

## Phonological Alexia with Optic and Tactile Anomia: A Neuropsychological and Anatomical Study

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We describe a patient with phonological alexia caused by a small hemorrhage in the posterior-inferior portion of the left temporal lobe. The lesion induced a highly selective impairment of phonological reading without concomitant oral language deficits other than anomia for objects presented in the visual and tactile modalities. We propose that an intact dorsal pathway from inferior visual association areas to Wernicke's area via the angular gyrus could mediate reading by the lexical route, while damage to a ventral pathway disrupted the patient's ability to read nonwords. We suggest further that although visually and tactually presented objects could be recognized and both verbally and nonverbally identified, they could not be named because of a disconnection from the area of word representations.

### INTRODUCTION

According to an information-processing model of reading, at least two separate reading strategies are available to normal readers. These are referred to as the lexical route and the nonlexical (or phonological) route (Coltheart, Patterson, & Marshall, 1980). When reading familiar words, skilled readers preferentially use the lexical route, which relies on a direct visual association between the written form of the word and its orthographic representation in the lexicon. The phonological representation of the word is accessed after its lexical recognition. This can be accomplished either by transcoding directly from the orthographic form of the word to its phonological form or by an indirect route to the phonological representation via semantics (Coltheart, 1984). The form of phonological

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coding that takes place subsequent to word recognition is referred to as postlexical, or addressed, phonology (Patterson, 1982).

The nonlexical phonological route presumably uses grapheme-phoneme conversion rules to arrive at the phonological representation of the word (Coltheart, 1978, 1980, 1984; but see Glushko, 1979, 1981, and Kay & Marcel, 1981, for an alternative hypothesis of phonological reading by lexical analogy). Grapheme-to-phoneme conversion rules can be applied to any letter string, regardless of whether it is part of one's lexicon, and therefore the phonological route is considered to be nonlexical. This type of phonological coding can also be used to achieve word recognition and is referred to as prelexical, or assembled, phonology (Patterson, 1982).

Although reading regular words can be successfully accomplished by using either route, irregular words can be read correctly only by the lexical route. Because nonwords have no representation in the lexicon, they can be read only by the nonlexical phonological route.

Research on acquired alexia has shown that brain damage can selectively impair a patient's ability to use one or the other of these routes. The type of reading disturbance that is thought to reflect such selective impairment of the nonlexical phonological route has been labeled phonological alexia by Beauvois and Déroutesné (1979), who first reported the syndrome. Patients with phonological alexia are severely limited in their ability to read nonwords, although they can still read both regular and irregular words. It has been postulated that these patients lost their ability to perform grapheme-phoneme conversions and therefore cannot use the nonlexical phonological strategy critical for reading nonwords (Beauvois & Déroutesné, 1979; Coltheart, 1984). Because reading by a lexical strategy is still possible, they can read both regular and irregular words.

Since the original publication by Beauvois and Déroutesné (1979), there have been several additional reports of this syndrome (see Sartori, Barry, & Job, 1984, for a review). However, the patients' reading abilities varied widely, and not all patients showed the dramatic dissociation between the reading of nonwords and real words that is the hallmark of this syndrome. In addition, most of the reported patients were aphasic; therefore, their errors in reading nonwords aloud may have been due not only to a failure to perform grapheme-to-phoneme conversion per se, but also to an aphasic speech defect. Last, most of these prior reports did not attempt to elucidate the anatomical substrate of this syndrome.

We recently observed a patient who had phonological alexia without an aphasic speech defect, thereby providing support for the postulate that aphasia and phonological alexia are dissociable phenomena. The patient described in this report had a small lobar hemorrhage in the posterior-inferior portion of the left temporal lobe. The fact that a relatively "pure" alexic syndrome was induced by a circumscribed lesion makes this case ideal for clinicoanatomical correlation.

## CASE REPORT

A 39-year-old right-handed hypertensive man suddenly collapsed in a restaurant and had a generalized tonic-clonic seizure. He was admitted to his local hospital in a postictal state and right-side paralysis was noted. Although detailed language evaluation was not carried out, spontaneous speech and comprehension were reported to be normal after his recovery from the postictal state. A few days later the patient was transferred to the Veterans Administration Medical Center in Gainesville. He was alert and oriented and did not complain of any language difficulties. Samples of free conversation, obtained while a detailed medical and social history was taken, disclosed that his spontaneous speech was fluent with good information content. There were no paraphasic errors, circumlocutions, or word-finding difficulties. Comprehension and repetition were intact. However, his performance on confrontation naming was impaired. (A detailed analysis of his language functions and the description of his reading and writing abilities is provided in the section on neuropsychological studies.) He was not apraxic and did not have any elements of the Gerstmann syndrome except for agraphia. Visuospatial and visuoconstructional functions were normal. Digit span was seven forward and five backward. He could recall the names of three of four unrelated objects after 5 min.

Cranial nerve functions were intact. The right-side paralysis had resolved completely, and motor strength was normal in all extremities. Sensation was also normal. Deep tendon reflexes were symmetrical, and the plantar responses were flexor bilaterally.

Computerized tomography at the time of admission showed a discrete lobar hemorrhage in the posterior-inferior portion of the left temporal lobe in the region of the temporo-occipital junction. The lesion involved the posterior portion of the middle and inferior temporal gyri (Brodmann's cortical areas 21 and 37) and the underlying white matter. Wernicke's area, the angular gyrus, and the supramarginal gyrus were spared (Fig. 1).

## NEUROPSYCHOLOGICAL STUDIES

*Oral Language Functions*

Oral language functions were assessed with the Western Aphasia Battery (Kertesz, 1982). The patient's scores on this test were in the nonaphasic range (AQ 98.4). He experienced difficulties only on the object-naming section of the test; he was unable to name 6 of 20 visually presented objects. However, he provided accurate functional descriptions of the objects he could not name and was able to demonstrate their use, which indicated that recognition of the objects was intact. Allowing him to palpate the objects he could not name on visual presentation did not improve his performance, thus excluding the possibility that his failure

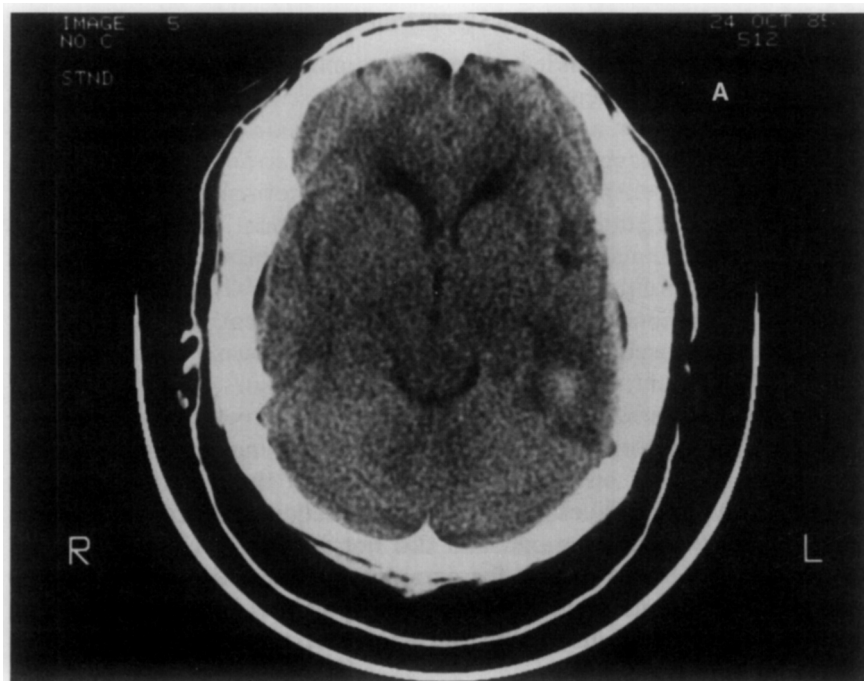


FIG. 1. CT scan without contrast showing a small hemorrhage in the posterior-inferior portion of the left temporal lobe. Wernicke's area, the angular gyrus, and the supramarginal gyrus are spared.

to name was due to optic aphasia. Phonemic cues were helpful in naming 3 of the 6 objects. Written naming was impaired to a similar degree. We compared naming of visually presented objects with naming objects presented in the tactile modality alone (both hands tested), and with ability to name environmental sounds. We attempted to keep the level of difficulty equal across the different modalities by using common objects with which the patient probably had frequent contact. He was unable to name 5 of 15 visually presented objects and 4 of 15 objects presented in the tactile modality. His performance on the tactile naming task was not influenced by the hand used. He named all 15 environmental sounds correctly and also named items from functional verbal descriptions provided by the examiner. Although some high-frequency words (e.g., pen, fork) were not available to the patient when the corresponding objects were presented in the visual or tactile modalities, he could produce lower frequency words (e.g., typewriter) when naming objects from their characteristic sound. Naming of colors and body parts was intact, and he pointed accurately to objects named by the examiner.



FIG. 1—Continued

### Reading

We first evaluated the patient's reading functions 2 weeks after the hemorrhage. He was college educated and did not spontaneously complain of any reading difficulties.

He performed flawlessly on the reading section of the Western Aphasia Battery that assesses reading comprehension of sentences and ability to follow written commands. Reading skills were further evaluated with the Battery of Adult Reading Functions (Rothi, Coslett, & Heilman, 1984). This battery contains subsections for evaluating reading of nonwords and regular and irregular words, as well as ability to read nonwords that are homophonic with real words (pseudohomophones). A separate section allows a comparison of the reading of function words versus content words that are balanced for frequency and graphemic length.

*Results.* Of the regular words 28 of 30 were read correctly (93%). The two reading errors were visual (e.g., *holder* for *halter*).

Of the irregular words 27 of 30 were read correctly (90%). The reading errors were again visual (e.g., *health* for *height*). Both regular and irregular words were read rapidly and confidently.

From a list of 30 pronounceable nonwords the patient read only 4

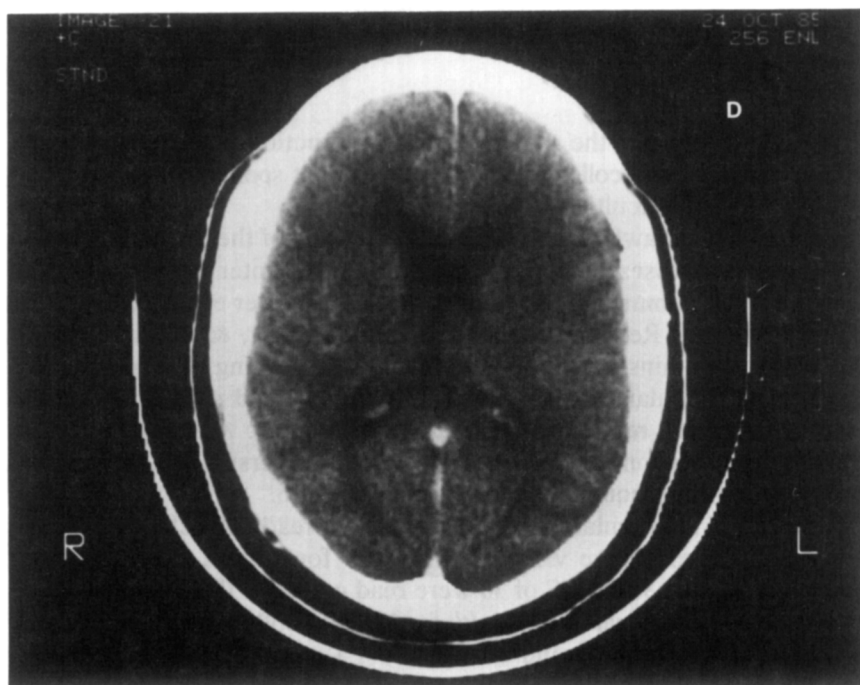


FIG. 1—Continued

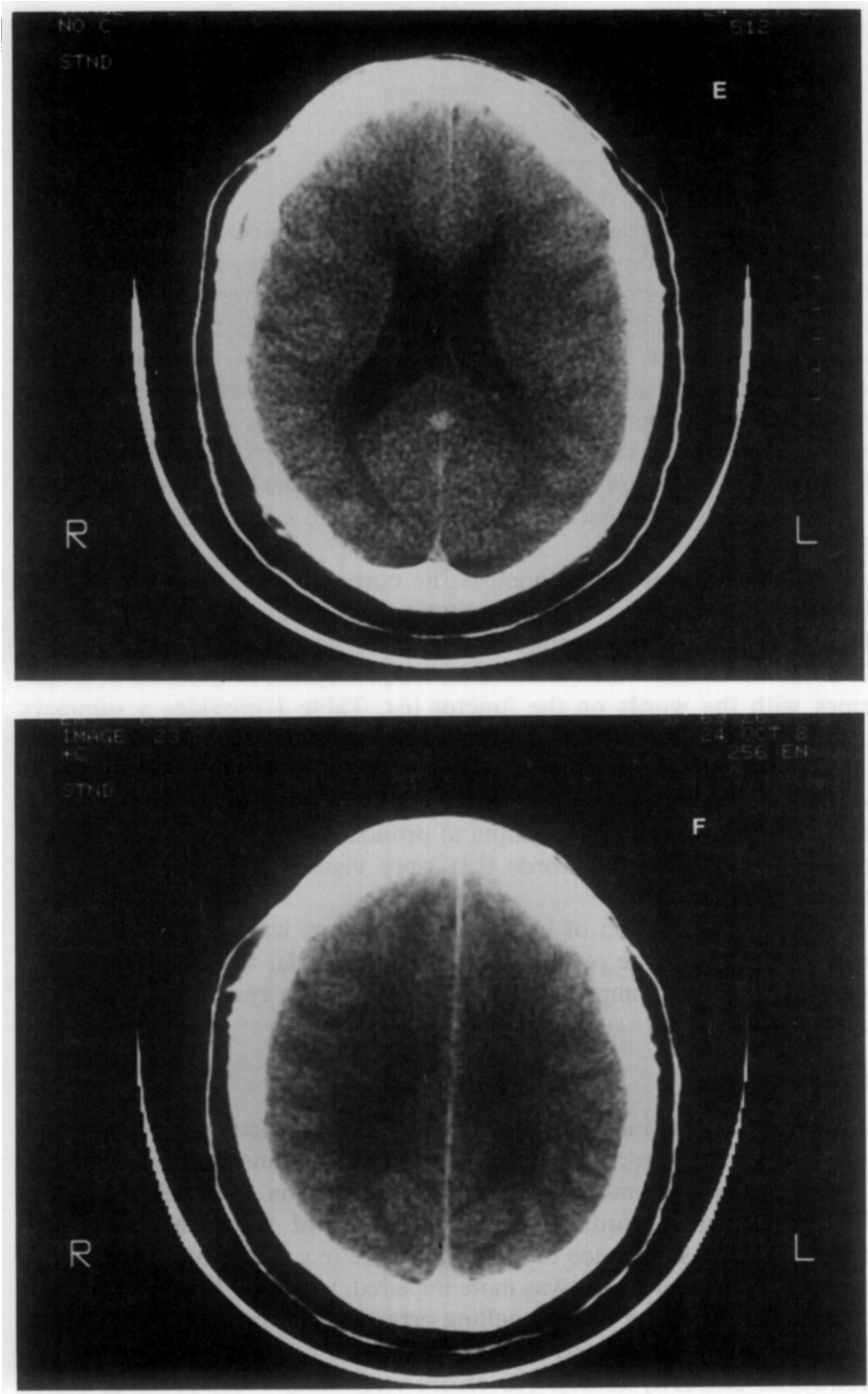


FIG. 1—Continued

TABLE 1  
SUMMARY OF READING FUNCTIONS

	Regular	Irregular	Nonwords	Pseudohomophones	Funcors
% Correct	93	90	13	78	90

correctly (13%). the overwhelming majority of his reading errors (85%) were real words that were visually similar to the target nonwords (e.g., *trade* for *trad*, *slim* for *slem*). The remaining errors were phonologically incorrect but visually similar nonwords. The patient's responses to nonwords were slower, and he lacked confidence in them, admitting that he was "only guessing." He frequently expressed surprise over his poor performance on this task. Although severely impaired in his ability to read nonwords, he could name the individual letters in the letter string he attempted to pronounce and could repeat nonwords he could not read.

From a list of 18 pseudohomophones (e.g., *coam*, *ile*), he read 14 correctly (78%).

In reading grammatical funcors, he correctly read 18 of 20 funcors (90%). The two errors were substitutions of another funcor (e.g., *thus* for *though*). At the same time he was 100% accurate in reading a list of 20 contentives that were balanced for frequency and for number of graphemes with the words on the funcor list. Table 1 provides a summary of our patient's initial reading performance.

When the patient was asked to pronounce words that the examiner spelled aloud, his performance was essentially flawless both with regular and irregular words. His attempts to pronounce nonwords spelled aloud to him resulted in real words that were visually similar to the target nonwords.

A second evaluation of his reading functions was conducted 1 week later. By this time he read all regular and irregular words correctly and showed significant improvement in his ability to read nonwords (90% correct).

### Writing

The first evaluation of the patient's writing abilities was performed 2 weeks after the hemorrhage. He always prided himself in his ability to spell and was very much disturbed by his agraphia. He could spell only 17 of 30 regular words correctly (57%). Most errors had a visual-phonological resemblance to the target word (e.g., *submicht* for *submit*). Spelling of irregular words was more impaired, with only 12 of 30 responses correct (40%). Many of his spelling errors were phonologically correct (e.g., *ritchewal* for *ritual*, *sy* for *sigh*). He could spell only 9 of 30 nonwords correctly (30%). All errors were nonwords, some of which



had a visual-phonological resemblance to the target word (e.g., *anthon* for *andon*). A second evaluation of his writing a week later showed significant improvement in ability to write nonwords (80% correct). He spelled 80% of regular words correctly; his spelling of irregular words was 71% correct. All his errors were phonologically correct (e.g., *denigh* for *deny*).

Oral spelling showed similar impairments to written spelling for all types of words.

## DISCUSSION

The patient's spontaneous speech and verbal comprehension were normal; in fact, his only oral language deficit was an anomia for objects presented in the visual and tactile modalities.

Lesions in the left posterior-inferior temporal region, involving the posterior portion of the middle temporal gyrus or Brodmann's area 37, had been previously reported to cause a distinct type of anomic aphasia (Benson, 1979; Brown, 1972). However, patients with this type of "word-selection" anomia have abnormal spontaneous speech characterized by word-finding difficulties and circumlocutions, and the anomia does not appear to be modality specific (Benson, 1979).

Multimodal (visual and tactile) agnosia without aphasia had been reported to follow an infarction in the ventromedial occipitotemporal region of the left hemisphere (Feinberg, Rothi, & Heilman, 1986). That our patient's failure to name objects could not be attributed to agnosia is evidenced by his ability to provide adequate functional descriptions of the objects he could not name and by his appropriate demonstration of their use.

Lesions in the left occipitotemporoparietal region may induce the syndromes of optic aphasia and tactile aphasia (Lhermitte & Beauvois, 1973; Beauvois, Saillant, Meininger, & Lhermitte, 1978). Bilateral tactile aphasia can also be associated with phonological alexia (Beauvois et al., 1978). Patients with optic aphasia and tactile aphasia may have intact oral language functions, and they can demonstrate the use of the same objects they cannot name when presented in the visual or tactile modality. The patients predominantly produce semantic paraphasic errors on naming tasks and also tend to make paraphasic errors when requested to provide a functional verbal description of the objects they cannot name (Beauvois, 1982). Our patient, however, never produced an incorrect response; he simply failed to name. In addition, his functional descriptions of the objects he could not name were free of dysphasic errors. Therefore, his naming disturbance does not simply represent a combination of optic and tactile aphasia and could probably be best described as an optic and tactile anomia. This disorder seems to be due to a failure to retrieve the specific names for objects—presented in the visual and tactile modalities—from an intact internal store of word representations. This failure occurs

despite the fact that the adequate visual and tactile percepts activate the appropriate conceptual representations. It may be that the word representations can be accessed through modality-specific systems as well as through a verbal semantic system that mediates spontaneous speech. Our patient could retrieve specific names for objects through the verbal semantic system, as evidenced by his normal spontaneous speech and his ability to name from functional verbal descriptions. Since he was able to name environmental sounds, the word representations could also be accessed through a nonverbal auditory system. However, the lesion may have disconnected the visual and tactile systems from the internal store of word representations and caused optic and tactile anomia.

Neuronal systems that subserve tactile and visual recognition may overlap. The patient with bilateral tactile aphasia described by Beauvois et al. (1978) was much better at naming tactually presented objects that were likely to have precise visual representations. Feinberg et al. (1986) postulated that their patient with visual and tactile agnosia also may have relied on visualizable aspects of tactually explored objects to facilitate recognition. They further hypothesized that visual and tactile percepts are converted to visuospatial percepts before their recognition and that the ventromedial occipitotemporal region was critical for this task.

Patients may indicate recognition of an object by verbal or nonverbal identification (describing or demonstrating its use) or by naming. Recognition, identification, and naming may be dissociable. Although probably mediated by different systems, recognition of faces may illustrate this point. One may look at a face and recognize it as familiar, yet be unable to retrieve either the name or any other information about the person. Alternatively, one may recognize the face and recall other pertinent facts about the person without being able to recall the name. Finally, one may be able to recognize, identify, and name the person.

Perhaps the intact connections from the ventromedial occipitotemporal region to the parietal lobes and/or other supramodal association cortices allowed our patient to manifest recognition of visual and tactile percepts by verbally and nonverbally identifying them. However, the lesion in the posterior-inferior portion of the left temporal lobe prevented access to the area of word representations and resulted in optic and tactile anomia. The area that actually contains these word representations was spared, however, as evidenced by the patient's preserved ability to name environmental sounds and by his otherwise intact oral language functions.

Our patient clearly shows the dramatic dissociation of nonword reading from the reading of real words that characterizes the syndrome of phonological alexia. According to psycholinguistic theories of reading, this dissociation reflects the selective impairment of the nonlexical phonological reading route. The lexical route, however, was still available to our patient and allowed him to read most regular and irregular words suc-

cessfully. In reading aloud nonwords the patient showed a tendency to "lexicalize," by responding with real words that were visually similar to the target. This occurred despite the fact that he was explicitly told that the target words were not real words and that his task was only to try to pronounce them as best he could. When normal subjects read nonwords by the nonlexical phonological route, the lexical route is "mute." It appears that in our patient impairment of the nonlexical phonological route was accompanied by "disinhibition" of the lexical route that led to an obligatory processing of unfamiliar letter strings by a lexical strategy. Like some previously described patients with this syndrome, he was better at reading nonwords that were homophonic with real words. As pointed out by Patterson (1982), this may be because some of these nonwords are also visually similar to their real word homophones. Although we did not test this hypothesis, Dérouesné and Beauvois (1979) could not find a difference in their patients' reading of nonwords as a function of visual similarity to real word homophones.

That a severe impairment of the nonlexical phonological route did not significantly compromise our patient's overall reading performance raises questions about the role of assembled phonology in reading. In his review of this topic Coltheart (1980) concluded that there was indeed very little evidence from experimental work on reading to suggest a significant role for assembled phonology in normal readers. Our observations of this patient support these findings. Skilled readers may only rely on assembled phonology to read unfamiliar words (or nonwords). However, ability to assemble phonology is probably essential for the acquisition of reading skills (Patterson, 1982; Doctor & Coltheart, 1980).

Another constant feature of the syndrome of phonological alexia is the presence of a writing disturbance. All 16 patients with phonological alexia reviewed by Sartori et al. (1984) had agraphia, and our patient is no exception. There are significant quantitative and qualitative differences between the patients' writing performances, and no consistent pattern of writing disturbance has emerged from the studies. Impairment of phonological reading can occur with or without impaired phonological spelling, suggesting that the neuropsychological mechanisms of phonological reading and phonological writing are potentially dissociable.

The neuropsychological syndrome of phonological alexia in our patient was induced by a small lobar hemorrhage in the posterior-inferior portion of the left temporal lobe. This anatomical region is not usually implicated in neuroanatomical studies on acquired alexia. Lesions that are more posteriorly and medially placed, involving the ventromedial occipital association cortex and the white matter of the left occipital lobe in the paraventricular region, are often associated with the syndrome of alexia without agraphia (Benson & Geschwind, 1969; Damasio & Damasio, 1983; Dejerine, 1892; Greenblatt, 1983). According to the disconnection

theory, such lesions cause alexia by compromising both inter- and intrahemispheric visual input to the angular gyrus, the center for visual word images (Dejerine, 1891, 1892; Geschwind, 1965). More dorsally placed lesions that involve the dominant angular gyrus have been associated with the syndrome of alexia with agraphia (Benson & Geschwind, 1969; Dejerine, 1891).

That visual information from ventral visual association cortex could reach the angular gyrus and subsequently gain access to Wernicke's area was evidenced by our patient's ability to read irregular words. Without access to the orthographic code contained in the dominant angular gyrus, such words could not be read correctly. We propose that reading by the lexical route could be accomplished by an intact dorsal pathway from the inferior visual association cortex to Wernicke's area via the angular gyrus. This pathway, however, could not mediate the reading of nonwords.

Our patient's lesion at the left inferior temporo-occipital junction is ideally situated to disrupt the transfer of visual information from ventral visual association cortex to Wernicke's area by damaging the middle and inferior temporal gyri and/or the underlying white matter. We postulate that a ventral pathway from inferior occipital association cortex to Wernicke's area via the posterior-inferior portion of the left temporal lobe may be involved in mediating reading by the nonlexical phonological route. According to Dejerine, the inferior occipital association cortex is connected with the temporal lobe by the long association fibers of the inferior longitudinal fasciculus (Dejerine, 1895). Recently Tusa and Ungerleider (1985) suggested that these areas are connected by a series of U fibers, for which they proposed the term "occipitotemporal projection system." As shown by our patient, damage to this pathway can induce the syndrome of phonological alexia in a relatively "pure" form. The recovery of the patient's reading and writing performance was paralleled by an improvement of his ability to name objects presented in the visual and tactile modalities. The rapidity of this recovery can be attributed to the hemorrhagic nature of his lesion and is probably related to the resolution of the edema and retraction of the clot.

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