

COLOR DUPLEX ULTRASONOGRAPHY IN THE DIAGNOSIS OF TEMPORAL ARTERITIS

WOLFGANG A. SCHMIDT, M.D., HELGA E. KRAFT, M.D., KLAUS VORPAHL, M.D., LUTZ VÖLKER, M.D.,
AND ERIKA J. GROMNICA-IHLE, M.D.

ABSTRACT

Background The diagnosis of temporal arteritis usually requires a biopsy of the temporal artery. We examined the usefulness of color duplex ultrasonography in patients suspected of having temporal arteritis.

Methods In this prospective study, all patients seen in the departments of rheumatology and ophthalmology from January 1994 to October 1996 who had clinically suspected active temporal arteritis or polymyalgia rheumatica were examined by duplex ultrasonography. The final diagnoses, made according to standard criteria, were temporal arteritis in 30 patients, 21 with biopsy-confirmed disease; polymyalgia rheumatica in 37; and negative histologic findings and a diagnosis other than temporal arteritis or polymyalgia rheumatica in 15. We also studied 30 control patients matched for age and sex to the patients with arteritis. Two ultrasound studies were performed and read before the biopsies; one ultrasonographer was unaware of the clinical information.

Results In 22 (73 percent) of the 30 patients with temporal arteritis, ultrasonography showed a dark halo around the lumen of the temporal arteries. The halos disappeared after a mean of 16 days (range, 7 to 56) of treatment with corticosteroids. Twenty-four patients (80 percent) had stenoses or occlusions of temporal-artery segments, and 28 patients (93 percent) had stenoses, occlusions, or a halo. No halos were identified in the 82 patients without temporal arteritis; 6 (7 percent) had stenoses or occlusions. For each of the three types of abnormalities identified by ultrasonography, the interrater agreement was ≥ 95 percent.

Conclusions There are characteristic signs of temporal arteritis that can be visualized by color duplex ultrasonography. The most specific sign is a dark halo, which may be due to edema of the artery wall. In patients with typical clinical signs and a halo on ultrasonography, it may be possible to make a diagnosis of temporal arteritis and begin treatment without performing a temporal-artery biopsy. (N Engl J Med 1997;337:1336-42.)

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TEMPORAL arteritis is sometimes diagnosed clinically, but a temporal-artery biopsy is generally recommended to confirm the diagnosis. The American College of Rheumatology requires three of the following five criteria to be met to establish the diagnosis: age ≥ 50 years, new onset of localized headache, temporal-artery tenderness or decreased pulse, erythrocyte

sedimentation rate ≥ 50 mm per hour, and histologic findings.¹ Although temporal-artery biopsy is a minor operation, not all patients agree to this procedure. There are occasional complications, including damage of the facial nerve,² skin necrosis,³ drooping of the eyebrow,⁴ and stroke due to an interruption of a collateral circulation.⁵ It takes time to obtain the histologic results, and they are sometimes normal because the biopsy specimen was obtained from an area without lesions.

Color duplex ultrasonography is an excellent method of examining blood vessels. It combines the imaging capabilities of B-mode ultrasonography with the flow-velocity determinations of Doppler ultrasonography and permits accurate assessment of both the arterial anatomy and the flow characteristics of the vessel at specific sites. The new high-frequency ultrasound scanners with a high resolution make it possible to examine even small vessels such as the superficial temporal artery, which is located 3 to 4 mm below the surface of the skin. We have observed a hypoechoic halo around the perfused lumen of inflamed temporal arteries, which disappeared with treatment.⁶ We undertook this study to assess the use of this method in the diagnosis of temporal arteritis.

METHODS

Patients

All consecutive patients seen in the departments of rheumatology and ophthalmology between January 1994 and October 1996 with clinically suspected active temporal arteritis and polymyalgia rheumatica as well as all patients who had temporal-artery biopsies to rule out the condition were included in the study. Once a diagnosis was established, patients were classified according to whether they had temporal arteritis, polymyalgia rheumatica, or negative histologic findings and a diagnosis other than temporal arteritis or polymyalgia rheumatica. In addition, control patients matched for age and sex to the patients with temporal arteritis were included. Patients with a history of temporal arteritis but no evidence of active disease and patients who had had a previous biopsy of the temporal arteries were not included.

Patients with Temporal Arteritis

Thirty patients (21 of them women) with a mean age of 73 years (range, 52 to 86) had active temporal arteritis. Five had not

From the Clinic of Rheumatology, Berlin-Buch (W.A.S., E.J.G.-I.), and the Departments of Ophthalmology (H.E.K.), Pathology (K.V.), and Angiology (L.V.), Klinikum Buch — both in Berlin, Germany. Address reprint requests to Dr. Schmidt at the Clinic of Rheumatology, Zeperner Strasse 1, 13125 Berlin, Germany.

received corticosteroids before undergoing ultrasonography, 10 had received corticosteroids for less than 24 hours beforehand, 11 had received corticosteroids for 1 to 10 days beforehand, and 4 had been treated with doses of prednisolone that were too low to suppress disease activity. They all met at least three criteria for the diagnosis of temporal arteritis¹: 7 patients met three of the criteria, 9 met four, and 14 met all five criteria. The clinical diagnosis was confirmed by two rheumatologists and one ophthalmologist who examined every patient.

Twenty-seven patients underwent biopsy, and three declined to undergo biopsy. The findings were positive in 21, and negative in 4. In two patients the biopsy yielded insufficient material for analysis.

Clinically, 16 patients had symptoms of temporal arteritis and polymyalgia rheumatica, 12 of temporal arteritis alone, and 2 of polymyalgia rheumatica alone. The last two were classified as having temporal arteritis on the basis of histologic findings. Thirteen patients (43 percent) had visual disturbances: seven had definite anterior ischemic optic neuropathy (repeated episodes in three patients and affecting both eyes in two), three had amaurosis fugax (repeated episodes in one), and one had a single definite episode of occlusion of the central artery of one eye (which did not resolve), one had a single transitory episode of cotton-wool exudates affecting both eyes, and one had a single transient episode of bilateral abducens paralysis.

Patients with Polymyalgia Rheumatica

Thirty-seven patients (28 women and 9 men; mean age, 70 years; range, 51 to 86) were given a diagnosis of polymyalgia rheumatica because they met three or more of the seven criteria of the Bird classification.⁷ Four of the patients met three criteria, 12 met four, 9 met five, 10 met six, and 2 met all seven criteria. All patients met fewer than three of the criteria for temporal arteritis.¹ The diagnosis was confirmed by two rheumatologists who examined every patient. Seven patients had biopsies of the temporal arteries, all of which were negative. No patient had been treated with corticosteroids for more than 10 days. Sixteen patients had received corticosteroids for 1 to 10 days before ultrasonography, 7 had received corticosteroids for less than 24 hours beforehand, and 14 had not received corticosteroids beforehand.

Control Patients

Thirty patients with an established diagnosis of rheumatoid arthritis and no signs of temporal arteritis were chosen from the clinic of rheumatology and matched for age and sex to the patients with temporal arteritis. Fifteen patients received corticosteroids before undergoing ultrasonography.

Patients with Negative Histologic Findings and Diagnoses Other Than Temporal Arteritis or Polymyalgia Rheumatica

Fifteen patients (six women and nine men; mean age, 70 years; range, 49 to 90) were given the following diagnoses after completing the diagnostic procedure: noninflammatory anterior ischemic optic neuropathy in six, bronchial carcinoma in two, and noninflammatory central-artery occlusion, stroke, Hodgkin's lymphoma, glioma plus osteomyelitis, plasmacytoma, and idiopathic abducens paralysis in one patient each. The diagnosis remained undefined in one patient. Four patients received corticosteroids for 1 to 10 days before undergoing ultrasonography.

Ultrasonographic Evaluation

Simultaneous color Doppler and duplex ultrasonography was performed with a high-resolution linear scanner (L, 10 to 5; 10 to 5 MHz; length of scanner, 38 mm; ATL Ultramark 9 HDI, Advanced Technology Laboratories, Bothell, Wash.). We examined both common superficial temporal arteries and the frontal and parietal rami as completely as possible in a longitudinal and transverse plane to see whether they were perfused, whether there

was a halo around the lumen, and (using simultaneous pulsed-wave Doppler ultrasonography) whether there was a stenosis. Stenosis was considered to be present if blood-flow velocity was more than twice the rate recorded in the area before the stenosis, perhaps with wave forms demonstrating turbulence and reduced velocity behind the area of stenosis. In addition, the diameters of the systolic lumen and wall as well as peak systolic blood-flow velocity were measured at four defined points on each side of the head (Fig. 1).

Two trained physician ultrasonographers performed the examination, which takes an experienced sonographer 20 to 30 minutes. One investigator, who was unaware of the patients' diagnoses, looked only for the existence of a halo, stenosis, or occlusion. If their results disagreed, the investigators examined the results together and reached a consensus on the findings.

The arterial wall was defined to include the intima, media, adventitia, and temporal fascia. The wall could not be seen in the proximal common superficial temporal artery, which is located outside the temporal fascia. The anterior and posterior diameters of the wall were measured throughout the longitudinal plane, and the mean value was calculated as the diameter of each point. When there was a stenosis at one of these points the measurement was made 3 mm proximal to it. Ultrasonography was always performed before biopsy. If there was a halo, both investigators repeated the ultrasound examination every three to four days until the halo disappeared.

For the measurements, the settings on the scanner were always uniform. The settings for color Doppler sonography with our ATL scanner were as follows: dynamic range, 50 dB; signal-processing characteristic, G6; dynamic-contrast gain, K4; color gain, 78 percent; type of color gain, V; color scale, 4; color sensitivity, 10; color wall filter, 100 Hz; color persistence, 4; pulse-repetition frequency, 2500 per second; dynamic-movement differentiation, D3; and focus-point position, 7 mm.

Biopsy

In all cases biopsies were bilateral. Temporal arteritis was diagnosed if there was vasculitis with a predominance of mononuclear-cell infiltration or granulomatous inflammation with or without giant cells.¹

Statistical Analysis

The SPSS statistical package was used for statistical analysis. The Mann-Whitney U test or the chi-square test was used to compare the results between groups.

RESULTS

Figure 1 shows the diameters of the lumen and wall as well as peak systolic blood-flow velocity at four defined points along the superficial temporal artery in the four groups of subjects. The diameter of the artery wall was significantly larger in patients with temporal arteritis than in those in the other three groups. The diameters of the temporal arteries, including the lumen and wall, measured by ultrasonography can be compared with those determined in anatomical studies. Stock et al.⁸ recorded the following diameters in cadavers: common superficial temporal artery, 2.03 mm; frontal branch, 1.74 mm; and parietal branch, 1.83 mm. The mean (\pm SD) diameters in all subjects except the patients with temporal arteritis were as follows: common superficial temporal artery, 1.7 ± 0.43 mm (our value was smaller because we could not see the wall); frontal ramus (1 cm distal to the bifurcation), 2.2 ± 0.38 mm; and

Site	Patients with Temporal Arteritis (N=30)	Patients with Polymyalgia Rheumatica (N=37)	Control Subjects (N=30)	Patients with Negative Histologic Findings and Other Diagnoses (N=15)
Parietal ramus (15 mm distal to bifurcation)				
Systolic lumen (mm)	0.79±0.29	0.76±0.20	0.89±0.24	0.81±0.30
Wall (mm)	0.94±0.28*	0.70±0.08	0.72±0.13	0.79±0.11
Maximal velocity (cm/sec)	52±18	59±14	54±14	57±18
Frontal ramus (25 mm distal to bifurcation)				
Systolic lumen (mm)	0.67±0.20	0.66±0.22	0.74±0.24	0.68±0.23
Wall (mm)	0.95±0.20*	0.66±0.07	0.65±0.13	0.72±0.09
Maximal velocity (cm/sec)	48±13	53±16	47±15	55±19
Frontal ramus (10 mm distal to bifurcation)				
Systolic lumen (mm)	0.74±0.24	0.71±0.17	0.86±0.26	0.78±0.30
Wall (mm)	0.95±0.22*	0.69±0.09	0.71±0.13	0.76±0.10
Maximal velocity (cm/sec)	50±14	56±15	48±13	59±20
Common superficial temporal artery (8 mm below skin surface)				
Systolic lumen (mm)	1.51±0.44	1.54±0.41	1.70±0.35	1.85±0.54
Maximal velocity (cm/sec)	62±22	61±16	55±13	64±16

Figure 1. Measurement of the Superficial Temporal Arteries.

Plus-minus values are means ±SD of the right and left sides. The arterial wall was defined to include the intima, media, adventitia, and temporal fascia. Asterisks indicate a significant difference ($P < 0.01$ by the Mann-Whitney U test) between the patients with temporal arteritis and the other three groups.

parietal ramus (1.5 cm distal to the bifurcation), 2.3 ± 0.38 mm (our value was higher because we also measured the temporal fascia).

Unexpectedly, there was no significant difference between the group with temporal arteritis and the other three groups with respect to the diameter of the systolic lumen and maximal velocity of systolic blood flow along the nonoccluded segments. Men tended to have bigger lumina and higher velocities than women. In some regions of measurement the difference between men and women was significant. The forms of the Doppler curve did not differ significantly between the groups.

Of the 30 patients with temporal arteritis, 22 (73 percent) had a hypochoic halo around the perfused lumen of the temporal arteries (Fig. 2). The sagittal diameter of the halo was between 0.3 and 1.2 mm. On the anterior and posterior sides of the halo a hyperechoic region remained. We assume that this structure is the temporal fascia, which is not involved in the inflammatory process. The halo was bilateral in 17 patients. The common superficial temporal artery was involved in 15 patients, the frontal ramus in 16, and the parietal ramus in 14. Five patients had the halo in all eight segments analyzed (the common superficial temporal artery, proximal frontal ramus, distal frontal ramus, and parietal ramus on both sides of the head), one had it in seven segments, four had it in six segments, one had it in five segments, three had it in four segments, one had

it in three segments, three had it in two segments, and four had it in one segment. On repeated ultrasonography, the halos were always seen in the same segments.

The halo disappeared a mean of 16 days (range, 7 to 56) after the start of therapy with corticosteroids, with no significant difference between the investigators in the mean values. After the biopsy the lumen was smaller and velocity was slower in the ramus proximal to the site of the biopsy. None of the 82 patients in the other three groups had a halo around the perfused lumen of the temporal arteries; thus, this finding seems to be specific for temporal arteritis.

Table 1 compares the results of ultrasonography with those obtained by histologic analysis. Table 2 shows the sensitivity and specificity of the test.

In two patients a halo was seen in the proximal frontal ramus, but the histologic findings were negative in the biopsy specimens, which were taken from another location to avoid the branches of the facial nerve at the proximal frontal ramus. Both patients (age, >50 years) had a clinically established diagnosis of temporal arteritis with new localized headache, tender temporal arteries with decreased pulse, and elevated erythrocyte sedimentation rates. The diagnosis has remained unchanged after more than one year. Both patients had received prednisolone in the past, and the daily doses had been reduced to 5 mg and 2 mg. The discrepancy between the ultrasonographic and histologic findings may be due to the low dose

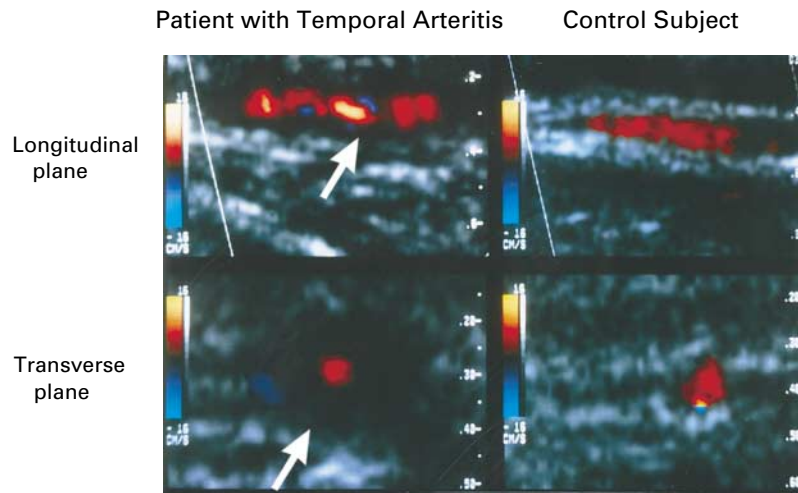


Figure 2. Parietal Ramus of the Superficial Temporal Artery in a Patient with Temporal Arteritis and a Control Subject in a Longitudinal and a Transverse Plane. The hypochoic (black) area is indicated by the arrows.

of corticosteroids and, perhaps, to the presence of segmental temporal arteritis.

In 12 of the 16 patients with both positive biopsy findings and a halo on ultrasonography, the biopsy specimen included the region with the halo. All 12 biopsy specimens had mononuclear-cell infiltrates, and 9 of the 12 contained giant cells. There was no association between the presence of a halo and the intensity of mononuclear-cell infiltration, involvement of the intima, or the presence of granulomas or giant cells. Three patients underwent biopsy of occluded segments, which showed giant cells and obliteration due to inflammation.

It was possible to detect nonperfused vessels. The superficial temporal artery is located between the superficial and profound laminae of the temporal fascia. Both laminae are usually found close together but separate at the temporal artery. It is possible to locate the artery at the point of separation by normal B-mode ultrasonography even without color Doppler ultrasonography. Color Doppler ultrasonography is necessary, however, to show whether the lumen is perfused and whether a halo is present.

Patients with polymyalgia rheumatica, control subjects, and patients with negative histologic findings and a diagnosis other than temporal arteritis or polymyalgia rheumatica had significantly fewer stenoses and occlusions than the patients with temporal arteritis ($P < 0.01$ by the chi-square test). Two patients with polymyalgia rheumatica had occlusions; one had negative biopsy findings, and the other had not undergone biopsy. Two control subjects had stenoses, and two had occlusions. All four of these control subjects had arteriosclerotic disease. None of the

4 and none of the 10 other subjects with arteriosclerotic disease but not temporal arteritis had a halo.

The rates of agreement between the two ultrasonographers were 100 percent for the initial diagnosis of the halo (112 of 112 tests), 98 percent for stenoses (110 of 112), 96 percent for occlusions (108 of 112), and 95 percent for all three features (106 of 112). Each investigator missed one apparent stenosis and misidentified two areas as occlusions.

Among the patients with temporal arteritis, 11 with a halo and 2 without a halo had eye involvement; 11 with a halo and 6 without a halo had no eye involvement (P not significant by the chi-square test). Among the patients with temporal arteritis, there was no relation between the presence of a halo and the clinical appearance. Of the 22 patients with a halo, 9 had symptoms of isolated temporal arteritis, 12 of temporal arteritis and polymyalgia rheumatica, and 1 of isolated polymyalgia rheumatica. Of the eight patients without a halo, three had symptoms of isolated temporal arteritis, one of polymyalgia rheumatica, and four of both syndromes. Thirteen patients with a halo had hard and prominent temporal arteries, whereas this clinical sign was not found in nine with a halo. Two patients with hard and prominent arteries had no halo; six had neither a halo nor hard and prominent arteries.

There was no association between the presence of a halo and the erythrocyte sedimentation rate. One patient with no visual disturbance had a unilateral headache, with the halo found only on that side. The other patients reported bilateral headache. A halo was found to be topographically related to a diffuse swelling of the right frontal region in another patient.

TABLE 1. COMPARISON OF RESULTS OBTAINED BY ULTRASONOGRAPHY AND HISTOLOGIC ANALYSIS IN 30 PATIENTS WITH TEMPORAL ARTERITIS.*

NO. OF PATIENTS	ULTRASONOGRAPHY		HISTOLOGIC ANALYSIS
	HALO	STENOSIS OR OCCLUSION†	
14	+	+	+
3	+	+	ND
2	+	-	+
1	+	-	ND
1	+	+	-
1	+	-	-
4	-	+	+
1	-	+	-
1	-	+	ND
1	-	-	+
1	-	-	-

*ND denotes not done or that there was insufficient material for analysis. A plus sign indicates positive findings, and a minus sign negative findings.

†Twelve patients had both stenosis and occlusion, six patients had occlusion alone, and six patients had stenosis alone.

TABLE 2. SENSITIVITY AND SPECIFICITY OF DUPLEX ULTRASONOGRAPHY OF THE TEMPORAL ARTERIES FOR THE DIAGNOSIS OF TEMPORAL ARTERITIS AND TO CONFIRM HISTOLOGIC FINDINGS.

FINDING	DIAGNOSIS*		CONFIRMATION OF HISTOLOGIC FINDINGS†	
	SENSITIVITY	SPECIFICITY	SENSITIVITY	SPECIFICITY
	positive tests/total (%)	negative tests/total (%)	positive tests/total (%)	negative tests/total (%)
Halo	22/30 (73)	82/82 (100)	16/21 (76)	24/26 (92)
Stenosis or occlusion	24/30 (80)	76/82 (93)	18/21 (86)	23/26 (88)
Halo, stenosis, or occlusion	28/30 (93)	76/82 (93)	20/21 (95)	22/26 (85)

*Thirty patients had temporal arteritis, and 82 patients had been given other diagnoses.

†Twenty-one patients had positive histologic findings, and 26 patients had negative histologic findings (4 in the temporal-arteritis group, 7 in the group with polymyalgia rheumatica, and 15 with other diagnoses).

DISCUSSION

Of the 30 patients we studied with temporal arteritis, 73 percent had a hypoechoic halo around the perfused lumen that disappeared with treatment. At first we thought that this halo might be due to cell infiltrates, but cell infiltrates may be absent from the intima, and the hypoechoic region always began right next to the perfused lumen. In nine of our patients with temporal arteritis we found an intima without cell infiltrates, yet seven of these patients had a halo. We believe that the halo is a sign of fluid in the artery wall. Indeed, edema has been described in temporal arteritis,⁹ although it is difficult to document histologically. We think that ultrasonography identifies a different aspect of the disease (edema) than histologic analysis (cell infiltrates), yet both types of findings may be signs of arteritis.

Certain factors could have increased the frequency of the halo in our group. Only 7 of the 37 patients with polymyalgia rheumatica underwent biopsy; therefore, some additional patients might have been classified as having temporal arteritis if they had had biopsies. Many (43 percent) of our patients with temporal arteritis had visual symptoms, so we might have overestimated the incidence of the halo as a result of studying so many patients with severe disease. The diagnosis was confirmed by biopsy in only 21 of the 30 patients with temporal arteritis. With respect to the clinical diagnoses, bias may have resulted from the fact that those who made the diagnosis were in many cases aware of the results of the ultrasound studies.

Angiography has been used in patients with temporal arteritis to pinpoint the region for biopsy and to detect stenoses and occlusions,¹⁰⁻¹³ and conventional Doppler ultrasonography has been studied as well.¹⁴⁻²⁷ A Doppler ultrasound flow detector was used to identify the anatomical location of the temporal arteries preoperatively,¹⁴ and such an approach can show that there are more stenoses and occlusions in patients with biopsy-proved temporal arteritis than in patients with negative histologic findings.^{15,17} In three studies of 30 to 59 patients with temporal arteritis, Doppler ultrasonography identified abnormalities in 77 to 90 percent of the patients.^{24,25,27} These findings can be compared with our results (stenosis or occlusion was identified by pulsed-wave Doppler ultrasonography in 80 percent of patients) (Fig. 3). In contrast to conventional Doppler ultrasonography, duplex ultrasonography can distinguish between aplasia and occlusion of the vessel because of the imaging capabilities of the B-mode ultrasound. The superficial temporal artery was found to have an abnormal course in 0 to 8 percent of cadavers.^{8,28,29} Stenoses or occlusions may also occur in persons without temporal arteritis, although they are less common. Continuous-wave Doppler ultra-

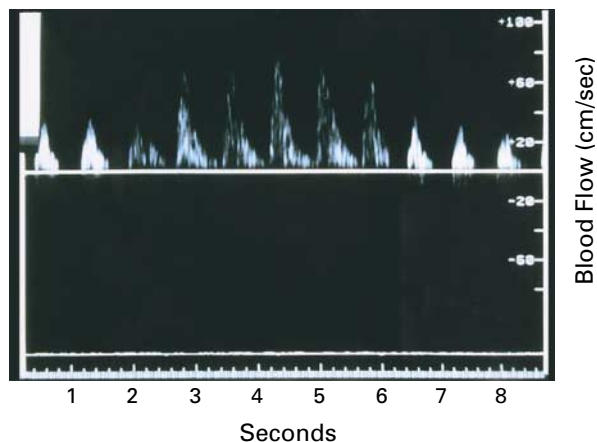


Figure 3. Stenosis of the Superficial Temporal Artery. On the left-hand side, blood flow before the stenosis is normal. As the pulsed-wave Doppler beam moves through the stenosis, blood flow velocity increases. In the area to the right beyond the stenosis, blood flow velocity again decreases.

sonography identified abnormalities in 9 to 18 percent of control subjects^{18,25} and in 20 to 24 percent of patients with negative histologic findings who were suspected of having temporal arteritis.^{18,24,27} In our study, pulsed-wave Doppler ultrasonography also identified abnormalities in some of the patients without temporal arteritis (Table 2). The use of duplex ultrasonography can increase the sensitivity and specificity of this approach by its ability to visualize the halo around perfused lumina of inflamed arteries. Orbital duplex ultrasonography can demonstrate reduced blood flow, stenoses, and occlusions, especially in the central retinal and posterior ciliary artery, in patients with temporal arteritis.³⁰⁻³⁵

An investigation of the superficial temporal arteries by duplex ultrasonography requires a linear scanner with a high resolution in the area that is 3 to 10 mm below the surface of the skin. We tried several 7.5-MHz scanners and found that it was very difficult to visualize the artery adequately with them. In most cases it was difficult to find especially narrow rami and to be sure that a halo was actually present, since it is sometimes quite small.

There are reports of the use of histologic analysis of temporal arteries to diagnose Wegener's granulomatosis,^{36,37} polyarteritis nodosa,³⁸ Churg-Strauss syndrome,³⁹ and primary amyloidosis.⁴⁰ Certainly, ultrasonography cannot be used to differentiate between those diagnoses and temporal arteritis, but such syndromes are clinically very rare in comparison with typical temporal arteritis.

We suggest that duplex ultrasonography of the temporal arteries can be used in the diagnosis of temporal arteritis and polymyalgia rheumatica as fol-

lows. Patients with typical clinical signs of temporal arteritis and a clear halo on ultrasonography might be treated without a biopsy, unless there is a reason to suspect another form of vasculitis. Patients with strong clinical evidence of temporal arteritis who have only stenoses or occlusions or no abnormalities on ultrasonography should still undergo biopsy. Patients with clinical signs of polymyalgia rheumatica who have no symptoms of temporal arteritis but who have abnormal findings on ultrasonography (a halo, stenosis, or occlusion) should undergo biopsy and be treated with a higher initial dose of corticosteroids to protect against blindness, at least until the biopsy results are known. In patients with equivocal clinical evidence of temporal arteritis, ultrasonography should be particularly helpful.

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