## Clinical and scientific communications

## Anatomical Study of a Posterior Cerebral Lesion Producing Dyslexia

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After an "occipital lobectomy" that resulted in a severe dyslexia and a moderate dysgraphia-dyscalculia, anatomical study showed damage to the posterior extremity of the angular gyrus and degeneration in the posterior pulvinar. This is in contrast to an earlier case that had degeneration in the anterosuperior pulvinar associated with a small anterior temporoparietal infarct and a well-documented receptive-expressive aphasia. However, the role of the pulvinar in speech function remains uncertain. The surgeon should be aware of the short distance between the angular gyrus and both the midline and the occipital pole because a lesion here during an "occipital lobectomy" produces a distressing and durable speech impairment.

Key words: Angular gyrus, Dyscalculia, Dysgraphia, Dyslexia, Occipital lobectomy, Pulvinar, Speech

#### INTRODUCTION

Déjerine recognized four major areas in which lesions cause clinically distinguishable speech defects: (a) Broca's region (in which Déjerine included the posterior part of the 3rd frontal gyrus and the surrounding area); (b) Wernicke's region, corresponding to the posterior part of the 1st and 2nd temporal convolutions and considered as a center for the auditory images of words; (c) the angular gyrus, considered as the center for visual images of words; and (d) the subcortical region, in which lesions cause verbal deafness and pure alexia (5, 6).

Déjerine's opinions regarding the localization of specific speech functions were countered by the globalistic doctrines of Marie (14), but (with Foix (15)) Marie did acknowledge the predominance of alexia in lesions of the angular gyrus. The swing toward specific localization of speech disturbances was continued by Henschen (11, 12) and Kleist (13), reaching an extreme position with Neilsen's monograph (18).

Current textbooks show a remarkable diversity in the cortical regions designated as areas subserving language function. Many texts restrict the temporoparietal speech representation to the cortex immediately about the posterior extremity of the sylvian fissure of the dominant hemisphere, perhaps because of evidence presented by Penfield and Roberts (22). As a result of the restricted cortical exposures used for anterior temporal lobectomy, they found the majority of the aphasic responses to cortical stimulation in this region. However, aphasic responses can also be elicited well back to the parieto-occipital junction (34), conforming to Déjerine's original outline (Fig. 1).

In the present case of dyslexia with a mild dysgraphiadyscalculia, it was possible to define the cortical damage and thalamic degeneration with some precision. There has been no previous detailed anatomical study of a lesion lying this far posteriorly in the dominant hemisphere that resulted in the disturbance of language.

#### CASE REPORT

### Summary

This 38-year-old woman had an acute hemorrhage about a metastatic melanoma in the left occipital region. This was decompressed with amputation of the occipital pole. For 2 weeks she had cortical blindness attributed to transient compression of the posterior cerebral arteries by acute transtentorial herniation. Later, she had a right hemianopsia with a trace of visual sparing in the right periphery.

For the remaining 9 months of her life, the patient remained oriented and spoke without difficulty. She could give a reasonable description of a picture, named objects without difficulty, differentiated right from left, and showed no apraxia. She continued, however, to have difficulty reading. Reading aloud was laborious and words were often substituted without recognition of the error. There was little or no comprehension of what she read either aloud or silently. Writing and calculation were accomplished with considerable effort, and the results were much below the level expected from her educational background.

#### Report

This right-handed woman was 38 years old at the time of her death on September 9, 1957. In April 1954, a primary melanoma was discovered in the left axilla and was resected with a wide axillary lymph node dissection. In May 1955, there were multiple cutaneous metastases and metastases to the lungs. The cutaneous metastases subsequently disappeared spontaneously. In February 1956, the patient had a convulsion, nausea, and headache.

On December 18, 1956, examination showed an incomplete right homonymous hemianopsia with retention of vision of gross objects in the upper portion of the visual field on the right. By December 25, 1956, this hemianopsia had become

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complete and there was bilateral early papilledema. There was a fine nystagmus on extreme lateral gaze to either side. On December 27, 1956, a left occipital craniotomy was carried out because of sudden semicoma. The metastatic melanoma and surrounding blood clot were removed from the occipital lobe.

During the postoperative period the patient showed confusion and right-left disorientation. Although she denied any difficulty with vision, she was obviously blind to light and movement. The pupils reacted to light and the extraocular movements were full. By January 5, 1957, she began to have some correct visual responses but often confabulated about a variety of impossible activities while she was actually confined to bed. By January 9, 1957, vision was 10/30 in both eyes (chart held 10 feet away over the foot of the bed) but there was a dense right homonymous hemianopsia.

By March 11, 1957, she was affable but slow to respond. She was unable to read usefully, substituting letters (e.g., "forever" was read as "foreven"). She gave a sparse but reasonable description of a picture, did simple sums orally, named simple objects, and differentiated right from left.

On June 3, 1957, speech examination was as follows:

Verbal commands. "Close your eyes and open your mouth" and "Pat your left ear with your hand and open your mouth three times" (done correctly).

Written commands. "Stick out your tongue and close your eyes" (read slowly, then reread, but she did it correctly). "Pat your head three times" (done correctly).

Writing. Wrote name correctly. Correctly wrote "It is a rainy day" to dictation. When asked to describe what she saw out the window, she wrote, "I see lots of green trees and the golf course. There are some houses in the background." She remarked that it was hard work writing. "I never was a writer." (She was a college graduate.)

Drawing. A daisy and a clock face were drawn without loss of symmetry.

Oral arithmetic. The following were done correctly: 12 + 13; 17 + 18;  $11 \times 12$ ; 27 - 18; 47 - 13; and  $3 \times 7 - 1 \times 2 + 10$ . Written arithmetic.

When she was asked to check this she changed the answer to 472.

Reading. She remarked that "things don't look right" and that she could not read for pleasure. When asked to read aloud she did so very slowly, one word at a time and without inflection. She replaced words at times, such as "bulk" for "bull," and did not recognize the error. Her comprehension of the paragraph was very scanty, but what was given was correct.

In March, June, and August 1957 she was examined by J. M. Olhoeft, M.A., speech pathologist and audiologist. The tests administered were the Halstead-Wepman aphasia screening test, the Eisenson test, and proverbs and nouns. Her responses stabilized by the summer of 1957 and examination on August 15, 1957, was reported as follows:

Despite her obvious discomfort, Mrs. S cooperated well and appeared alert.

Testing revealed no demonstrable agnosias, apraxias or left-right disorientation. Simple arithmetic reasoning, thought problems, continued to present difficulties but paper and pencil executions of simple arithmetic problems were completed correctly. She stated that she no longer has difficulty in finding words and almost no

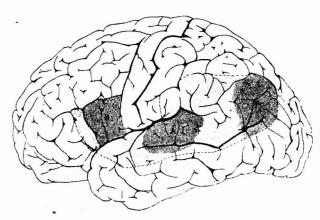


FIG. 1. The major speech areas of the dominant hemisphere as outlined by Déjerine. The angular gyrus (Pc, pli corbe) is outlined medially by the interparietal sulcus (unidentified in the present illustration). (A, Wernicke's area; B, Broca's area.) Only the posteromedial margin of the area associated by Déjerine with the "visual images of words" was affected in the present case. (From J. Déjerine: Semiologie des affections du système nerveux. Paris, Masson et Cie, 1914.)

aphasic errors were observed. Verbal abstractions fluctuated and appeared to be of the same quality as those given in June. Written abstractions were not tested.

Silent and oral reading remained difficult for the patient. She is a slow word by word reader who tends to mis-see or misread pictures and words. (A button was called a ball. Trees was read "tries").

Tangent screen examination on June 6 and July 31, 1957, with 1- and 10-mm white objects at a 1-m distance showed a right hemianopsia with mild enlargement of the left blind spot. On perimeter examination (330 mm) the patient reported that she saw the test object in poorly defined areas 25 to 45 mm to the right of the fixation point with both eyes. The responses were not consistent enough to plot. The visual acuity was normal.

In the spring of 1957 the patient received a course of thiotepa (Lederle Laboratories, Pearl River, New York), and then she received methotrexate (Lederle Laboratories), 4 mg/kg, for three doses at 3-week intervals in the summer of 1957. She died on September 9, 1957, of widespread metastatic malignant melanoma and pneumonia.

#### MATERIAL AND METHODS

The brain was suspended in 10% neutral formalin. After 2 weeks, the pia-arachnoid was removed from the left hemisphere to clarify the gross cortical morphology. The thalamus and brain stem were dissected away and embedded in paraffin. Sections transverse to the longitudinal axis of the hemispheres disclosed a  $3-\times 4-\times 5$ -cm metastatic melanoma in the medial right temporal region. Sections of the left hemisphere at 1-cm intervals were prepared in cresyl violet and myelin (Heidenhain) pairs from the lateral geniculate body to the occipital pole. In the brain stem, similar sections (25  $\mu$  thick and oriented perpendicular to the longitudinal axis of the brain stem) were retained at 250- $\mu$  intervals. These ran from the thalamus down to the upper third of the pons.

The reconstructions were made by placing the slides in the negative carrier of a 5-  $\times$  7-inch enlarger and preparing 11-  $\times$  14-inch negative prints on Kodak photomechanical "T" paper. Enlargements were used for the cerebral sections (about 3 $\times$ ) and for the brain stem (12 $\times$ ). At the latter magnification, the

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larger neurons appeared clearly in the cresyl violet preparations. The reconstruction was made from cresyl violet myelin pairs of sections at about 500- $\mu$  intervals. The differences between numbers of the reconstructions indicate the interval in microns

The nomenclature used in the figures was derived from several sources. The cortical nomenclature was taken from Economo and Koskinas (7), the tract terminology from Riley (25), and the nomenclature of the brain stem nuclei from Olszewski and Baxter (20). Regretfully, we found it necessary to abandon the macaque thalamic nomenclature (37), which does not conform to the realities of the human thalamus in the ventral and lateral region. In its place we have used the system proposed by the Vogts (35, 36) with slight modification (30–32). This system has been presented recently with cytoarchitectural diagrams (33); the approximate Anglo-American equivalents are given in Table 1.

#### **EXAMINATION OF THE BRAIN**

In the right medial temporal region, a metastatic melanoma invaded the hippocampal gyrus and extended anteriorly to the uncus and posteriorly to the transverse level of the colliculi. The ventricular system was displaced to the left, and the right cerebral peduncle was indented on its anteroinferior aspect. The inferior aspect of both hemispheres showed prominent tentorial notching that extended to all of the cerebral cortex immediately adjoining the tentorial opening on both sides.

No other metastases were found in the brain. No lesion other

than the region of cortical infarction and surgical removal was seen in the left hemisphere.

In the left occipital lobe, the surgical removal extended 2.5 cm anterior to the pole and destroyed the posterior calcarine

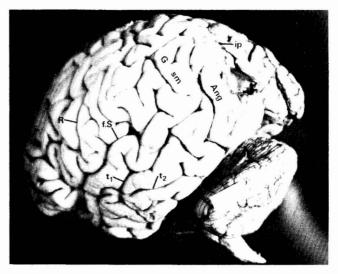


Fig. 2. The surgical lesion destroyed the cortex within the interparietal sulcus (ip) and the medial portion of the adjacent angular gyrus (Ang). R, fissura Rolandi; f.S, fissura Sylvii;  $t_1$   $(t_2)$ , sulcus temporalis primus (secundus); Gsm, gyrus supramarginalis.

TABLE 1
Abbreviations

	ucleus amygdalae	P1	Pallidum laterale
B Nu	ucleus basalis	Pm	Pallidum mediale
B cj Bra	rachium conjunctivum	P pd	Nucleus peripeduncularis
	rachium colliculi inferioris	Pret	Regio pretectalis
Ce Nu	ucleus centralis (Nucleus centrum medianum)	pr G	Nucleus praegeniculatus
	orpus mammillare	Pu	Nucleus pulvinaris
Cm a Cor	ommissura anterior	Put	Putamen
Cm p Cor	ommissura posterior	Pv	Nucleus paraventricularis hypothalami
Coe Nu	ucleus locus coeruleus	R	Nucleus reticularis
Co i Col	olliculus inferior	Ru	Nucleus ruber
Co s Col	olliculus superior	Smth	Stria medullaris thalami
D b cj Dec	ecussatio brachii conjunctivi	Sn	Substantia nigra
EW Nuc	ucleus Edinger Westphal	So	Nucleus supraopticus hypothalami
F For	ornix	Spt	Nucleus supratrochlearis
Fa Nu	ucleus fasciculosus (Nucleus ventralis anterior, medial	Sth	Nucleus subthalamicus
p	part)	TM	Tractus Meynerti
F Is Fas	asciculus longitudinalis superior	Tmth	Tractus mammillothalamicus
G 1 Cor	orpus geniculatum laterale	TO	Tractus opticus
	orpus geniculatum mediale	Vc	Nucleus ventrocaudalis (Nucleus ventralis posterior, pos-
iLa Nuc	ucleus intralamellaris (the intralaminar nuclei)		terior part)
Is Nuc	ucleus interstitialis	Vc e	Nucleus ventrocaudalis externus (Nucleus ventralis pos-
IV Ner	ervus trochlearis		terolateralis, posterior part)
Li Nu	ucleus limitans	Vc i	Nucleus ventrocaudalis internus (Nucleus ventralis poster-
	emniscus medialis		omedialis, posterior part)
	ucleus lateropolaris (Nucleus ventralis anterior, lateral two-thirds)	Vc pc	Nucleus ventrocaudalis parvocellularis (Nucleus ventralis posterior medialis parvocellularis)
M Nuc	ucleus medialis (Nucleus medialis dorsalis)	V im	Nucleus ventrointermedius (Nucleus ventralis posterior,
N Nuc	ucleus		anterior part)
N III Nuc	ucleus nervi oculomotorii	Voe	Nucleus ventrooralis externus (Nucleus ventralis lateralis,
N IV Nuc	ucleus nervi trochlearis		ventrolateral part)
N V me Nu	ucleus nervi trigemini mesencephalicus	Voi	Nucleus ventrooralis internus (Nucleus ventralis lateralis,
N tub Nu	ucleus tuberalis		ventromedial part)
Pf Nu	ucleus parafascicularis	Zi	Zona incerta
Pi Pin	neal		

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cortex. The surgical defect extended 2.5 cm lateral to the midline at the occipital pole and forward from the occipital pole to destroy the most posterior and medial portion of the angular gyrus (Fig. 2). The inferior and basal portions of the parietal cortex were normal. On the medial aspect of the occipital lobe, the resection included the posterior 2.0 to 2.5 cm of the calcarine cortex, the upper portion of the lingual gyrus, and the lower portion of the cuneus (Fig. 3).

In the left parieto-occipital region (Fig. 4), the cavity occupied by the hemorrhage and metastatic lesion removed at operation undercut the angular gyrus some 0.5 cm anterior to its cortical extent on the lateral surface. The posterior portion

of the remaining calcarine cortex was surrounded by the operative cavity. A narrow extension of the cavity came to the cortical surface in the lingual gyrus.

Examination of the histological sections (Fig. 5) disclosed small areas of infarction in the superior lip of the calcarine fissure and within the occipitotemporal sulcus. Segments of myelin loss and gliosis appeared in the stratum sagittal externum and internum that could be followed forward into the temporal lobe. The tapetum remained well myelinated, and no damage was found in the splenium of the corpus collosum (Fig. 5c). Microscopic examination of the right occipital lobe and calcarine cortex did not show any damage.

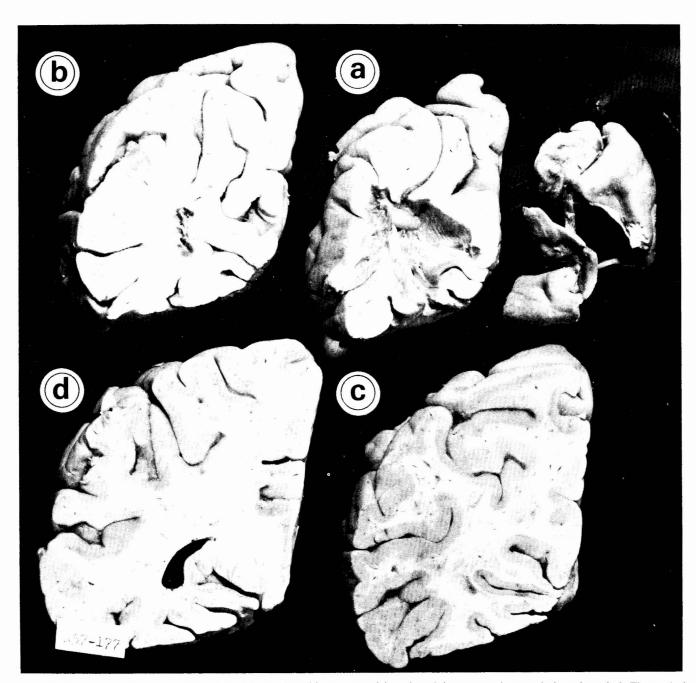


Fig. 3. Transverse sections of the left occipital lobe starting with remnants of the pole and then progressing anteriorly (a through d). The surgical cavity undercuts slightly the remaining angular gyrus.



FIG. 4. The medial aspect of the left occipital lobe to show the levels from which the sections shown in Figure 5 were taken. Note the shrinkage of the superior lip of the calcarine fissure, which was the site of infarction. The corpus callosum including the splenium was intact.

### ANATOMICAL RECONSTRUCTION STUDIES

Thalamus (Fig. 6)

Pulvinar. There were two distinct areas of cell loss and gliosis. One was inferolateral, limited by the external medullary lamina laterally and the posterior margin of the nucleus ventrocaudalis anteroinferiorly. The degeneration extended downward to include the posterolateral inferior extremity of the pulvinar.

The second region of degeneration in the pulvinar was seen on the ventricular aspect of the nucleus behind the habenula and extended forward (#220) to the scattered groups of cells of intralaminar type that separate the nucleus medialis and the pulvinar. It retained this position in more inferior sections, coming into contact with the posterior aspect of the nucleus limitans. Due to the mode of trimming the block, the full superior and posterior excursion of the pulvinar cell loss lesions could not be determined.

Lateral geniculate body. Total cell loss and gliosis affected the apex of the geniculate pyramid and became constricted to a central region of degeneration (#8320) in the lower segment of the nucleus. At this level, cells were retained in the anterior as well as the medial and lateral margins of the nucleus. The

remainder of the thalamus that was available for study was negative.

Brain stem (Fig. 7)

In the caudal mesencephalon, there was a linear lesion of small diameter that extended some 4 mm (#2820 to 6820) but at no point exceeded 2 mm in its greatest diameter. This was first noted in the inferolateral portion of the inferior colliculus; then it gradually shifted to a position in the dorsal tegmentum immediately lateral to the locus coeruleus and the nucleus nervi trigemini mesencephalicus. The appearance of the lesion with striking gliosis and loss of myelinated fibers suggested a microinfarct rather than secondary degeneration.

This lesion seemed to be related to the patient's nystagmus. On March 11, 1957, she had horizontal nystagmus on lateral gaze bilaterally, enhanced by gaze to the left. On June 3, 1957, she had a horizontal central gaze nystagmus with the quick phase to the right on fundiscopic examination. The amplitude increased on lateral gaze bilaterally with a higher frequency on gaze to the left.

#### DISCUSSION

The patient's right hemianopsia correlated with the extensive lesion of the left calcarine cortex. The small remaining fragment in the anterior portion apparently permitted her to vaguely see large moving objects in the far periphery of the right visual field.

Cell loss in the lateral geniculate body spared only a thin rind on the anteromedial and anterolateral aspects of the nucleus. The cell loss corresponds to the macular representation found by Polyak and Hayashi (23) in the chimpanzee plus additional anterior extension correlated with the lesion that spared only the anterior calcarine cortex.

The hemianopsia was considered to contribute little to the language defect. In our experience, even lesions of the anterior radiation producing a macula-splitting right hemianopsia (Ref. 29, Case UN) do not diminish the patient's ability to read or enjoyment of reading. Another case of right hemianopsia without reading difficulty was reported by Foix and Hillemand (9). In agreement with Déjerine (4, 5), these authors pointed out that an additional lesion of the splenium was needed to produce alexia

Déjerine provided the first well-described case of verbal "blindness" and agraphia with a reasonable anatomical correlation (3). Immediately after his stroke, the patient had a "probable" right hemianopsia. After recovery from paraphasia, alexia remained complete although the patient could recognize figures. Writing ability was limited to tracing his name; he could write neither spontaneously nor to dictation. In Déjerine's drawing of the brain, he indicated a softening ("the size of a 5-franc piece") that occupied the inferior three-quarters of the left angular gyrus. This lesion penetrated to the ventricle at the trigone, transecting the radiations of Gratiolet.

Hécaen et al. made a detailed study of language and cognitive defects after occipital lobectomy in seven patients (10). Three of these cases were followed for 9 to 33 months after operation. Immediately after the lesion there was total alexia that included numbers and musical notation, but no agraphia. Words posed more of a problem than letters. This improved in about a month so that the patients could read familiar texts in a broken fashion. After 4 to 5 months, reading became more rapid but still involved hesitation, a tendency to spell words, and occasional paralexias. The patients were often troubled if the texts were exposed only briefly or they were asked to read in silence. They tended to follow the text with a finger, com-

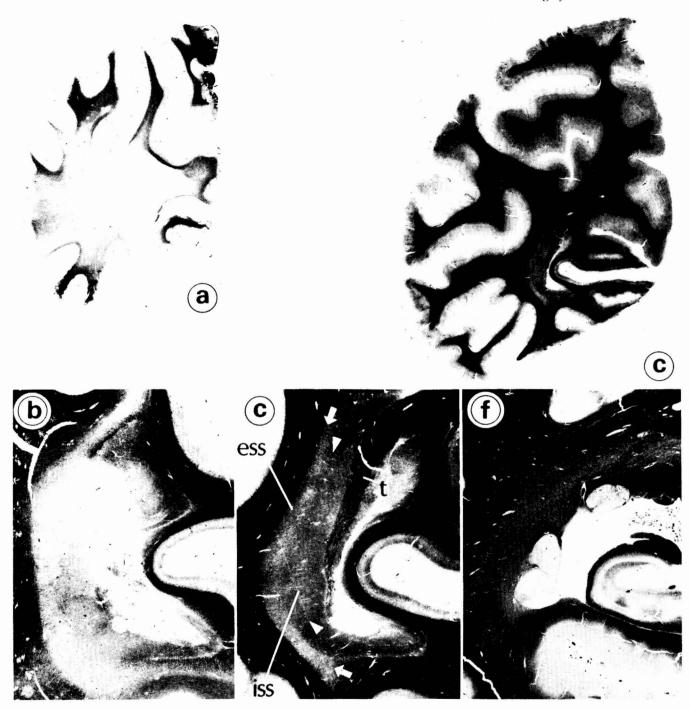


Fig. 5. Sections of the left occipital lobe taken at the levels indicated in Figure 4. As best seen in Section c, the central (lateral) portion (between the arrows) of the external sagittal stratum (ess) showed marked demyelination. The demyelination of the internal sagittal stratum (iss) was of lesser degree and the vertical extent of this was less (between the arrowheads) than that in the external sagittal stratum. The tapetum (t), lying yet more medially, showed no degeneration. Section f was taken through the posterior temporal region at the level of the lateral geniculate body. The myelin loss in the sagittal stratu remains more posterior but the external sagittal stratum at this level is thinner and is seen only as a light band on the lateral margin of the internal sagittal stratum. (The dark cortical markings on the superomedial and inferolateral corners of Section a are staining artifacts.)

plaining that the lines ran together. A distinctive feature, despite a greater or lesser degree of recuperation, was persistent lack of inclination to read. The reading problems seemed to be independent of other speech difficulties that might be minimal or transitory. Postoperatively, none of the patients could undertake the same social activity that they had enjoyed prior to

operation. Patients were tested with a variety of perceptive tests. The most important defect was inability to abstract and understand the total significance of a situation.

The "occipital lobectomies" encroached more or less upon the parietotemporal cortex. Reference in some cases to opening the occipital horn suggested that considerable undercutting of

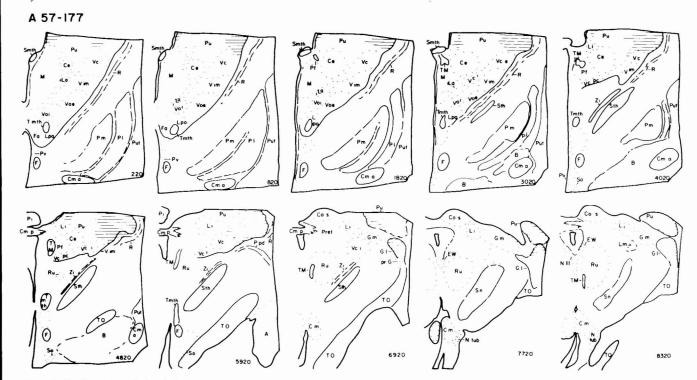


FIG. 6. Sections through the thalamomesencephalic region oriented perpendicular to the longitudinal axis of the brain stem. Areas of cell loss are indicated by horizontal hatching in the pulvinar (Pu) and the lateral geniculate (G l). See text for description. The other abbreviations are given in Table 1. The difference between two section numbers indicates the interval between the sections in microns.

the parietal cortex may have occurred. Unfortunately, there was no detailed anatomical account to compare with the painstaking clinical records.

Understanding of the relationship between natural lesions of the thalamus and language function has been hampered by a lack of anatomical studies. Earlier cases suggested that damage to the pulvinar itself might cause less severe dysphasia than damage to the cortical regions to which it projects.

Only 2 of 24 thalamic tumors reported by McKissock and Paine were accompanied by aphasia (16). Ciemins reported an instance in which a hemorrhage seemed to destroy all of the thalamic nuclei on the left side, but the patient had only a decrease in spontaneous speech, difficulty in carrying out complex commands, difficulty in writing, and a defect in describing material already read (2). In other cases (1, 2, 8, 17, 26), there was only mild dysphasia despite large thalamic lesions. In their case, Reynolds et al. showed a posterior thalamic lesion by computed axial tomography, but clinical details were limited to a report of "severe aphasia" (24).

The limited clinical data suggest that the stable "thalamic speech deficit" may appear most commonly as anomia with neologisms and paraphasic logorrhea but relatively intact repetition and comprehension of spoken speech. Testing is frequently difficult because of a fluctuating level of response. None of the earlier reports provided more than a gross indication of the size and position of the lesion.

However, specific areas in the pulvinar may be related to specific speech function. In one case of predominantly receptive aphasia with good clinical documentation, cell loss in the anterosuperior portion of the pulvinar was associated with a lesion at the posterior extremity of the sylvian fissure severing fibers from the supramarginal and anterior angular gyri (27). Taken in conjunction with the stimulation evidence of Ojemann et al. (19), this seemed to support the importance of the anterosuperior pulvinar to the receptive aspects of speech.

In the present case, the region of the pulvinar projecting to the posterior angular gyrus (hence related to language function, primarily reading) lay in the inferior, posterior, and lateral portion. Another case studied in serial section (MU-64-64) also suggested that the posteroinferior and inferolateral portion of the pulvinar might be related to cortex needed for reading skills (32). This patient could read but had little understanding of what he read; however, these symptoms were overshadowed by memory loss and other symptoms related to the bilateral cerebral lesions. The cell loss in the pulvinar of the present case was quite similar to that of MU-64-64 with the exception that degeneration also extended into the medial portion of the pulvinar immediately behind the nucleus medialis.

In reconstructions of 28 human hemispheres with degeneration in the pulvinar after focal cerebral lesions (31), only 1 case (W-69-47) showed cell loss immediately behind the nucleus medialis in the medial portion of the pulvinar. This was associated with an extensive lesion of the occipitoparietal region that spread forward on the medial aspect of the hemisphere into the retrosplenial portion of the limbic (cingulate) gyrus, including the anterior paracalcarine cortex. In contrast, Case A-64-96-R, with a cortical infarct destroying the superior medial face of the hemisphere in the precuneus and the superior portion of the cuneus but sparing the anterior paracalcarine cortex, had no degeneration in this region of the pulvinar. In the present case, the lesion undercut the anterior paracalcarine cortex, confirming a relationship between this cortical region and the segment of the pulvinar lying immediately behind the nucleus medialis. Although it is part of the visual association area, this region and the associated anteromedial region of the pulvinar seem unrelated to the primary cortex subserving

From a surgical aspect, the present case emphasized the short distance between the angular gyrus and the occipital pole. In fixed postmortem brains, the distance between the tip of the

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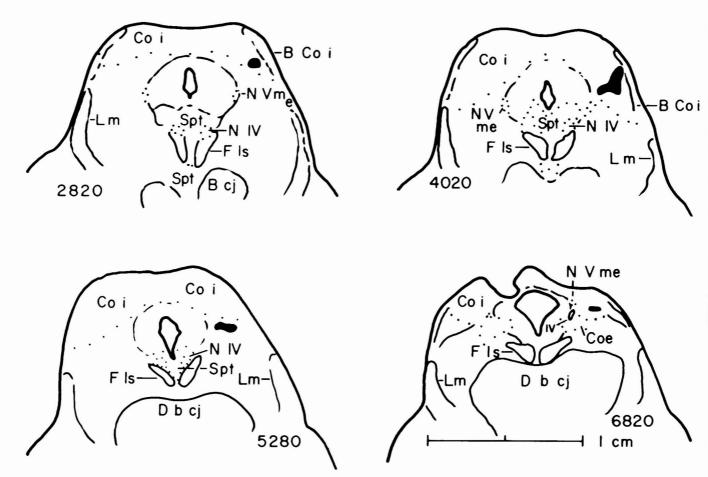


Fig. 7. Sections showing the small area of infarction (black) inferior and lateral to the left inferior colliculus (Co i). See text for description. The other abbreviations are given in Table 1. The difference between two section numbers indicates the interval between the sections in microns.

occipital pole and the posterior margin of the angular gyrus averaged only 3.5 cm (34). At operation, the narrow gyri may identify the occipital lobe, but with the pia-arachnoid intact the outlines of the angular gyrus may be vague. The most reliable means of identification of cortex indispensable for speech is electrical stimulation of the cortex during craniotomy under local anesthesia (21, 28). Short of this, recognition of the anatomical relationships should permit the surgeon to use a more medial or posterior entry into the occipital region and thereby spare language function.

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## **COMMENTS**

The case reported by Dr. Van Buren re-emphasizes the importance of the posterior part of the language area of the dominant hemisphere, particularly in the region of the angular gyrus, to a disorder in which difficulty in reading is the most prominent finding. In a case such as his, with a relatively large lesion, it is hard to know whether the defect is due to cortical damage or to destruction of fibers connecting with the intact visual cortex of the other side or perhaps with the thalamus. But isolated dyslexia can occur in the absence of a visual field defect, as was seen in a patient of mine who has intact visual fields, a CT scan-documented stroke involving the posterior region of the parieto-occipital junction in the back end of the posterior language area, and a persisting moderate dyslexia without oral or written language disturbance. A somewhat

similar case, although with a less persisting deficit, was described by Greenblatt (2).

The difficulty with the models derived from these individual cases is that, if one maps the location of the cortex concerned with various aspects of language in individual patients, one finds a substantial degree of individual variability. I have reported this for oral language function, as measured by electrical stimulation mapping during naming (3), and have seen an even greater individual variability in the location of sites where electrical stimulation alters reading. Indeed, these presently unpublished findings suggest that reading is a process that involves rather wide brain areas, not only the cortex around the posterior language area but also extending forward into the superior temporal gyrus on occasion and in portions of the frontal lobe superiorly and anteriorly to the classical anterior language area of Broca. Of course, these findings are all in a select patient population, only those coming to craniotomy under local anesthesia for medically intractable epilepsy, but alexia without agraphia has also been recently described with strokes in the frontal lobe of the dominant hemisphere (1). The neurosurgeon should recognize that if he approaches close to the language cortex from any direction he is running a risk of a potentially quite disabling reading disturbance. I reemphasize Dr. Van Buren's suggestion that the only way to avoid this problem reliably is to individually map these language functions in the cortex of the awake patient.

What role the thalamic deficits might play in reading is still undetermined. Dr. Van Buren summarizes the data indicating the relationship of the dominant thalamus to spoken language and points out the considerable specificity of lesions within the pulvinar that are associated with oral language deficits and, in this case, a reading deficit. This high degree of specificity in the pulvinar probably explains why the induced pulvinar lesions that at one time were popular for treatment of spasticity did not seem to be associated with much of a language disturbance because they were placed relatively low in the pulvinar, apparently well away from the areas that may be related to spoken language. Reading was less thoroughly tested in those

patients, however.

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This case report by Van Buren is unique because of its anatomical elegance. In this respect, there are two distinct and seemingly unrelated topics that deserve comment:

- 1. The thalamic lesions. In the late 1960s and early 70s there was a flurry of activity among investigators who were trying to understand the relationship between known thalamocortical connections and clinical disorders of language. A lot of apparently valid data has been accumulated, and the work still goes on, but a viable working model of the relationship still eludes us. Detailed studies such as the present one can only serve to advance the cause.
- 2. The cortical and connecting white matter lesions. Whatever meager reading ability remained to the patient could have been mediated only by visual information which was arriving in the right occipital lobe and thence transmitted to the left angular gyrus region. Because the splenium was intact, the information could potentially get across the midline. But it could not reach the largely intact left angular gyrus because the connecting pathways in the left occipital lobe had been

<sup>1.</sup> Benson, D. F. The third alexia. Arch. Neurol., 34: 327-331, 1977.

Greenblatt, S. Subangular alexia without agraphia or hemianopsia. Brain Lang., 3: 209–228, 1976.

Ojemann, G. Individual variability in cortical localization of language. J. Neurosurg., 50: 164–169, 1979.

removed. (For further details and diagrams of the pathways, see Greenblatt (5)). Particularly important in the present case is Van Buren's notation of the fact that the angular gyrus had been significantly undercut during the lobectomy. Undercutting of the left angular gyrus can lead to alexia even without a formal lobectomy (4). The pathways that are thought to be interrupted include the extreme sagittal stratum (vertical occipital fasciculus) (3, 5). In the present case the more anterior limb of this pathway was found to be degenerated.

The practical surgical implications of Van Buren's case are all derived from the anatomy of the cortical and white matter lesions.

- 1. The warning to the surgeon about dominant occipital lobectomies invading the angular gyrus has been sounded before by Hecaen et al. and Rasmussen (6), among others, and the surgical location of the angular gyrus has been emphasized (5), but caveats such as this are justifiably repeatable.
- 2. The importance of avoiding undercutting of the angular gyrus, as well as direct damage to it, also bears repeating.
- 3. The urgent nature of the surgery in this particular case shows the importance of having some useful surgical guidelines to the location of language-related structures for those occasions when it is impractical to do cortical stimulation. Even the stimulation technique may lead one astray if specific reading tests are not included.
- 4. With regard to "surgical locations," we must be careful in our statements about surgical distances. When Van Buren speaks of a lobectomy carried 2.5 cm anterior from the occipital pole, he presumably means that it was carried in a straight posterior-to-anterior direction, at right angles to a true coronal section. But surgical distances also may be measured (before resection) along surfaces that are actually curvilinear, and then the distances may seem to be much larger. For example, the

approximate center of the angular gyrus is 9 cm forward from the inion and 4½ cm lateral to the midline when measured along the surface of the exposed skull (5).

Many reports of cases such as this are strong on the clinical side but weak anatomically. Here, on the other hand, we have the reverse situation. It would be of interest to know more about the patient's ability to perform verbal repetition, her reading of single words, her spelling, and her color-naming abilities. In addition, the report would have been considerably strengthened if more extensive formal psychological and reading achievement tests had been done. By and large, these omissions are probably attributable to the fact that the patient was seen in the middle 1950s, a decade before the beginning of the current surge of interest in the corticocortical disconnection syndromes (1, 2). The publication of a case such as this in a neurosurgical journal seems to indicate that renewed interest in this fascinating field is beginning to develop among neurosurgeons as well as neurologists.

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