

Right hemianopia with memory and color deficits in circumscribed left posterior cerebral artery territory infarction

J. P. Mohr, M.D., J. Leicester, M.D., L. T. Stoddard, Ph.D.,
and M. Sidman, Ph.D.

INFARCTION IN THE TERRITORY of the left posterior cerebral artery with accompanying right homonymous hemianopia, color deficits, and memory disorder has been described previously. The present case is of new interest in view of the special features of the individual deficits, the discrete foci of pathologic lesions, and the bearing these findings have on current views of brain function.

METHODS

The patient was evaluated repeatedly at the bedside and in a behavior laboratory. In the laboratory, the patient was tested in a small, quiet, softly lit room. He was seated before a display panel embedded in the wall at eye level. The panel had 9 translucent windows, each 2 in. square, arranged in a 3 by 3 matrix. In matching-to-sample tests, each trial began by presenting the test stimulus. Visual test stimuli were projected from the rear onto the center window of the matrix. Auditory test stimuli were dictated from a prerecorded tape through an endless loop tape player and presented repetitively through a loudspeaker mounted in the ceiling of the room.¹ Somesthetic test stimuli were presented for palpation, unseen, to either hand on a shielded shelf mounted below the visual matrix. The patient responded to the test stimulus by pressing the center window of the matrix; when he did so, visual choice stimuli were projected onto the 8 outer windows of the matrix. One choice,

the correct one, corresponded to the test stimulus; the others did not. The patient indicated his choice selection by pressing the appropriate choice stimulus window. Correct selections were rewarded by the ringing of chimes and the delivery of a nickel through a chute next to the matrix. After incorrect selections, neither chimes nor nickel occurred. After each trial, regardless of correct or incorrect selection, the next trial followed immediately.

In some tests, the test stimulus remained after the choices appeared (simultaneous matching); in others, the test stimulus was removed when the subject pressed the center window, and the choices were presented zero to forty seconds later (delayed matching).

The test stimuli were presented in the same way for oral naming. Chimes and nickel reinforcement followed correct oral naming re-

From the Behavior Laboratory, Neurology Service, Massachusetts General Hospital, Boston, and the Division of Neuropsychiatry, Walter Reed Army Institute of Research, Washington, D.C.

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Dr. Mohr's address is Division of Neuropsychiatry, Department of the Army, Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, D.C. 20012.

Reprint requests to Dr. M. Sidman, Behavior Laboratory, Neurology Service, Massachusetts General Hospital, Boston, Massachusetts 02114.

sponses. The apparatus and procedures have been described elsewhere.²

CLINICAL REPORT

A 72-year-old right-handed accountant was admitted to the Massachusetts General Hospital on Dec. 26, 1967, after being discovered in his partly damaged car.

The patient had had arteriosclerotic heart disease. He suffered myocardial infarctions in 1959 and 1963 and had been maintained on digitalis therapy since 1963. He had had no previous neurologic symptoms.

On the evening of admission, the patient attended a fraternal meeting. He was well known for his memory of proper names and was a high-ranking member of the society. His car was noted by police at 11:00 P.M. The patient was alone in it, sitting behind the wheel and staring through the windshield. On direct questioning, he could give no account of the accident and had to consult his wallet to give his address. He was brought to the emergency ward of the Massachusetts General Hospital.

On examination, the blood pressure was 150/80 and the pulse, regular at 80 beats per minute. He was fully alert and conversed freely but remained unable to give details of the previous few hours. At sight, he named several common objects of furniture, body parts, and items of clothing. He easily repeated unfamiliar foreign words from dictation with no hint of paraphasia or dysarthria. He defined a few words and solved simple word problems from dictation. On direct confrontation, a dense right upper and partial right lower quadrantanopia was detected. The pupils were equal and reacted to light. Eye movements were full. There was no facial weakness. The limbs had normal power. The tendon reflexes were normal and the plantar responses flexor. Sensory testing of the limbs gave normal results. Cerebellar tests were done normally. There was a small laceration at the base of the nose. The heart was enlarged to the sixth intercostal space. No murmurs were heard. Pulses were full throughout.

Laboratory findings were normal for hematocrit, white blood count, serum sodium and potassium, fasting and two-hour postprandial blood sugar, blood urea nitrogen, prothrombin time, stool guaiac, lumbar puncture, skull X-ray, and electroencephalogram. Chest X-ray showed biventricular hypertrophy. The electrocardiogram showed a sinus rhythm at 80 to 90, normal P-R and QRS intervals, an axis of +30, and frequent ventricular ectopic beats but was little changed from the record of 1963. His central retinal artery pressure was 55 bilaterally. Visual examination by ophthalmology consultation showed visual acuity of 20/80 on the right and 20/30 on the left. Formal visual fields with a 9-mm. flashing light showed a dense right upper quadrantanopia. Opticokinetic nystagmus was equal to each side.

From the admission evaluation until discharge seventeen days later, the patient's condition remained relatively unchanged. He was followed at intervals as an outpatient. During this time, his wife observed no significant improvement in his memory. On day 82, March 17, 1968, he died suddenly while riding in an auto as a passenger. Autopsy was performed the next day in a funeral home. Permission was limited to the removal of the brain.

Visual field defect. The patient demonstrated a dense upper and partial lower right homonymous hemianopia on direct confrontation and by Bjerrum screen testing. The visual fields showed no essential change after his discharge.

Color deficit. A persisting deficit occurred only on those tasks requiring the patient to respond to a single-color test stimulus for which no simultaneous comparison with other colors was available to him.

1] *Color test stimuli, naming.* The patient's scores in naming singly presented colors never exceeded 67%. On day 2, he named colors correctly in 5 of 10 trials. On day 9, he named 11 of 18. On day 70, he named 12 of 18 (Fig. 1A).

Although the patient failed to name singly presented colors correctly, he did not fail other naming tests with these same color names. On day 2, asked to recite color names from memory, his 12 uttered names included the 6 used in the tests. Further, in response to the dictated names of 5 common items, he easily named their usual color. He was, therefore, capable of saying the color names and of comprehending the "concept" of color.

2] *Color test stimuli, matching.* Persisting deficits occurred in both simultaneous and delayed matching of singly presented colors to printed color-name choices (Fig. 1B). Simultaneous matching of colors to color choices was satisfactory, but a persisting deficit was observed in the delayed matching (Fig. 1C). Although the patient was inaccurate in matching color test stimuli to printed color-name choices (Fig. 1B), he performed perfectly in both simultaneous and delayed matching of dictated color names to the same printed choices (Fig. 1D); his deficit, therefore, did not reflect an inability to read color names. Nor could his difficulty be interpreted simply as a general color-input deficit or as a color agnosia; Figure 1E shows, after some initial impairment, satisfactory performances in tests that had colors as the choice stimuli. Although the patient was inaccurate in matching color test stimuli to printed color-name choices (Fig. 1B), he was able to match color-name test stimuli, dictated and even printed, to color choices (Fig. 1E). The improvement in the delayed-matching performances in Figure 1E shows also that the persisting deficit in delayed matching of colors to color choices (Fig. 1C) was not a consequence of the delay per se. It did not reflect a memory disorder per se.

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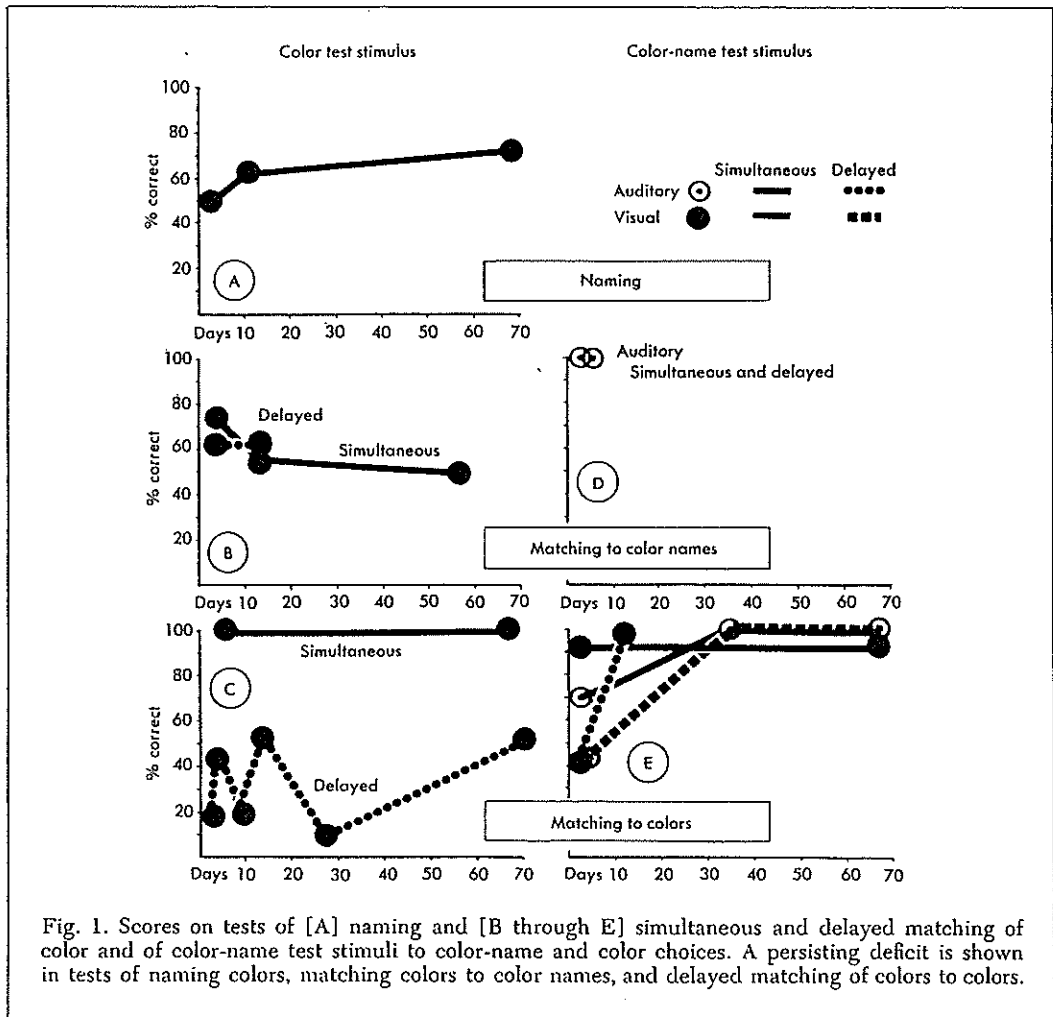


Fig. 1. Scores on tests of [A] naming and [B through E] simultaneous and delayed matching of color and of color-name test stimuli to color-name and color choices. A persisting deficit is shown in tests of naming colors, matching colors to color names, and delayed matching of colors to colors.

to printed color-name choices were not random but were related to the correct responses: the patient named or chose a similar color name, e.g., red for orange. In delayed matching of colors to color choices, the relation between errors and correct choices was somewhat obscured by a prominent bias for the selection of the color green, and less so for white, regardless of the correct color. Error choices were not related to the length of the delay.

3) *Other tests with color stimuli.* On day 2, the patient performed well in the Ishihara test for color blindness. On day 5, pairs of similar color cards were shown to the patient, and he was consistently able to point correctly to the lighter color of a pair.

4) *Other matching-to-sample tests.* Matching-to-sample with materials other than color showed no deficits. The patient easily performed simultaneous and delayed matching of printed words with their

pictures, dictated (spelled or pronounced) words with their pictures, and printed or dictated trigrams with printed trigrams. These satisfactory performances suggested that the deficits were confined to colors and color names, did not reflect a more general disorder, and were not explainable as part of a memory disorder.

Memory deficit. On admission and thereafter, the patient gave evidence of a persisting and severe impairment of recent memory. He remained unable to give any account of the events immediately prior to admission, failed to recall many of the events during hospitalization, and persisted in failure to recall the names of his physicians, despite repeated efforts to teach him their names. On ordinary verbal tests of immediate memory, he failed repeatedly. Given the dictated story, "Tom and Bill went fishing; they caught 3 black bass," on day 1, he repeated it aloud immediately. Yet after ten minutes he failed to recite a single item

in the story and after fifteen minutes denied being told a story to recall. On days 2, 10, and 15 he repeatedly failed to recite the story after brief delays. In the months following discharge, on return visits to the laboratory, he smiled and greeted those in the laboratory but remained unable to recall their names. He often walked in the wrong direction from that leading to the experimental chamber.

He also failed some tests requiring the recall of names learned long before his illness. Most severe was the deficit with proper names of people. On admission, he treated as unfamiliar the names of his nephew and brother-in-law. Although highly regarded in his fraternal organization for his recall of names, he remained unable to recall a single name on demand. He was puzzled by his own failure and repeatedly testified as to his previous prowess. By day 2, he was able to give the names of his wife, son, business partner, and brother. He failed to recall the names of a number of familiar political figures either when shown their photographs or when their job titles were dictated to him. Although unable to name these men, he did describe their attributes. For example, he characterized the (then) President (Johnson): "Well, he's tall, not bad looking, cocky, not forceful, thinks he knows it all . . . family man . . . wife is his boss . . . two daughters and now two sons-in-law." Later, presented with a picture of the President, he said, "He's the President. I can't remember his name."

His verbal recall was relatively better for names of local towns, states, proper first names of men and women, automobiles, items of clothing, rooms in a house, forms of money, vegetables, holidays, body parts, the numbers 1 through 20, the alphabet, the days of the week, and the months of the year. At sight, he named these same letters, numbers, body parts, articles of clothing, 10 of 12 pictured items in the Stanford-Binet picture test, 9 facial expressions, and 14 manipulable objects. The 14 manipulable objects named at sight were also easily named by touch when placed individually in his right hand unseen.

Discrimination tests. In the laboratory, on day 1, attempts were made to teach the patient a simple discrimination preparatory to special tests of memory. On this discrimination task, he was required to select a circle from a display of 1 circle and 7 identical ellipses (Fig. 2A). Instruction was nonverbal; nickel reinforcement was used for each trial in which he selected the circle. He failed to learn this task, even by means of a special teaching program⁹ in which the wrong choices were slowly faded into view over a series of trials.

On day 9, he succeeded in learning the task of discriminating the circle from the ellipses by means of the teaching program. He was then given a 32-trial test in which he had to select the circle from 7 ellipses of varying vertical heights' (Fig. 2B); he selected the circle in 24 of 32 trials (Fig. 2, day 9). He never selected the 3 smallest ellipses.

This satisfactory gradient demonstrated that he had learned to select the circle and reject the ellipses.

On day 13, the patient was given the gradient test again as a retention test without benefit of reexposure to the teaching program. He showed no evidence that he had done the test before; he pressed the same choice window on every trial, disregarding the circle and the ellipses. As a result, his performance showed no preference for selecting the circle (Fig. 2, day 13). His remarks during this retention test also indicated no verbal

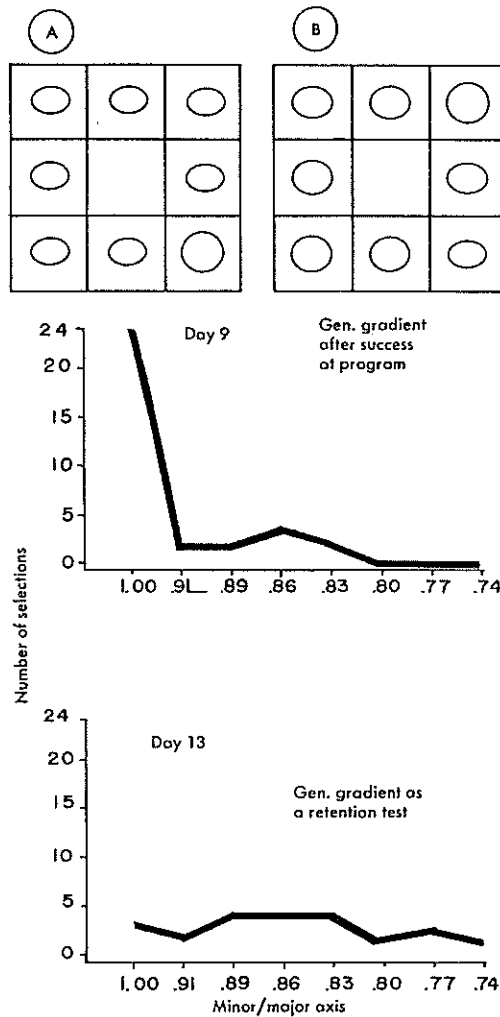


Fig. 2. Impaired performance in a generalization test on day 13 after successful acquisition of the discrimination on day 9. The task of the patient was to select the circle but not the ellipses. [A] An example of the easy version of the task used in training the discrimination and [B] an example of the generalization test.

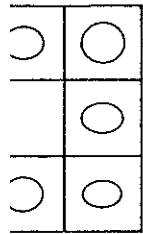


Fig. 3. Medial geniculate nucleus, sectioned at level of 200.

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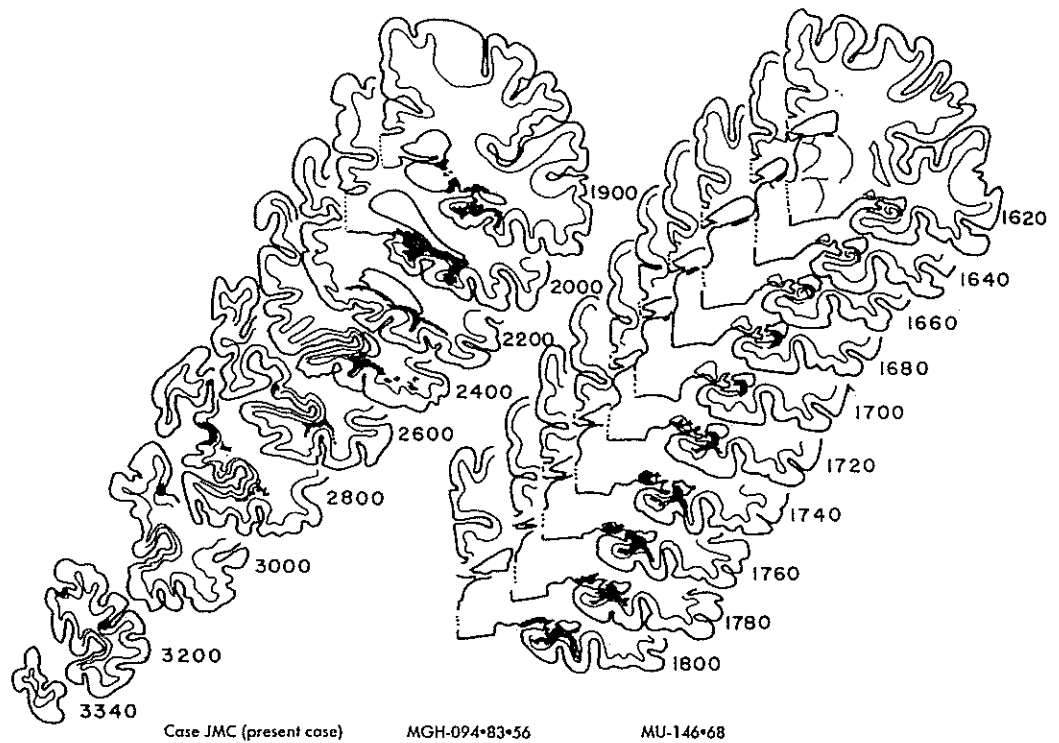


Fig. 3. Montage view of the left cerebrum in coronal serial section showing infarction of the lateral geniculate body and hippocampal formation, slight infarction of the thalamus, and undermined lingual gyrus. [Right] Plates are shown at intervals of 20 sections and [left] at intervals of 200.

recall of the generalization test nor even of how to proceed in the test: "What would you like to have me do? Tell you what the letters are? It's an 'o.' Am I supposed to do anything now? Press this one (the center window containing no form)? There are all o's here."

Neuropathologic examination. Gross findings. There was severe atherosclerosis of the cerebral arteries. The two posterior cerebral arteries were patent throughout their courses.

The external surface of the brain was entirely normal save for the depths of the left parieto-occipital sulcus found by detailed dissection to be within the territory of the posterior cerebral artery, where a tiny old brownish-colored area of collapse of the cortex was noted.

Microscopic findings. After formalin fixation, the brain was embedded in toto in colloidin and serially sectioned at 35 μ . Pairs of sections at 20-section intervals were stained with cresyl violet and by the Loyez method for myelin. Sections taken at appropriate intervals were traced on paper to provide a sequential three-dimensional montage view of the extent of the lesions, as illustrated in Figure 3. The preparation of the histologic material was accomplished by the Laboratory of Normal and Pathological Anatomy, then at the Warren Mu-

seum of the Harvard Medical School, through the courtesy of Dr. Paul Yakovlev. The sections were reviewed with Dr. Yakovlev for accuracy in transcription of the lesions to the montage tracings.

The left lateral geniculate body showed infarction in the center of its crown beginning in section 1,720, enlarging posteriorly to include the entire posterior aspect of the crown and lateral corner of the hat-shaped nucleus (Figs. 3 and 4). The medial corner and the very anterior portion of the crown were partially spared.

A discrete infarct involved the posterior two-thirds of the left hippocampus, beginning 5 mm. posterior to its anterior end, enlarging from its beginnings in Sommer's sector to include cavitation of the entire hippocampus, and including the white matter of the parahippocampal and fusiform gyri (Fig. 3). From sections 1,620 to 1,800, infarction spread into the strata radiatum, lacunosum, and moleculare of the hippocampus (section 1,680) and into the alba of the fusiform gyrus (section 1,720). More posteriorly, the fimbria showed infarction. The alveus and left fornix showed slight gliosis. The precommissural bed nuclei of left fornix and the left mammillary body were slightly shrunken. The right hippocampus showed no abnormality save for slight perivascular



Fig. 4. Close-up ($\times 5$) myelin stain of section 1,740

calcification, which was also found in left hippocampus and bilaterally in the globus pallidus and dentate nuclei of the cerebellum. The right fornix, mammillary body, and anterior commissure appeared normal. A tiny infarct, approximately 1 mm. in diameter, was found in the right thalamus (section 1,660) in the medial aspect of lateral dorsal nucleus, impinging slightly on the adjacent intralaminar nucleus but sparing the centrum medianum and dorsomedial nuclei. The left thalamus showed a band of infarction along the juxtaventricular border of lateral dorsal nucleus (sections 1,620 through 1,700) and along the posterior wall of the pulvinar (section 1,900).

The left lingual gyrus beginning just posterior to the isthmus of the gyrus fornicatus (section 2,200) was variably undermined in its entire extent. Only scattered discontinuous foci of cortical infarction were observed and these, only in the more posterior calcarine cortex and parieto-occipital fissure. The visual radiations were entirely spared save for the small edge of the radiation passing below the temporal and occipital horn of the left lateral ventricle. The corpus callosum, tapetum, and inferior longitudinal fasciculus appeared entirely spared.

The infarctions were all of the same approximate age, consistent with the eighty-two-day period from onset of the deficit to death.

DISCUSSION

Right homonymous hemianopia. We attribute the complete upper quadrant and partial lower quadrant right homonymous hemianopia to infarction of the lateral corner and central crown of the hat-shaped left lateral geniculate body.^{5,6} The occipital lesions appear insufficient to account for the extent of the visual field deficit.

Color deficit. Our patient's initial test scores might have been taken to indicate a bidirectional deficit in relating colors to their names—a "disconnection" syndrome. Errors on these tests were not random, however, suggesting an impairment, but not a complete loss, of controlling relations between stimulus and response. If this is so, the term "dysrelation" would seem a more appropriate characteriza-

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tion of the deficit. The nonrandom character of the errors might also suggest that the patient's responses were controlled by aspects of the stimuli other than their hue, for example, brightness or saturation. If this is so, "disconnection for hue" might be an adequate descriptive term. Because the different attributes of the stimuli were not systematically controlled, these questions regarding the patient's initial deficits cannot be resolved.

Within several sessions, an even more specific deficit evolved. Initially the deficit was bidirectional, involving the relating of colors to color names and color names to colors. Later it became more refined, expressing itself as a unidirectional deficit in relating color test stimuli to printed color-name choices; the converse performance of relating printed color-name test stimuli to color choices had cleared. In all the tests showing a persisting deficit, the patient was required to respond to a single test color with no other colors available for comparison. The recovery of the deficits in matching dictated color names to colors and the unidirectionality of the visual color to color-name matching deficit rule out an explanation of the continuing deficits in terms even of a "partial" disconnection of color names and colors. Instead, the data suggest that color as an input was disturbed, manifesting itself as deficient performance on all behavioral tasks requiring the patient to respond to a single color when no comparison colors were available. This suggestion encompasses the feature common to the three continuing deficits—color naming, matching of colors to color names, and delayed matching of colors to colors—but not shared by the intact performances. Such an input deficit, however, is unusual and unexpected, raising the question of how the availability of comparison colors could help the patient overcome it. His early demise prevented further exploration of this problem.

The role played by each or all of the separate pathologic foci in the production of the color deficit cannot be concluded with certainty. The lateral geniculate body is considered to play a role in color discrimination,^{7,8} but deficits in color discrimination (normal in our case) would be expected to occur only when the lesion is bilateral. Recent renewed interest in the thalamic role in verbal behavior⁹

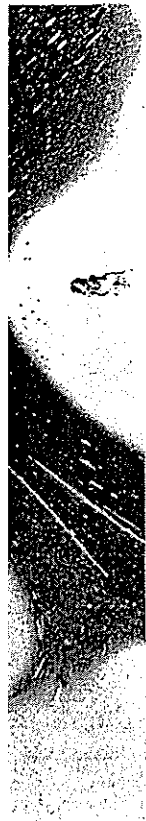
suggests that the pulvinar involvement might have been of importance in our patient's color deficit. No precedent was found to bear on the present case. The issue is further complicated by the undermined lingual gyrus. In the older literature,¹⁰ lingual gyrus involvement was considered the basis for "color blindness." Until other cases with neuropathologic involvement restricted to each of these areas are found and similarly tested, the relative significance of the individual lesions, and with it the clinicopathologic correlation, will remain unsettled.

In seeking corroborative case reports, our patient's deficit and neuropathologic findings appear to have no certain precedent. Nor is his deficit adequately encompassed by the traditional forms of color syndromes reviewed below.

Color blindness from cerebral disease shows impaired performance on tests of color discrimination (Ishihara plates, etc.) and may be unilateral on either side¹⁰⁻¹² or bilateral.¹³ In typical cases, colors are described as gray, pale, or washed-out in the involved field(s). Our case performed well on the Ishihara test for color blindness and gave other color names in response to presented colors.

Color agnosia refers to impairment in relating a color to its name in the absence of deficits in color discrimination. This syndrome is traditionally revealed by normal performance on tests such as the Ishihara, with errors in naming colors at sight and in matching dictated color names to color choices. Our case appeared to satisfy these criteria in the initial stages, but the subsequent evolution of his deficit ruled out classical color agnosia.

Unfortunately, the available details for most of the reported cases do not permit precise comparison with ours.¹⁴⁻²¹ Only a few have shown adequate color discrimination, impaired color naming, and impaired matching of colors with color names.²²⁻²⁴ Clinically, these cases also showed a right homonymous hemianopia and dyslexia. (Our case showed no dyslexia.) For the 2 autopsied cases, those of Lissauer²² and of Geschwind and Fusillo,²⁴ the lesion lay in the distribution of the left posterior cerebral artery, destroying most of the gray and deep white matter of the medial occipital lobe including the visual radiation, inferior longitudinal fasciculus, and the crossing fibers through



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the splenium of the corpus callosum in the tapetum. It is to this type of case that Geschwind and Fusillo have applied the term "disconnection syndrome" to stress the point that the lesion may have separated the adequately discriminated visual input in the right hemisphere from access to the language region of the left hemisphere. As a result, visual stimuli would presumably be unable to be associated with their names, causing alexia (letters and words), defective naming of colors, and defective matching of color names to colors. In support of the disconnection notion, Geschwind and Fusillo stressed that their patient ". . . would answer at random"²⁴ when shown an object and asked whether it was a certain color.

Other cases,²⁷⁻²⁹ however, including one considered a classical example of alexia without agraphia,^{10,25,26} have not shown this defect with color even while satisfying the criteria of the disconnection syndrome—right homonymous hemianopia and alexia without agraphia.

Our case provides further evidence that alexia and color deficits need not be related. For our case, the right hemianopia and lack of alexia appear readily explained within the disconnection hypothesis: the hemianopia was a likely result of the geniculate infarct, and the transcallosal pathways relating the right to the left cerebral hemisphere remained intact to preserve normal reading. The color deficit, by the same hypothesis, appears difficult to account for. Why did not the same pathways sparing reading also spare naming and matching for colors? Our specification of the patient's impaired color performances as a color-input deficit helps resolve this problem. Perhaps the opinion of Pötzl³⁰ is appropriate: the frequent coexistence of color deficit with alexia does not reflect a common mechanism for their occurrence; rather, spatially proximate regions of the cerebrum serving different functions are susceptible to simultaneous involvement by the same lesion. It is unfortunate that few of the previously reported cases provide details sufficient to bear upon this issue.

Amnesic color anomia refers to a color deficit in which color discrimination and cross-matching of color with color names is adequate. The deficit is confined to naming col-

ors.³¹ In our case, both naming and matching were disturbed.

Other color deficits of other types appear in cerebral disease,³² most requiring the examiner to depend on the patient's subjective description of the altered appearance of color and of its relationship to the environment. Our patient volunteered no evidence to suggest a defect of these types.

Our case indicates several points: First, alexia need not occur in a case with right hemianopia and deficit with colors and color names. Detailed behavioral examination is required to delineate color-input deficits from disconnection-type deficits; alexia need not be expected to accompany the former. Second, one or all of the lesions found neuropathologically may serve to impair the normally precise control exerted by a single-color test stimulus over other behavior, the resulting deficit appearing in tasks that require a response to a single color in the absence of other colors for comparison. Dysrelations of this type may be the rule from which disconnections are the exceptions.

Memory deficit. The patient showed a retrograde and anterograde amnesia for the events surrounding his admission, faulty retention of verbal material, impaired retention of a form discrimination test, and an amnesic dysnomia.

The clinical features of his memory impairments and his performance on our laboratory tests were similar to those reported⁴ for H.M. in whom the features appeared twelve years after resection of the bilateral medial temporal regions.³³ His clinical features were also similar to those in patients with autopsy-demonstrated hippocampal damage^{24,34,35} and to those described in unilateral temporal lobe removals.³⁶⁻³⁸ These comparisons suggest that the deficits in our patient were related to the hippocampal infarction.

The pathologic findings indicate only unilateral involvement. One can never positively rule out the possibility that bilateral ischemia occurred initially but we have no evidence of damage in the right hippocampus and related structures. For these reasons, we suggest that the memory deficit was due to involvement of the left hippocampus.

Substantial evidence supports the proposition that bilateral hippocampal involvement

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SUMMARY

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is a necessary condition for amnesia to occur and persist.³⁹ Few cases have been reported of memory loss with a unilateral lesion.^{37,40} In our case, severe memory disorder was still present when last evaluated in detail on day 15. To less systematic evaluation, he did not subsequently improve before his death on day 82. Whether his deficit would have persisted over a longer period remains an open question.

We consider the amnesic dysnomia to be separate. The patient failed to recall the names of people as well as many other individual nouns when the stimuli were presented in visual or auditory form. However, he gave descriptive evidence of previous exposure to these stimuli, that is, he "recognized" them. His failures in naming were associated with circumlocutions, lame excuses for failure, and a general acceptance of the correct name when offered, all characteristic of amnesic aphasia deficits.³¹

Although classically considered a sign of deep temporal lobe involvement,³¹ amnesic aphasia has occasionally been reported, often incidentally,^{17,41} in cases showing infarction in the territory of the dominant posterior cerebral artery. The exact pathologic correlation for such deficits remains unclear, as does the issue of whether memory deficit and amnesic aphasia are functionally related or simply result from a lesion simultaneously involving physically proximate regions of the cerebrum serving separate functions.

SUMMARY

An elderly accountant suffered sudden onset of partial right homonymous hemianopia with memory deficit and impaired performance on certain tests with color. His deficits were fol-

lowed until eighty-two days later when he died. The serially sectioned brain showed patchy infarction confined to the left posterior cerebral artery territory, involving the lateral geniculate body and the hippocampus, slightly involving left lateral dorsal nucleus and the pulvinar of the thalamus, and only undermining the lingual gyrus.

The hemianopia correlated well with the geniculate infarct.

Severe recent memory deficit was repeatedly demonstrated clinically and quantitated by special laboratory tests. An amnesic dysnomia was also found for many previously acquired proper names. The unilateral left hippocampal infarct appears the most circumscribed lesion yet reported with memory deficit.

In color tests, he initially made errors naming colors and crossmatching colors with color names but performed normally on discrimination tests with these stimuli and showed no alexia. The actual error responses on individual trials were not random but bore a relation to the correct response for that trial. The deficit was more a "dysrelation" than a "disconnection" between colors and their names. Later, scores in matching printed color-name test stimuli to color choices became satisfactory while naming of colors and matching colors to printed color-name choices remained unchanged. This evolved deficit was confined to a loss of the precise control normally exercised by single-color stimuli over behavior in the absence of other colors for comparison. The importance of each or all of the lesions found in the lateral geniculate, the pulvinar of the thalamus, and the lingual gyrus remains uncertain.

REFERENCES

1. Fletcher FG, Stoddard LT, Sidman M: A system for unlimited repetitive presentation of auditory stimuli. *J Exp Child Psychol* In press.
2. Sidman M, Stoddard LT, Mohr JP, et al: Behavioral studies of aphasia: Methods of investigation and analysis. *Neuropsychologia* 9:119, 1971
3. Sidman M, Stoddard LT: Programming perception and learning for retarded children. In: *International Review of Research in Mental Retardation*, Vol 2. Edited by NR Ellis, New York, Academic Press, 1966, pp 151-208
4. Sidman M, Stoddard LT, Mohr JP: Some additional quantitative observations of immediate memory in a patient with bilateral hippocampal lesions. *Neuropsychologia* 6:245, 1968
5. Mackenzie I, Meighan S, Pollock EN: On the projection of the retinal quadrants on the lateral geniculate bodies, and the relationship of the quadrants to the optic radiations. *Trans Ophthal Soc UK* 53:142, 1933
6. Kupfer C: The projection of the macula in the lateral geniculate body of man. *Amer J Ophthal* 54:597, 1962
7. LeGros Clark WE: The laminar pattern of the lateral geniculate nucleus considered in relation to color vision. *Docum Ophthal* 3:57, 1949
8. Sheppard JJ: *Human Color Perception*. New York, Elsevier Publishing Co, 1968, p 98
9. Ojemann GA, Blick KI, Ward AA: Improvement and disturbance of short-term verbal memory during human ventrolateral thalamic stimulation. *Trans Amer Neurol Ass* 94:72, 1969

10. Landolt E: De la cécité verbale. In: *Nederlansch tijdschrift voor geneeskunde Feestbundel oan Franciscus Cornelius Donders op den 27 Mei 1888*. Amsterdam, F Van Rossen, 1888, pp 418-433
11. Pözl O: Ueber einige zentrale Probleme des Farbensehens. *Wien Klin Wschr* 61:706, 1949
12. Polyak S: *The Vertebrate Visual System*. Chicago, University of Chicago Press, 1957, p 666
13. Lenz G: Zwei Sektionsfälle doppelseitigen zentraler Farbenhemianopsie. *Z Ges Neurol Psychiat* 71:135, 1921
14. Sittig O: Störungen in Verhalten gegenüber Farben bei Aphasischen. *Mtschr Psychiat Neurol* 49:63, 1921
15. Heidenhain A: Beitrag zur Kenntnis der Seelenblindheit. *Mtschr Psychiat Neurol* 66:61, 1927
16. Schober H: Erworbene Farbenblindheit nach Schädeltrauma. *Graef Arch Ophthal* 148:93, 1948
17. Pallis CA: Impaired identification of faces and places with agnosia for colors. *J Neurol Neurosurg Psychiat* 18:218, 1955
18. Reinhard C: Zur Frage der Hirnlocalisation mit besonderen Berücksichtigung der cerebralen Sehstörungen. *Arch Psychiat Nervenkr* 18:240, 1887
19. Siemerling: Ein Fall sogenannter Seelenblindheit nebst anderweitigen cerebralen Symptomen. *Arch Psychiat Nervenkr* 21:284, 1889
20. Stengel E: The syndrome of visual alexia with color agnosia. *Brit J Psychiat* 94:46, 1948
21. Urechia CI, Cremene V, Popescu P: Hemianopsie avec chromoagnosie. *Rev Neurol (Paris)* 80:70, 1948
22. Lissauer H: Ein Fall von Seelenblindheit nebst einem Beitrage zur Theorie derselben. *Arch Psychiat Nervenkr* 21:222, 1890
23. Lewandowsky M: Ueber Abspaltung des Farbensinnes. *Mtschr Psychiat Neurol* 23:488, 1908
24. Geschwind N, Fusillo M: Color-naming defects in association with alexia. *Arch Neurol (Chicago)* 15:137, 1966
25. Dejerine J: Différentes variétés de cécité verbale. *C R Soc Biol Paris* 4:61, 1892
26. Dejerine J, Vialet N: La localisation anatomique de la cécité verbale pure. *C R Soc Biol Paris* 5:790, 1893
27. Ziehl-Lübeck: Ueber einem Fall von Alexia und Farbenhemianopsie. *Verh Ges Deutsch Natur Aertze* 67(Part 2):184, 1895
28. Merle P: Aphasie et hemiachromatopsie. *Rev Neurol (Paris)* 21:1129, 1908
29. Cumming WJK, Hurwitz LJ, Perl NT: A study of a patient who had alexia without agraphia. *J Neurol Neurosurg Psychiat* 33:34, 1970
30. Pözl O: Die zweite Gruppe der optischen Agnosien. In: *Handbuch der Psychiatrie. Die Aphasielehre. I. Optische-agnostischen Störungen*. Edited by G Aschaffenburg. Wien, Franz Deuticke, 1928, pp 80-193
31. Goldstein K: *Language and Language Disturbances*. New York, Grune & Stratton Inc, 1948
32. Critchley M: Acquired disturbances of color perception of central origin. *Brain* 88:711, 1965
33. Scoville W, Milner B: Loss of recent memory after bilateral hippocampal lesions. *J Neurol Neurosurg Psychiat* 20:11, 1957
34. Victor M, Angevine JB, Mancall EL, et al: Memory loss with lesions of the hippocampal formation. *Arch Neurol (Chicago)* 5:244, 1961
35. DeJong RN, Itabashi HH, Olson JR: Memory loss due to hippocampal lesions. *Arch Neurol (Chicago)* 20:339, 1969
36. Walker AE: Recent memory impairment in unilateral temporal lesions. *Arch Neurol Psychiat (Chicago)* 78:543, 1957
37. Dimsdale H, Logue V, Piercy M: A case of persisting impairment of recent memory following right temporal lobectomy. *Neuropsychologia* 1:287, 1964
38. Milner B: Laterality effects in audition. In: *Interhemispheric Relations and Cerebral Dominance*. Edited by VB Mountcastle. Baltimore, Johns Hopkins Press, 1962, pp 177-195
39. Milner B: Amnesia following operations on the temporal lobes. In: *Amnesia*. Edited by CMW Whitty and OL Zangwill. London, Butterworth, 1966, pp 109-133
40. Sweet WH, Talland GA, Ervin FR: Loss of recent memory following section of fornix. *Trans Amer Neurol Ass* 84:76, 1959
41. Wyllie J: *The Disorders of Speech*. Edinburgh, Oliver & Boyd, 1894, pp 340-344