>
<tr>
<td>WE</td>
<td>F</td>
<td>68</td>
<td>Thalamus</td>
<td>0</td>
</tr>
<tr>
<td>ME</td>
<td>F</td>
<td>66</td>
<td>Putamen+VH</td>
<td>0</td>
</tr>
<tr>
<td>HL</td>
<td>F</td>
<td>71</td>
<td>Lobar/cortical</td>
<td>Not diagnosed</td>
</tr>
<tr>
<td>DM</td>
<td>F</td>
<td>84</td>
<td>BG (total)</td>
<td>Lobar/HS</td>
</tr>
</tbody>
</table>

BG indicates basal ganglia; VH, ventricular hemorrhage; lobar/HS, extensive hemispheric hematoma of cortical/subcortical extension; lobar/SC, intralobar hematoma of subcortical localization; lobar/cortical, intralobar hematoma of cortical localization; and 0, inadequate conditions for ultrasound scanning (classified by computed tomographic findings).""
Hematoma morphology has been described after experiments on the trephined canine skull in which sonographic, computed tomographic, and histological findings were compared. According to the results, a homogeneous, sharply demarcated, hyperdense image of the hematoma was obtained during the acute phase (days 1 to 3 after the start of the hemorrhage) when using a 10-MHz linear transducer. Histologically, this corresponds to tightly packed intact erythrocytes. Directly around the hematoma, an inflammatory infiltrate consisting primarily of small lymphocytes and large mononuclear cells, as well as a narrow band of edema and infarction, can be demonstrated. There is no sonographic correlation to this perihematomal edema. On computed tomography, the intracerebral blood typically appears hyperdense, compact, and sharply demar
FIG 3. A shows duplex sonographic imaging (left) and scheme (right) of a large left-sided lobar hematoma in coronal (top) and axial (middle) planes. A clearly visible midline shift can be seen when comparing sonographic imaging from both sides (middle and bottom). H indicates hematoma; S, skull; SB, skull base; M, midline structures; CP, choroid plexus; and CV, cysterna venae Galeni. B shows the time course (days 1 and 5) of angle-corrected blood flow in the middle cerebral artery (MCA). At the top is the duplex sonographic image of the M1 section of the MCA. The Doppler wave form of the MCA appears below the image. Note the reduction in diastolic flow on day 5. Cerebral blood flow was no longer demonstrable on day 6. C shows duplex sonographic imaging (left) and scheme (right) revealing swelling of the basal cysternae on day 7 (top) contrasted with the findings on day 3 (bottom). S indicates skull; M, midline structures; MES, mesencephalon; and T, temporal bone.
cated during this phase (40 to 90 Hounsfield units, depending on the hemoglobin content of the blood).13

The investigations carried out in the present study revealed that there are patches of reduced echogenicity in the hyperdense hematoma in some patients on the first day following hemorrhage. These patches may indicate blood that has not yet clotted. With ultrasound probes of higher frequency (10 MHz), such as were used in animal experiments,9 this has a hyperdense appearance similar to coagulated blood. When low-frequency probes (2.5 MHz) are used, as in the present study, the signal is hypodense.14

According to investigations carried out in dogs,9 there is an amorphous alteration in erythrocyte shape (at first in the center; later, at the periphery of the hematoma) during the subacute phase (4 to 8 days after hemorrhage). Eventually, the erythrocytes burst. Thus, the echogenicity at the center of the hematoma diminishes. At the end of this phase (from days 6 to 8), the hemoglobin at the periphery of the hematoma is begin-

ning to be converted to hemosiderin; on computed tomography, this leads to a decrease in the density of the edge of the hematoma.13

The capsular phase (from days 9 to 13) described in the experimental animal is characterized histologically by a network of collagen and macrophages. The sonographic correlate is an hypodense, sharply demarcated seam around the hematoma.

Histologically, in the last phase, which may be referred to as the phase of organization (from day 14), there is an increase in the thickness of the connective tissue capsule around the hematoma, which now has a collagen matrix. On ultrasound, the echo-dense seam begins to narrow.

Altogether, the results obtained in animal experiments correlate well with our investigations in humans. Complications of intracerebral hematomas, such as ventricular involvement, cerebrospinal fluid circulation disturbance, and space-occupying effects, can be recognized by means of transcranial duplex sonography. Hemorrhage into the lateral ventricles, when limited to the posterior horn, leads to diagnostic difficulties because the choroid plexus is of similar echogenicity. An unequivocal differentiation may only be possible by serial scanning, since intraventricular blood disappears sonographically during the early days of illness, whereas the echogenic structure of the choroid plexus remains unaltered.

Additional information about alterations in intracranial pressure may be obtained by analyzing the Doppler frequency spectrum of the basal cerebral arteries when an increase in peripheral resistance accompanied by a decrease in flow becomes apparent during the course of the condition. In the present study, other hemodynamic data yielded no evidence of vessel spasm. A more detailed analysis of hemodynamic phenomena in connection with intracerebral hematomas is in progress.

Our results show that transcranial duplex sonography is capable of imaging the majority of intracerebral hematomas, that it is suitable for follow-up of these stroke patients, and that it is less stressful than other imaging

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**TABLE 2. Color-Coded Duplex Sonography of the Middle Cerebral Artery With Reference to the Cause of the Failure of Imaging, the Extent of Imaging, and Sex**

<table>
<thead>
<tr>
<th>Cause/Extent</th>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not imaged (n=9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No bony echo window</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Echo window, only hematoma imaged</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Cerebral circulatory arrest</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Imaged (n=14)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unilateral</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
<td>Bilateral</td>
<td>1</td>
<td>7</td>
</tr>
</tbody>
</table>
procedures. There are no sonographic differences between ischemic areas of the brain and healthy brain tissue. The method used in the present study not only facilitates diagnosis on the basis of morphology but also allows hemodynamic aspects to be assessed, independent of possible vascular displacement. From preliminary communications, it seems probable that, under certain conditions, etiologic factors such as larger cerebral aneurysms, arteriovenous malformations, or vascular tumors can be demonstrated as the source of the hemorrhage.15

One disadvantage of transcranial duplex sonography undoubtedly lies in the hitherto limited spacial resolution due to the attenuation of sound waves upon penetration of the skull. As a result of this, 13% of our patients could not be examined by this technique. In addition, frontal and parasagittal regions of the brain cannot at present be accurately assessed by transcranial duplex sonography.

The advantage of this method is that stroke patients can be examined directly on admission to hospital by a simple, noninvasive, and time-saving method. Since transcranial color-coded duplex sonography can contribute to the differential diagnosis of a stroke when computed tomography is not immediately available, it seems worthwhile to develop this method further, particularly when fibrinolytic or anticoagulant therapy is being considered.

References
Transcranial color-coded duplex sonography of intracerebral hematomas in adults.
G Seidel, M Kaps and W Dorndorf

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