Doppler Sonography Diagnosis of Cerebrovascular Disease

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SUMMARY Sonographic patterns for the Doppler ophthalmic test (DOT) were determined by reviewing the type of graphic record obtained from 25 patients who had undergone angiography. Three principal patterns were identified, depending upon whether there was augmentation (Type 1), diminution (Type 2) or no change (Type 3) in the character of the supraorbital Doppler signal upon temporal artery compression. Type 1 records were seen in 28 of 33 instances where the carotid arteries remained patent. Type 2 records occurred in 10 of 14 instances where there was significant carotid stenoses or tortuosity and in two instances in a patient with a subclavian steal syndrome. A "false-positive" test was recorded in one instance in a patient with vascular headaches. There were three Type 3 records.

Introduction

WITH THE ADVENT of reconstructive surgery of the extracranial vasculature, considerable emphasis has been placed on the early detection of extracranial vascular lesions. Lesions amenable to surgery are found in 40% to 75% of stroke patients. Screening techniques for cerebrovascular disease are of obvious interest, since this disease is the third leading cause of death in the United States. Arteriography, the only conclusive tool for identifying vascular lesions, is not suited for screening due to its small but inherent risks. However, ultrasound diagnosis does meet the criteria for a screening procedure in that it is reliable, noninvasive, inexpensive and safe.

The clinical value of Doppler ultrasound in the diagnosis of extracranial cerebrovascular disease was limited until the introduction of the Doppler ophthalmic test (DOT) in 1970 by Brockenbrough. Interpretation of this test traditionally has been based on audio analysis. Since there are numerous advantages to recorded data, results from an evaluation of 250 patients were studied in order to determine the character of Doppler records (sonograms).

Background

Ultrasonic measurement of blood flow through a vessel is based on the Doppler effect. This measurement detects the shift in frequency that occurs when an ultrasonic wave is reflected from moving blood cells. The direction and velocity of blood flow determine the nature of this shift.

The DOT exploits the anastomotic connection between the internal and external carotid arteries via the supraorbital artery, a terminal branch of the ophthalmic artery (fig. 1). Normally, due to a pressure gradient between the internal and external carotid circulations, the major source of blood flow to the supraorbital artery is through the internal carotid artery (ICA). With ICA stenosis or occlusion, the superficial temporal artery provides a major source of collateral flow to the internal carotid circulation via the supraorbital artery. This will cause a reversal in the normal direction of supraorbital flow which can be detected by the DOT.

Methods

Two hundred fifty patients seen at the Scripps Clinic and Research Foundation were studied. Patients were referred for a variety of reasons, ranging from a history suggestive of cerebrovascular disease to such nonspecific complaints as dizziness. Ten percent of these patients (25) underwent bilateral carotid angiography. Results of the DOT in this group of patients were compared with the angiographical findings. The selection of these cases was not entirely random in that patients undergoing arteriography were more likely to be seriously ill; however, there was no other selection of cases except for the requirement that the arteriographical and Doppler data were technically adequate to allow interpretation. A few patients who had been evaluated by arteriography elsewhere were included in the study if the x-rays were relatively recent.

Supraorbital flow was measured with a Model 806 directional Doppler flowmeter equipped with a 10 mHz pencil probe transducer. The 806 flowmeter can detect reversals in the direction of blood flow, and provides two DC output channels for recording opposite directions of flow. The two channels, labeled "flow toward the probe" and "flow away from the probe," were recorded simultaneously on a Sanborn Model 150 EKG recorder.

The test was administered with the patient in the supine position. The pencil probe, covered with an ultrasonic coupling gel, was placed over the supraorbital notch in the rim of the orbit. The probe was maneuvered until the listener, using headphones, appreciated the loudest and clearest pulsatile sound corresponding to the supraorbital arterial pulse. The pulse represents blood flowing toward the probe.

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Since the probe was maneuvered so as to detect the loudest supraorbital pulse possible, either normal or reversed, supraorbital flow appeared on the channel recording flow toward the probe (fig. 2). When a stable pulse was obtained, the superficial temporal artery was compressed manually against the temporal bone and the subsequent change in the supraorbital signal noted. The position of the probe was not changed during compression. The direction of the flow being recorded, i.e., whether it was caused by blood flow via the internal (normal) or external carotid circulation, was determined upon temporal artery compression. Reversed supraorbital flow via the external carotid circulation will be obliterated when the temporal artery is compressed. This procedure, performed on both sides of the head, constitutes the Doppler ophthalmic test.

Results

Three typical DOT records were identified. Type 1 records were characterized by augmentation of the supraorbital pulse during temporal artery compression (fig. 3). Type 2 records were characterized by obliteration of the pulse during temporal artery compression (fig. 4). A variation of Type 2 records in which a pulse appears in the second channel (flow away from the probe) during temporal artery compression was also seen (fig. 5). Type 3 records showed no change in the supraorbital pulse during temporal artery compression (fig. 6). Results of the DOT for the total series of 250 patients are summarized in table 1.

Results from the 25 cases undergoing carotid angiography are tabulated in table 2. The angiographical findings for each category of sonograms are presented in table 3. Fifty sonograms (two per patient) are represented. In 28 of the 33 Type 1 sonograms, the angiogram demonstrated complete

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**Figure 1.** Arteries of the carotid vasculature: (1) common carotid, (2) internal carotid, (3) external carotid, (4) ophthalmic, (5) supraorbital, (6) superficial temporal.

**Figure 2.** Normal and reversed flow. Either normal or reversed flow can be recorded as "flow toward the probe" by maneuvering the probe relative to the direction of blood flow (arrows indicate direction of blood flow).

**Figure 3.** Type 1 sonogram. In this and subsequent figures, the black bar under each recording indicates the period of temporal artery compression. The lower trace (channel 1) represents flow toward the probe, and the upper trace (channel 2) represents flow away from the probe. Paper speed = 10 mm per second.

**Figure 4.** Type 2 sonogram. Patient with ICA occlusion.
patency of the ICA. Four patients showed significant ICA stenosis. One of these patients had marked internal and external carotid artery lesions (see Discussion).

Of the 12 Type 2 sonograms with angiographical data, nine showed at least minimal stenosis of the ICA. In one patient (L), no stenotic lesion was demonstrated. The angiogram did show, however, marked tortuosity of the ICA approximately three inches above the bifurcation. Bilateral Type 2 sonograms were recorded from another patient with patent internal carotid arteries and a subclavian steal syndrome. Six Type 2 sonograms showed reversal of flow during temporal artery compression. All of these patients had carotid stenosis.

Type 3 sonograms were seen in three patients who underwent angiography, one with patent arteries and the other two with stenosis of the internal carotid arteries.

The types of recordings also were analyzed with respect to the clinical diagnosis. Seven of 16 patients with TIAs and/or completed strokes had patent carotid arteries. Six of these cases had Type 1 records. In the seventh case (L), the carotid artery was patent but extremely tortuous. Four patients (B, E, F and Q) with TIAs and/or strokes had bilateral carotid stenosis. In these cases, the Doppler was abnormal on at least one side. Five patients in this category had unilateral carotid stenosis — four such cases had Type 2 (F and W) or Type 3 (V and Y) records. There was one false-negative record (I).

One patient (T) had an asymptomatic carotid bruit associated with a Type 2 DOT. This patient had carotid stenosis verified by angiography. Those cases with a posterior fossa meningioma (G), seizure (N), and aneurysm (H), and another patient who was depressed had Type 1 sonograms. Angiograms were normal in each case regarding carotid artery disease. The three patients with migraine headaches were of interest in that one had a Type 2 record on three occasions, suggesting unilateral carotid disease. The angiogram was entirely normal. Both patients with a subclavian steal syndrome (V and T) had an abnormal DOT. One of these patients (T) had a right carotid lesion in addition to left subclavian stenosis, but in the other case (V) both carotid arteries were patent although Type 2 sonograms were seen bilaterally.

**Discussion**

Three typical DOT sonograms were identified in this study. These responses are similar to interpretations of the DOT based on audio analysis as described in the literature. 

**Table 2**

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*% = percent stenosis; BA = basilar artery; ICA = internal carotid artery; ECA = external carotid artery; SA = subclavian artery; VA = vertebral artery.
Type 1 Sonograms

Augmentation of the supraorbital pulse during temporal artery compression (Type 1 record) indicates increased supraorbital flow caused by accentuation of the normal internal to external carotid pressure gradient. In the majority of cases (29 of 33), a Type 1 sonogram correlated with either arterial patency or stenosis of less than 10%. Four false-negatives were seen (carotid stenosis 30% or greater).

In one of these cases (D), it is likely that a pressure gradient of normal polarity was maintained because of the presence of lesions in the internal and external carotid arteries. The supraorbital pulse was sufficiently small in amplitude, however, to lead us to suspect carotid insufficiency. This is consistent with the findings of Brockenbrough, who observed that a diminished supraorbital pulse in the presence of a normal response to temporal artery compression may be suggestive of carotid artery disease. Lesions in two cases (F and Q) were probably not hemodynamically significant.

The remaining false-negative test occurred in a patient (I) with occlusion of one ICA. In this circumstance, a change of circulation due to collateral flow via either the circle of Willis, the contralateral superficial temporal artery, or branches of the ipsilateral external carotid artery (other than the superficial temporal artery) could result in failure to appreciate the presence of carotid occlusion. All of these vessels have been demonstrated as sources of collateral flow in the presence of ICA stenosis.\(^{5,9}\)

Type 2 Sonograms

Obliteration of the supraorbital pulse during temporal artery compression (Type 2 record) indicates that the external carotid vasculature is providing collateral flow to the supraorbital artery. This suggests that ICA flow has been reduced sufficiently to reverse the normal internal to external carotid pressure gradient. The majority (9 of 14) of cases with Type 2 sonograms had demonstrable carotid artery disease. Interestingly, two of the false-positive tests were from a patient with a subclavian steal syndrome and patent internal carotid arteries. This seemingly "false-positive" test suggests the DOT may be sensitive to changes in collateral flow. In this case, the external carotid circulation was probably providing much of the blood going into the anterior circulation. With compression of the superficial temporal artery, flow from the external carotid artery via the supraorbital artery was blocked and thus the DOT appeared abnormal, suggesting carotid artery disease.

Another false-positive test was seen in a patient with a patent, yet markedly tortuous, left ICA. The clinical history of amaurosis fugax on the left and transient weakness in the right leg suggests that the positive DOT was probably due to a thrombotic event in the tortuous vessel which was resolved by the time the angiogram was done. The fourth false-positive test was recorded in a patient with chronic vascular headaches. Because of the patient's young age and the lack of findings on physical examination, the test was repeated after an interval of six weeks. It was positive again and the patient subsequently underwent angiography which was normal.

Four of the Type 2 sonograms exhibited a pulse in the channel recording flow away from the probe (channel 2) during temporal artery compression. This pulse appears in the interval during which flow toward the probe in the first channel (channel 1) is obliterated (fig. 3). Obliteration indicates that channel 1 is recording abnormal reversed flow. Since the two channels record opposite directions of flow, the pulse in channel 2 must represent supraorbital flow in the normal direction. The four sonograms exhibiting pulses in channel 2 were all from patients with carotid artery stenosis. In these cases, it appears that temporal artery compression reestablishes normal supraorbital flow via the stenosed ICA by reducing pressure in the external collateral circulation below that in the stenosed vessel.

Collateral flow from vessels other than the ICA might also account for the pulse seen in channel 2 of these recordings. However, the fact that this pulse is not seen in Type 2 sonograms from patients with ICA occlusion (W and T) seems to discount this possibility. Failure to see this pulse in all of the Type 2 sonograms from the patients with ICA stenoses may have been due to inadequate sensitivity of the recorder input or the input to the flowmeter channel detecting flow away from the probe. Both must be tuned so as to be sensitive enough to detect changes in the direction of supraorbital flow during temporal artery compression.

Hyman\(^{7}\) observed that flow through the stenosed artery can be identified as a higher frequency auditory signal upon temporal artery compression. Our experience indicates that the character of this signal may not be appreciated unless it is recorded. By recording our data, we were able to positively identify the directionality of this signal and graphically distinguish it from a normal response with which it can otherwise be confused.
Type 3 Sonograms

Type 3 sonograms were seen in only three patients with angiographical data. Although the exact interpretation of this recording is unclear, it may indicate that the supraorbital artery is receiving collateral flow through vessels which are not responsive to temporal artery compression. Machleder noted an analogous audio response in several normal patients who presented with significant peripheral vasoconstriction. In our total series of 250 cases, many of the Type 3 records were seen in patients with vascular headaches, including case C.

DOT Correlation With Angiography

In several cases, the percent of stenosis measured from the angiogram was below that generally considered to be hemodynamically significant. DeWeese et al., for example, have shown that a lesion compromising less than 47% luminal diameter will not produce a distal pressure drop greater than 10 mm Hg and, therefore, is not hemodynamically significant. The fact that Type 2 sonograms were obtained from patients with minimal angiographical abnormalities suggests that in some cases the DOT may be able to assess the hemodynamic significance of a lesion better than the angiogram (figs. 7 and 8). As Duncan et al., have pointed out, the capability of the angiogram to accurately assess vessel patency in the presence of a stenotic lesion may sometimes be limited. Our results would seem to confirm this statement.

Conclusions

Three identifiable patterns of the Doppler ophthalmic test have been identified by reviewing the types of graphic records obtained in 25 patients who have had angiography. In most cases (28 of 33), augmentation of the supraorbital Doppler signal upon compression of the superficial temporal arteries (Type 1 record) was associated with patent carotid arteries. Depression of the supraorbital signal with temporal artery compression (Type 2) was associated with carotid artery disease in 10 of 14 instances and with a subclavian steal syndrome in two instances. There was one "false-positive" result in a patient with vascular headaches. Failure of the supraorbital signal to change with temporal artery compression (Type 3 records) was observed on three occasions. The significance of this reading remains to be determined.

This preliminary investigation suggests a role for the DOT in the evaluation of patients with cerebrovascular disease. As expected, the DOT does not contribute to recognition of patients with TIAs without significant carotid stenosis. There also would appear to be a need for caution in interpreting results in patients with vascular headaches. Aside from these limitations, there is reason to be enthusiastic about use of this technique in a health care system which increasingly stresses the early detection and treatment of carotid vascular disease.

Acknowledgment

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References

Long-Term Anticoagulant Therapy for TIAs and Minor Strokes With Minimum Residuum

JAN E. OLSSON, M.D., RAGNAR MÜLLER, M.D., AND SUNE BERNELI, M.D.

SUMMARY One hundred seventy-eight patients with transient ischemic attacks (TIAs) or small strokes with slight symptoms persisting for more than 24 hours (incomplete recovery = IR) (TIA-IR) from both the carotid and the vertebrobasilar systems were treated with anticoagulants. Ten patients stopped the treatment because of severe side effects. Only one patient had a lethal cerebral infarction when the thrombostest values were above the therapeutic level; no other infarction happened during the treatment period. Moreover, the frequency of TIA decreased during the treatment, compared with descriptions of the natural course of TIA. One hundred four patients were observed for a mean of 21 months after the anticoagulant treatment ended. During the observation period, six patients had cerebral infarctions. This was a sixfold increase compared with the stroke incidence during treatment, and was almost identical with the incidence of strokes seen during the natural course of TIA. All the cerebral infarctions were in patients who had their initial TIA/TIA-IR from the carotid territory (within the same carotid artery which earlier had given symptoms).

The present investigation describes a group of patients with TIA and TIA-IR, and that this treatment should continue as long as the patients can manage it. In patients with vertebrobasilar symptoms of malignant character, it seems feasible to terminate the treatment after about one year. The mechanism of the anticoagulant treatment is obscure, but it does not appear to influence the progress of the atherosclerotic process.

The risk of development of a stroke is highest during the first months after the first TIA; it then decreases to about 5% to 6% per year during the next five years. There also is a difference between the number of TIAs and the risk of development of a definite cerebral infarction between the territory of the carotid and vertebrobasilar arteries, with a higher incidence of TIA and a lower incidence of cerebral infarction in the vertebrobasilar arterial system than in the carotid system. Descriptions of the natural history of patients with TIA-IR are lacking.

Methods

During five and one-half years (January 1, 1969 to June 30, 1974), 112 of 209 patients with TIA from the local area of the University Hospital of Lund (about 250,000 inhabitants) were selected and treated with anticoagulants. Thirty-three patients with carotid TIA (none with vertebrobasilar TIA) were surgically treated, whereas 64 were not treated with either anticoagulants or surgery. Forty-eight of the 64 patients had only slight symptoms of vertebrobasilar TIA; therefore, anticoagulant treatment was judged to be unnecessary (see below). The other 16 were judged to be unable to manage anticoagulant treatment either due to age (ad-
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