

smooth pursuit gaze opposite from the lesion cleared within weeks.

It appears that an acute, reversible loss of pursuit gaze contralateral to the lesion can occur following ablative lesions of the occipitoparietal region, and that a more chronic and partial defect in pursuit gaze ipsilateral to the lesion may persist. The acuteness and reversibility of this contralateral pursuit gaze paresis is comparable with the similarly short-lived loss of contralateral saccadic gaze occurring after a more anterior and global destruction of the frontal lobes in the middle cerebral artery distribution.³

It seems that ipsilateral and contra-

lateral, conjugate horizontal eye deviation can arise from either posterior hemisphere. The gaze functions of the posterior hemispheres under normal conditions have yet to be determined.

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Right Hemifield Alexia Without Hemianopia

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● We studied a 54-year-old man who suffered a stroke in the visual-association areas of the left hemisphere. He showed no right-sided visual field defect and results of a neuropsychologic examination were normal except for his performance in several tasks presented to the right visual field. Naming of drawings was normal in both hemifields but reading of letters, words, digits, and arithmetic symbols was defective in the right field. We believe that the lesion in the visual association areas of the left hemisphere that did not interrupt interhemispheric connections disconnected the primary visual areas from temporoparietal areas of the left hemisphere, affecting reading.

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Although pure alexia is generally accompanied by right-sided visual field defects, this is not a crucial feature of the syndrome, but rather a neighboring sign consequent to the extension of the lesion to the calcarine cortex or optic radiations.

Pure alexia can be the result of lesions that disconnect the pathways from both occipital lobes to the parietotemporal association areas responsible for visuoverbal association

tasks.¹⁻³ Such lesions are located in the left occipital region. Alexia can also be confined to the left visual field in callosotomized patients because visual information cannot travel from the right occipital lobe to the dominant parietotemporal association areas.⁴

Recent evidence supports the notion that specific regions of the visual association cortex may be specialized in the processing of specific types of stimuli or tasks. Strategically located lesions in peristriate regions can independently affect functions such as reading, visual object naming, color perception and face recognition.⁵⁻⁷ Such lesions can spare the optic radiations, primary calcarine cortex, and even callosal outflow. The resulting syndromes can thus be confined to an otherwise intact visual hemifield.

REPORT OF A CASE

A 54-year-old right-handed office clerk was referred to our laboratory because of reading difficulties. While at work one year before admission, he suddenly complained of dizziness, headache, and nausea and noted that he could not see in the right half of his visual field. His visual difficulties improved within a few days. It is unclear if during that period he was able to read, because he apparently did not attempt to do so. On returning to work he was able to read but did so slowly because he missed words and letters and had to return to the beginning of sentences to grasp the meaning of the material. These difficulties improved during the following year but, based on his testimony, he never regained his previous reading ability. He

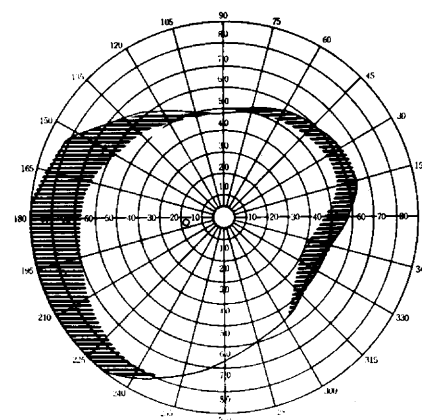


Fig 1.—Visual field of left eye.

never complained of other neurologic symptoms.

Twenty years earlier he had sustained a traumatic lesion of the right eye that rendered him completely blind on that side.

Results of a neurologic examination were normal, including examination of the visual fields (Fig 1).

A computed tomographic (CT) scan showed an area of hypodensity in the mesial and inferior left occipital lobe extending deeply into the white matter. It appeared to spare the calcarine cortex and the forceps major (Fig 2).

As assessed by the standard neuropsychological battery of the Language Research Laboratory,^{8,9} there was no evidence of aphasia or apraxia. Constructional ability, memory, and calculation were normal. The patient could read letters, words, and text without errors, although he complained of some difficulty in reading the newspaper, because his reading was slower than before. Spontaneous writing, copying, and writing to dictation were nor-

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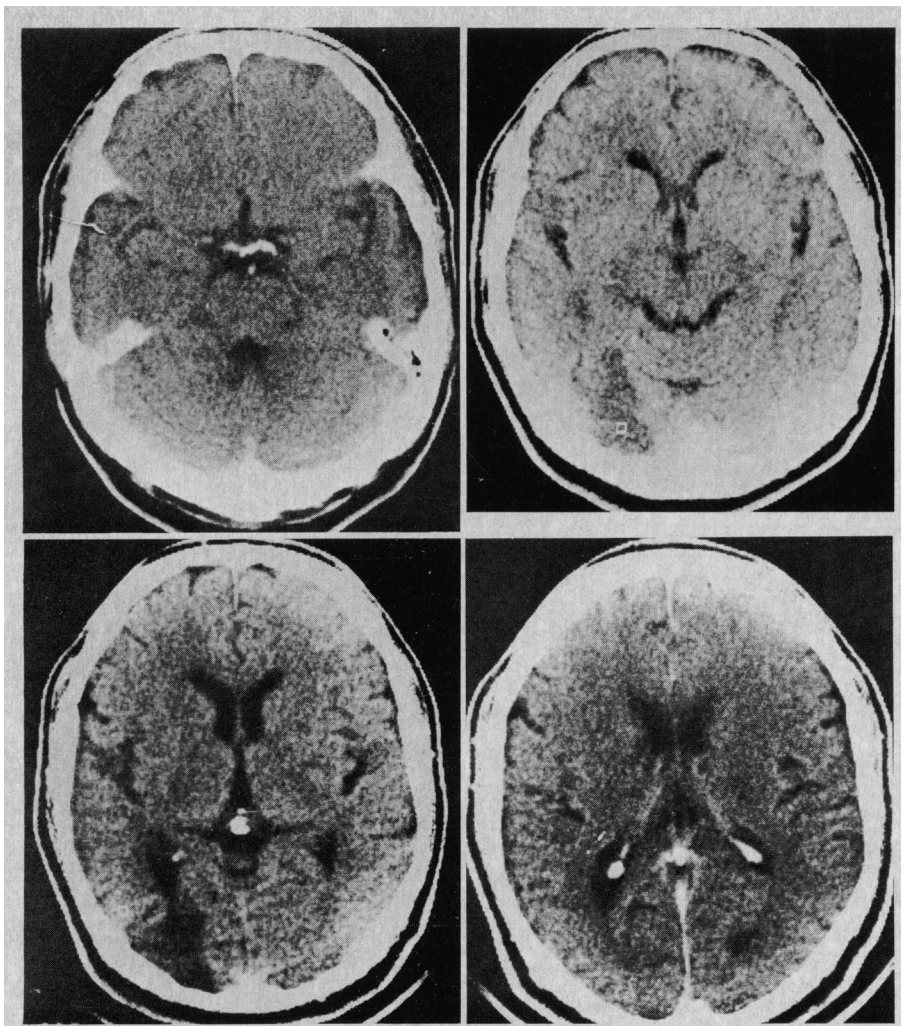


Fig 2.—Computed tomographic scan showing lesion on left occipital lobe sparing primary visual cortex and corpus callosum.

ing tasks: (1) naming of colors was defective in both hemifields but more disturbed on the right (12 [28%] *v* three (7%) of correct answers in a total of 42 stimulations); (2) naming of single letters was better when presented to the left field (26 [65%] *v* 14 [35%] in a total of 40 presentations); (3) reading of two-letter words was impossible in the right field and six (50%) were correctly read in the left field (in a total of 12 presentations); (4) naming of digits was also better in the left field (ten [63%] *v* two [12%] in a total of 16 stimulations); and (5) naming of arithmetic symbols was normal in the left field and impossible in the right (in a total of ten exposures). When the patient failed to name correctly the presented stimuli he was asked verbally to choose the items he did not name from a multiple choice card on which all of the stimuli were represented. He was able to match 21 of the 30 colors he could not name when stimuli were presented in the left field and 24 of the 39 he could not name when stimuli were presented in the right field. He was unable to match letters, words, digits, or arithmetic symbols he could not name (results were thus similar both for naming and matching tasks). When meaningless drawings were presented in each hemifield he could easily select the correct items from a multiple choice card.

Finally, pairs of different and similar stimuli were presented simultaneously in both hemifields (eg, red in the right field and green in the left or green in both hemifields). The patient was asked to state if the stimuli presented were similar or different. Performance was normal for colors and nonverbal stimuli and a chance score was obtained when pairs of letters were presented.

COMMENT

Based on the several available reports on pure alexia it is possible to postulate some of the areas and pathways involved in reading ability. Visual information arriving at the primary visual cortex proceeds to peristriate association areas in each hemisphere. From there it travels to the left parietotemporal cortex, from the left occipital lobe through the white matter underlying the occipitoparietal regions, and from the right occipital lobe through the inferior splenium of the corpus callosum. Three mechanisms have been postulated by Geschwind¹² for the right occipital lobe-inferior splenium pathway: (1) the pathway proceeds from the right Brodmann's area 17 to the right visual association areas, from there to the right angular gyrus, and finally across the corpus callosum to the left angular gyrus (Fig 3, A); (2) the pathway runs from the right Brodmann's area 17 to the right association areas, then crosses the callosum to the left visual association

Results of Tachistoscopic Tests			
Task	Field, No. Correct/ No. Presented (%)		χ^2
	Left	Right	
Naming of drawings	27/36 (75)	21/36 (58)	2.25, NS
Naming of colors	12/42 (28)	3/42 (7)	6.57, <i>P</i> < .025
Naming of single letters	26/40 (65)	14/40 (35)	7.20, <i>P</i> < .01
Reading of words	6/12 (50)	0/12 (0)	8.00, <i>P</i> < .005
Naming of digits	10/16 (63)	2/16 (12)	8.53, <i>P</i> < .005
Naming of arithmetic symbols	10/10 (100)	0/10 (0)	20.00, <i>P</i> < .005

mal and the patient felt that he performed as well as ever. Naming of digits, numbers, arithmetic symbols, geometric shapes, colors, familiar faces, European traffic signs, and photographs of city landmarks was performed accurately. His score was normal in the face recognition test¹⁰ and he had no abnormalities in the Ishihara test.¹¹ He then performed several tasks during a tachistoscopic examination of both hemifields.

Stimuli were presented in a tachistoscope at an exposure of 100 ms. Randomized stimuli were given 2.5° to the right

and to the left of the fixation point. Stimuli presented to the left field were always also presented to the right. The same series of stimuli were presented several times in different days. Performances were consistent in each trial. Results are summarized in the Table. For each task the total number of correct answers in all trials was considered. The patient correctly named 27 (75%) of the drawings presented to the left field and 21 (58%) of those presented to the right in a total of 36 stimulations, which can be considered a similar performance. Differences were obtained for the remain-

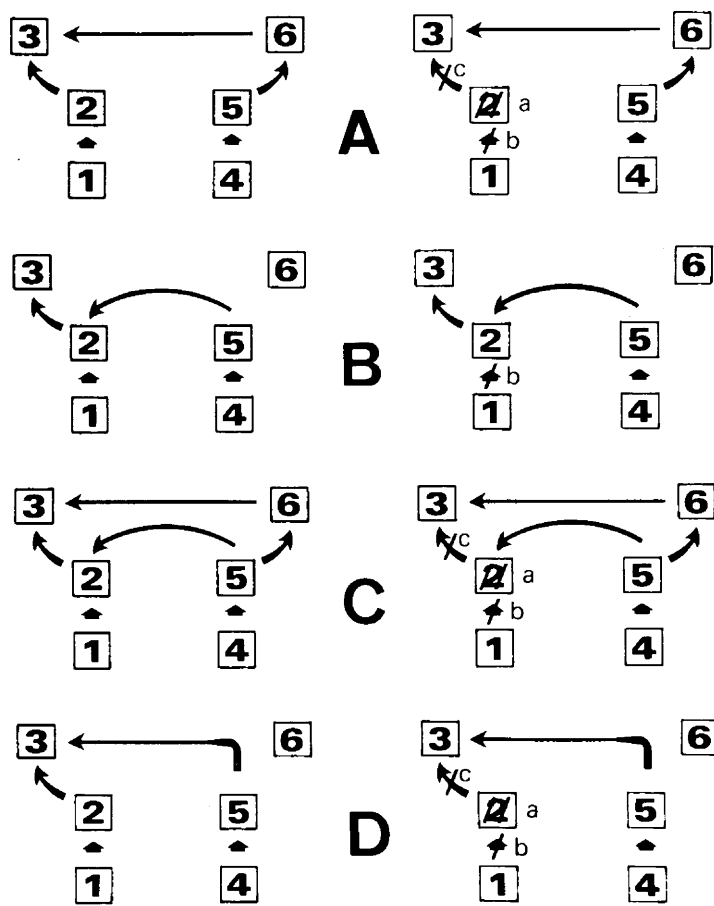


Fig 3.—Schematic representation of mechanism of connections necessary for reading. Left, Four possible mechanisms. Right, Interpretation of present case according to each mechanism. 1 indicates left visual primary cortex; 2, left visual association cortex; 3, left parietotemporal cortex; 4, right visual primary cortex; 5, right visual association cortex; and 6, right parietotemporal cortex. a, b, and c indicate possible localization of damage.

areas, and finally runs forward to the left angular gyrus (Fig 3, B); and (3) both pathways are used (Fig 3, C). A possible fourth mechanism, based on a model of nonhomotopic interhemispherical connections, would call for a pathway running from the right Brodmann's area 17 to the right association areas, then crossing the callosum directly to the left angular gyrus (Fig 3, D).

The most prominent clinical features of the present case were: (1) absence of abnormal findings in an open-field neuropsychologic examination; (2) alexia for letters, words, digits, and arithmetic symbols presented in the right visual field (the defect was present in naming and matching tasks); and (3) anomia for colors presented in the left and in the right visual fields (although less evident for colors presented in the left field) (color-matching tasks were better performed than color naming).

The CT scan showed a lesion in the left occipital lobe that was damaging visual association areas and the underlying white matter. The primary visual cortex, the corpus callosum, and the forceps major seemed to be spared.

Considering the first mechanism proposed (Fig 3, A), the lesion in this patient could have damaged the left visual association cortex (Fig 3, a) or disconnected the primary and association visual cortices (Fig 3, b). In either case, the cascade processing of visual information in the left occipital lobe was stopped, preventing the decodification of visual symbols presented to the right visual hemifield. However, right-left connections remained operative, allowing reading in the left field. The information processed in the right occipital lobe could be transported to the left parietotemporal cortex through the corpus callosum. The connections between the left

visual association cortex and the left parietotemporal cortex could have been damaged, with preservation of the right-left connections (Fig 3, c) but that would not explain the patient's inability to match letters presented to the right visual field. If the lesion spared the mechanisms of visual information processing in the left occipital lobe, the patient, although unable to perform visuo-verbal associations due to a disconnection between the left occipital cortex and the language areas, should still have been able to perform those associations without using language. He should thus have been able to match all material presented to the right hemifield.

If the second mechanism is considered (Fig 3), the lesion would be responsible for the disconnection between primary and association visual cortices, otherwise all the information arriving from the right hemisphere would not reach the left parietotemporal areas and the patient would have shown a typical pure alexia. However, the lesion seemed to be too large to be responsible for such a strategic localization.

For the last two mechanisms the explanation is similar to that used for the first: the lesion damaged the left visual association cortex or its connections with the primary visual cortex (Fig 3, C and D, and a and b). Disconnection of the visual association cortex from the parietotemporal cortex (Fig 3, c) is insufficient to explain the clinical picture, but could also be present.

We can conclude that all mechanisms except for the second (Fig 3, B) which seems improbable, are possible and that a lesion in the visual association cortex or in its connections with the primary cortex can cause an alexia confined to the right visual field.

The patient showed more intense defects in color naming than in the naming of letters in tachistoscopic presentation, although with the same asymmetry of performance between the two hemifields. This can be explained by the time required for color detection, which has proved to be longer even in normal subjects.¹³ However, he performed better in matching tasks of colors than in matching tasks of symbolic visual material. It is thus probable that he grasped information that was enough for nonverbal tasks (in particular those of matching) but insufficient for verbal ones. This means that his left occipital lobe could only treat color

information up to a certain point, that is, for tasks that did not require a verbal intervention.

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Transient Oculomotor Synkinesis in Temporal Arteritis

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● In a patient with temporal arteritis, ophthalmoplegia developed associated with a transient oculomotor synkinesis or aberrant regeneration. The synkinesis suggests that the ophthalmoplegia was neurogenic rather than myogenic. Based on published experiments, it appears that transient synkinesis is compatible with the hypothesis that aberrant regeneration of axons in the third nerve is the cause of oculomotor synkinesis.

(*Arch Neurol* 1984;41:87-88)

A patient with a pupil-sparing third-nerve palsy due to temporal arteritis subsequently had a transient anomaly of ocular motility, oculomotor synkinesis or aberrant regeneration, unique in this disease. The site of the lesion responsible for ophthalmoplegia in temporal arteritis remains somewhat controversial, but the occurrence of synkinesis following a third-nerve palsy suggests that ophthalmoplegia, in this case, was neurogenic.

REPORT OF A CASE

In late November 1980, a 63-year-old man had bifrontal pain that radiated to the malar regions and upper gingivae. The

pain was not aggravated by chewing or talking. He also had such severe scalp tenderness that contact with a pillow was intolerable. Over the ensuing weeks he lost 9 kg but had no fever, muscle aches, or visual impairment. His pain then became localized above the right eye and diplopia and ptosis of the right upper lid developed. On admission to a hospital, a pupil-sparing right-sided third-nerve paralysis was recognized, but the results of the remainder of his examination were unremarkable. Computed tomographic scan of the head, orbital venogram, and right carotid arteriogram were normal. The right superficial temporal artery was opacified on the arteriogram and appeared narrowed, but it was partially obscured by branches of the internal carotid artery. Fasting blood glucose levels were consistently elevated; two-hour postprandial blood glucose level was normal. Five Westergren ESRs ranged from 52 to 87 mm/hr. He was discharged with a diagnosis of diabetic ophthalmoplegia. Persistent severe pain requiring narcotic analgesics prompted referral.

Examination on Jan 23, 1981, showed that his acuity was correctable to 20/15 OU. He missed all but two of the 20 plates in the Ishihara series with the right eye and made no errors with the left. Exophthalmometry was 14 mm OD and 13 mm OS with normal orbital resilience. There was moderate ptosis of the right upper lid, and the lid elevated 2 mm on adduction and on downward gaze. He had impaired elevation, depression, and adduction of the right eye. Abduction and intorsion were normal. Pupillary size and motility were unremarkable. Visual field examination revealed a 2° central scotoma in the right eye to the 2-mm white object as 2 m. The results of the remainder of the neurologic and ophthalmologic examinations were unremarkable.

Shortly after being seen, the patient's ptosis and diplopia spontaneously im-

proved so that by March 6, 1981, ocular and lid motility seemed normal both to the patient and to his neurologist. The intensity of the pain diminished; however, he noticed that his scalp vessels had enlarged. A repeated ESR of 117 mm/hr led to a right-sided temporal artery biopsy, which immediately and permanently eliminated his pain. Biopsy showed giant cell arteritis. Treatment started on March 9, 1981, with oral prednisone, rapidly normalized his ESR. Repeated examination on March 20, 1981, showed that his visual acuity was 20/15 OU with normal color vision, full visual fields, and normal motility. Neither disc pallor nor thinning of the retinal nerve fiber layer could be discerned in the fundi.

COMMENT

Estimates of the prevalence of ophthalmoplegia in patients with temporal arteritis vary from 10% to 50%.¹⁻³ Fisher² has observed that the ophthalmoplegia tends to be manifested by ptosis, "partial third-nerve involvement," or, less commonly, abduction paresis. As in our patient, recovery tends to be complete and may occur without treatment. Barricks et al⁴ have reported autopsy findings in a patient with ophthalmoplegia consequent to temporal arteritis. The ophthalmoplegia was due to muscle ischemia. In addition, there are some clinical observations that suggest a myopathic basis for some cases of ophthalmoplegia in temporal arteritis. Wagener and Hollenhorst¹ noted daily variation in the muscles involved and severity of the ophthalmoplegia, which seemed more compatible with a myogenic rather than a neurogenic process. In some cases of ophthalmople-

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