A-Mode Echoencephalography in the Evaluation of Cerebrovascular Disease

BY BURTON A. SANDOK, M.D.

Abstract:
A-Mode Echoencephalography in the Evaluation of Cerebrovascular Disease

The value of A-mode echoencephalography was studied in a series of patients with cerebrovascular disease of various types. The procedure proved to be restricted value limited to those situations in which (1) a midline shift was obtained, and (2) the midline shift was observed within 36 hours after the onset of symptoms.

Whereas after 36 hours some patients with cerebral infarction showed a midline shift (presumably due to edema), such a shift was not noted in patients with cerebral infarct who were examined within 36 hours after its occurrence. Those patients showing a midline shift before this interval should be suspected of having an intracerebral hemorrhage or other intracranial space-occupying process.

ADDITIONAL KEY WORDS
ultrasonic diagnosis cerebral infarction cerebral hemorrhage

To distinguish cerebral infarction from intracerebral hemorrhage and other focal brain abnormalities may be fraught with difficulty. The echoencephalogram may be a valuable aid in the resolution of this differential diagnosis.

In a summary of the existing echoencephalographical literature through 1968, Shiefer and associates observed that in 239 reported cases of intracerebral hemorrhage, the echoencephalograms of 206 or 86.2% showed a midline shift while in 743 reported cases of cerebral infarction, only 107 or 14.4% showed a shifted midline. This suggests a striking difference, although this type of statistical analysis has only limited clinical application when examining an individual patient. The absence of a significant midline shift is of no differential diagnostic value, and even in the face of a shifted midline, one is still unable to state whether the patient is one of the 86.2% with an intracerebral hemorrhage or one of the 14.4% with an infarction.

From the Department of Neurology and the Clinical Cerebrovascular Research Center, Mayo Clinic and Mayo Foundation, Rochester, Minnesota, 55901.

This investigation was supported in part by Research Grant NB-6663 from the National Institutes of Health, Public Health Service.

It has been suggested, therefore, that the decisive echoencephalographical criterion on which the differential diagnosis of infarct versus hemorrhage might be based is the time at which the echo shift appears, the presence of a shift prior to the appearance of cerebral edema in cases of infarction implicating an intracerebral hemorrhage.

Meaningful therapeutic intervention in cases of cerebrovascular disease is based on accurate diagnosis, and since it has been suggested that A-mode echoencephalography might be of value in this regard, this study was performed to ascertain the practical usefulness of this procedure in the evaluation of patients with cerebrovascular disease.

Methods
During one 12-month period, hospitalized patients seen by a consulting neurologist and strongly suspected of having a form of cerebrovascular disease were reported to the Clinical Cerebrovascular Research Center, St. Mary's Hospital, generally within 24 hours of their admission. Patients in whom a diagnosis of cerebrovascular disease was thought to be remote (that is, those regarded as "brain-tumor suspects"), those with known traumatic hemorrhages, and those in whom another diagnosis was established shortly after their
TABLE 1

Echoencephalographical Findings in 192 Patients With Suspected Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No shift (&lt; 2 mm)</th>
<th>Shift (2 mm or greater)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent focal neurological deficit</td>
<td>104</td>
<td>32</td>
<td>136</td>
</tr>
<tr>
<td>Transient ischemic attacks</td>
<td>32</td>
<td>0</td>
<td>32</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>23</td>
<td>1</td>
<td>24</td>
</tr>
</tbody>
</table>

admission were not reported to the Center and hence were excluded from further study.

Of the reported patients, 192 had satisfactory echoencephalographical examinations performed at some time during their hospital admission but prior to any definitive diagnostic radiological procedure; these patients form the basis for this study. All echoencephalography was performed with an Ekoline-20 ultrasonoscope and the echoencephalograms were interpreted without knowledge of the clinical case in accordance with the method reported previously. Midline shifts of 2 mm or greater were considered to be abnormal.

At the conclusion of the study period, the echoencephalographical results and the clinical summaries were reviewed and the results were tabulated (table 1).

**Results**

The series under study is primarily a clinical one, and the difficulty in separating the various types of focal neurological deficits without morphological examination is well recognized; therefore, little attempt was made to assign a specific pathological diagnosis to each patient under study. Echoencephalograms of the 136 patients who had persistent focal neurological dysfunction showed that 104 had no significant midline shift. Although most patients in this group were thought to have clinical evidence of cerebral infarction, a normal unshifted midline echo was present in rare cases of intracerebral hemorrhage and intracranial tumor.

The echoencephalograms of 32 patients with persistent focal neurological dysfunction showed a shift of the midline structures and the cases were analyzed further as to time of onset of symptoms and ultimate diagnosis (table 2).

Of the seven patients with a significant midline shift who were examined within 36 hours of their presumed cerebrovascular accident, five had an intracerebral hemorrhage, one a subdural hematoma, and one a grade 3 astrocytoma. No patient with a cerebral infarct examined within 36 hours of its occurrence had a significant midline shift.

Of the 21 patients with midline shifts who were examined within 36 hours to two weeks after their presumed cerebrovascular accident, one patient had had an intracerebral hemorrhage, one a subdural hematoma, and one a metastatic carcinoma; 18 patients were considered to have had a cerebral infarction. Follow-up serial echoencephalograms of six of these 18 patients demonstrated resolution of the shift within two to 18 days.

The remaining four of the 32 patients with midline shifts were examined initially more than two weeks after their presumed cerebrovascular accident. All were found to have had an infarction at least six months before examination and all echoencephalograms showed a significant shift toward the

**TABLE 2**

Thirty-Two Patients With Persistent Focal Neurological Deficit and Abnormal Echoencephalogram

<table>
<thead>
<tr>
<th>Clinical diagnosis</th>
<th>Time elapsed between last notable increase in symptoms and echoencephalographical examination</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0-26 hours</td>
</tr>
<tr>
<td>Cerebral infarct</td>
<td>0</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>5</td>
</tr>
<tr>
<td>Supratentorial mass</td>
<td>2</td>
</tr>
</tbody>
</table>
side of the infarct associated with an abnormally wide third ventricle (greater than 10 mm).

Thirty-two patients with a clinical diagnosis of transient ischemic attacks were examined; none of the echoencephalograms showed a shift of the midline structures.

Twenty-four patients with subarachnoid hemorrhage were examined; of 24 only one patient showed a 2.5-mm shift away from the side in which an aneurysm of the internal carotid artery was later identified. The echoencephalogram was obtained approximately 72 hours after the hemorrhage. The patient showed no focal neurological deficit and the angiogram performed seven days later did not show a significant shift.

In one patient with subarachnoid hemorrhage serial echoencephalograms over a three-week period showed progressive, pathological widening of the midline echo complex. Subsequent clinical and radiological investigation confirmed the diagnosis of communicating hydrocephalus.

During the course of the study an occasional patient was encountered who displayed abnormal "lateral" echoes. These echoes were present in patients with presumed acute cerebral infarction, intracerebral hematoma, and intracranial neoplasm; morphologically they did not appear sufficiently distinct to be of differential diagnostic value.

**Comment**

Review of the temporal sequence of events in the cases studied suggested that if the echoencephalogram is to be utilized in the differential diagnosis of presumed cerebrovascular disease, it will not be of significant value unless (1) it shows a midline shift, and (2) it is performed early in the course of the illness, probably within the first 36 hours.

A normal unshifted midline echoencephalogram, regardless of when it is obtained, may be of some inferential value to the clinician, but since we have found this evidence in cases of intracerebral hemorrhage and intracranial tumor as well as in cases of cerebral infarction, it is not diagnostic at any time.

Similarly, although most patients with cerebral infarction do not show a shift of the echoencephalographical midline even after 36 hours, a small percentage of such patients do have a midline shift; therefore, echoencephalographical midline shift does not differentiate cerebral infarction and intracerebral hemorrhage.

Since a midline shift was not found in echoencephalograms of patients who had a cerebral infarction of less than 36 hours' duration, it is concluded that the presence of a midline shift appearing within 36 hours is of great differential diagnostic value and should be viewed as presumptive evidence against the diagnosis of an uncomplicated cerebral infarction; it points strongly toward either an intracerebral hemorrhage or other space-occupying intracranial lesions.

This seemingly limited and highly restricted use of A-mode echoencephalography in the evaluation of the "stroke" patient is not without its practical application. While it appears that the test does not aid materially in specific identification of cerebral infarction, it does afford safe and efficient detection of at least a small percentage of patients suspected of having cerebral infarction but harboring other pathological lesions; thus it allows for more rapid institution of additional diagnostic and therapeutic procedures.

**Summary**

A-mode echoencephalography was utilized as part of the diagnostic evaluation in a series of patients with presumed cerebrovascular disease in an effort to determine its value in the differential diagnosis of varying types of pathological entities.

A normal echoencephalographical examination was not of diagnostic value since this was obtained in cases of intracerebral hemorrhage and other intracranial lesions as well as in cerebral infarction, subarachnoid hemorrhage, and transient ischemic attacks.

The test appeared to be of significant value only in those situations in which (1) a midline shift was present, and (2) the test was performed early in the course of the illness, generally within the first 36 hours. During this interval, midline shifts are not encountered in instances of cerebral infarction, while in a certain percentage of such patients, cerebral edema and midline shifts will develop later.

The finding of a midline shift on the echoencephalogram of a patient with presumed cerebrovascular disease examined within the first 36 hours after illness develops can be viewed, therefore, as presumptive evidence...
A-MODE ECHOENCEPHALOGRAPHY

against the diagnosis of an uncomplicated cerebral infarction and points strongly toward either an intracerebral hemorrhage or, on occasion, other intracranial space-occupying processes.

The test, when performed in accordance with the foregoing restrictions, can safely and efficiently detect at least a small percentage of patients suspected of having cerebral infarction but harboring other pathological lesions.

Acknowledgment

The technical assistance of Miss Lynda L. Reppart, Miss Susanne M. Bonin, and Mr. Richard D. Kopenski is gratefully appreciated.

References

A-Mode Echoencephalography in the Evaluation of Cerebrovascular Disease

BURTON A. SANDOK

Stroke. 1971;2:452-455
doi: 10.1161/01.STR.2.5.452

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 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/2/5/452

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/