

Neurologic Perspectives on Right Hemisphere Language Following Surgical Section of the Corpus Callosum

Michael S. Gazzaniga, Ph.D., Ruth Nass, M.D.,† Alexander Reeves, M.D.,‡ and David Roberts, M.D.‡*

Over the past 25 years, there have been more than 100 cerebral commissurotomy procedures carried out in an attempt to control intractable epilepsy. In the early 1960s, the surgical series initiated by Bogen and Vogel¹ stood alone in this effort and was not significantly augmented until the late Donald Wilson performed the operation starting in the early 1970s.^{2,3} The therapeutic success of his series gave rise to renewed interest in the procedure, and in the mid 1970s, several medical centers embarked on similar projects.⁴ As the number of patients increases, new insights are possible on a variety of issues concerning the lateral organization of cerebral functions. In this review, we will examine the issue of whether or not right hemisphere language, when it is present following commissurotomy, can be understood in terms of predisposing neuropathologic processes.

To date, only five of the approximately 50 patients who have undergone corpus callosotomy for seizure control and who have been neuropsychologically examined show evidence of right hemisphere language.⁵ There are no published studies other than on these five patients.⁶⁻¹¹ Furthermore, although the right hemisphere is language competent to varying degrees in these five patients, the left hemisphere remains the language-dominant hemisphere in all.

The apparent paucity of right hemisphere language in this population runs counter to the claim that right hemisphere language is rather common in the general population, but functionally suppressed by left hemisphere dominance.^{7,12} Moreover, the apparent rarity of right hemisphere language is somewhat paradoxical in view of the common criticism of split-brain studies, which suggests that the epileptic's brain is atypically orga-

nized as the result of neurologic disease. The implication of this criticism is that right hemisphere language should be excessively frequent in the split-brain population; yet, the frequency of right hemisphere language seen in the split-brain population is generously estimated at 10%, which is only slightly more frequent than in the typical right-hander.^{13,14}

In this article we will try to determine any features of the medical histories that suggest possible predisposing factors that may explain the presence of right hemisphere language in these five patients. In addition the history of five patients who do not show evidence of right hemisphere language will be summarized for comparison. We will discuss several critical factors that are thought to contribute to the biology of language processes, such as the timing of brain injury, the location and type of injury, mechanisms of reorganization, handedness, sex, individual variability, and intelligence, and assess their relevance to these particular case histories.

POSSIBLE FACTORS CONTRIBUTING TO BILATERALIZATION OF LANGUAGE AND SPEECH

TIMING OF INJURY

It is generally accepted that there is greater recovery due to reorganization of function when damage occurs in the immature compared with the mature nervous system.¹⁵ Rasmussen and Milner¹³ examined language lateralization in large numbers of epileptic patients with apparent brain damage

*Department of Neurology, Cornell University Medical College, New York, New York

†Departments of Neurology and Pediatrics, Cornell University Medical College, New York, New York

‡Division of Neurology and Department of Neurosurgery, Dartmouth Medical School, Hanover, New Hampshire

Reprint requests: Dr. Gazzaniga, Department of Neurology, Cornell University Medical College, 1300 York Avenue, New York, NY 10021

Publisher: Thieme-Stratton Inc., 381 Park Avenue South, New York, NY 10016

Table 1. Speech Lateralization as Related to Handedness in 134 Patients with Definite Clinical Evidence of an Early Left Hemisphere Lesion

Handedness	No. Cases	Speech Representation		
		Left	Bilateral	Right
Right	42	34 (81%)	3 (7%)	5 (12%)
Left or mixed	92	26 (28%)	17 (19%)	49 (53%)

Reprinted with permission from Rasmussen and Milner.¹³

using the intracarotid amobarbital procedure. The injection of a short-acting barbiturate into the internal carotid artery anesthetizes the ipsilateral hemisphere and allows the investigator approximately 3 to 5 minutes to assess the language capacities of the nonanesthetized hemisphere.

When there is definite clinical evidence of early (before age 6 years) left hemisphere injury, language lateralization is often altered (Table 1). Yet, this is not a constant finding. Among patients maintaining right-handedness, the majority continue to have only left hemisphere language representation despite the injury. Although reorganization of language representation occurs more frequently in those who are or become mixed or left-handed, bilateral language is the least common organizational outcome. In addition, it should be noted that bilateral language (as demonstrated at intracarotid amobarbital testing) may be present in two forms. Language representation may be a mirror image in the two hemispheres, or naming and series speech may be dissociated, each hemisphere being capable of mediating only one of these two expressive language functions. These two patterns occur with equal frequency when language is bilateral, regardless of the age of the patient at the time the lesion occurs.¹³

Another source of data regarding language reorganization after early left hemisphere injury (before language acquisition) comes from studies of patients with congenital and infantile right hemiplegia who undergo left hemispherectomy for control of seizures. As a rule, these patients do not develop aphasia at the time of hemispherectomy.¹⁶ Although the damaged left hemisphere may have had residual language capacity in some cases,¹³ sparing of language in this setting requires that the right hemisphere is (or has become) the language hemisphere.

The amobarbital and hemispherectomy data for patients with early lesions stand in marked contrast to the data on language reorganization when the mature central nervous system is damaged. Rasmussen and Milner's¹³ intracarotid amobarbital data for 262 epileptic patients, without clinical evidence of early damage to the left hemisphere, suggest that language representation in this population is identical with that of the normal population. Damage to the left hemisphere, be it neoplastic or vascular, beginning in middle childhood, results in progressively less transfer of language capacity to the right

hemisphere, as is evident from the decreasing language capacity of the solo right hemisphere after left hemispherectomy at increasing age.¹⁶⁻¹⁸ Thus, age at the time of gross brain injury affects the extent of reorganization of language and should be a crucial variable in defining the subset of commissurotomy patients with right hemisphere language competence.

LOCATION OF INJURY

Rasmussen and Milner¹³ suggest that gross brain damage in the perisylvian language areas is a prerequisite to reorganization of language. Patients with early injury who remain right-handed, and who have not sustained damage in crucial language zones, show relatively infrequent evidence of reorganization of language representation. Also, although the lesion in many of the patients who have presumably become pathologic left-handers is sufficiently close to the perisylvian language region to result in language reorganization, the injury can be discrete enough to affect motor function without affecting language organization. Thus, a sizable percentage of mixed and left-handers have left hemisphere language only (Table 1).

It is important to observe that gross early damage to the left hemisphere, although frequently resulting in right hemisphere language capacity as just discussed, does not necessarily do so. Determination of location of damage is crucial for any prediction about the probability of right hemisphere language. Moreover, even when the location of the lesion is documented to be within the perisylvian language region, reorganization of language within the left hemisphere rather than between hemispheres may result. For example, reorganization can occur within the left hemisphere, at the cortical level,¹⁹ as well as between centers located at different anatomic levels.²⁰ Evidence from stimulation mapping during craniotomy for epilepsy clearly documents a group of patients with left hemisphere lesions and left hemisphere language that is atypically localized within the hemisphere, so that language processing extends well into the parietal lobe.¹⁹ Thus, reorganization does not necessarily mean transfer to the opposite hemisphere.

Lateralization of language at the subcortical or thalamic level, is also now well documented.²¹ The pattern of event-related potentials measured during cortical resection of epilepsy is similar to that evoked from the thalamocortical activating circuit.²²⁻²⁴ A complete model of the physiology of language may well require consideration of thalamocortical systems. Again, reorganization may not necessarily mean transfer of language to the right. Subcortical processing may turn out to be especially relevant to the expression of language representation in the commissurotomy population. (See the articles by H. Damasio et al and Graff-Radford and Damasio in this issue of *Seminars*.)

TYPE OF INJURY

The next relevant issue is the type of damage required to induce reorganization. In many epileptics, brain damage is not apparent by standard diagnostic techniques, such as computed tomography (CT) scanning. For example, of 98 children with chronic seizure disorders, structural abnormalities were documented in only one-third,²⁵ and of 187 adults, abnormalities were present in about one-half.²⁶ Most of those abnormalities were non-specific, such as diffuse cerebral atrophy. With temporal lobe epilepsy requiring surgery, pathologic changes were absent in 21 to 48% of all patients,^{27,28} suggesting that in some instances no abnormality can be found with current methods.

Focal structural abnormality is most frequent in patients with partial epilepsy. Gastaut and Gastaut²⁹ suggest that the functional, constitutional epilepsies be distinguished from the organic epilepsies on the basis of the presence or absence of pathologic processes in the brain. In a subset of those patients with normal scans, microscopic damage is presumably present, for example, mesial temporal sclerosis, cortical dysplasia, hamartomas. With newer techniques, such as nuclear magnetic resonance, some of these "microscopic lesions" may prove to be discernable. Data from position emission tomography (PET) suggest that epileptic foci, whether or not they are associated with apparent brain damage, are metabolically hypoactive.⁴⁰ Thus, the impetus for reorganization may be present even when brain damage is not grossly apparent.

Several other caveats are in order. Localization of abnormal brain tissue based on seizure type and electroencephalographic (EEG) findings is notoriously unreliable in children. The occurrence of partial seizures in children does not reliably predict the location of EEG or brain abnormalities. Also, focal and multifocal seizures may occur in the child with generalized EEG abnormalities. Additionally, the EEG abnormalities may be a manifestation of primary or secondary generalization. Gibbs et al.³¹ describe an EEG pattern, frequent during development, in which the electrical focus moves anteriorly with time, starting occipitally in the young child and ending, by adolescence, in the anterior temporal region. It is not known where and what the underlying pathologic condition is. Additionally, benign focal epilepsy of childhood³² may well be a form of functional generalized epilepsy, the EEG characteristics reflecting the immaturity of the central nervous system and genetic factors rather than the location of the pathologic process.²⁹ In sum, the parameters of partial seizures in the child remain to be further elucidated. In the absence of structural brain damage, the significance of partial seizures in childhood for language reorganization is still a matter of conjecture.

A second consideration is that, although both electrophysiologic studies^{33,34} and PET³⁵ suggest a focal origin for some types of epilepsy classified as

generalized, such as Lennox-Gastaut or slow-spike and slow-wave epilepsy, the incentive for reorganization with generalized epileptiform activity without apparent focal damage is not known.

It is also important to realize that surgery for seizures is often a last-resort approach to epilepsy. Thus, most patients coming to commissurotomy surgery have had seizures for many years. The effect, for example, of the development of a mirror focus on already reorganized language functioning is not known. Kindling may have different effects on the right and left hemispheres. Stokes and McIntyre,³⁶ for example, report lateralized, asymmetric, state-dependent learning in kindled animals. Those kindled in the left hippocampus showed evidence of learning only when trained and tested in the normal state. On the other hand, animals kindled in the right hippocampus showed evidence of some state-dependent learning with some transfer occurring from the convulsed to the normal state. The implication of such hemispheric asymmetry for the reorganization of function in the kindled brain remains to be determined.

Clearly, the differences in the reorganizational impetus for language in patients with epilepsy secondary to gross brain disease, as opposed to those with epilepsy and no apparent disease, are still to be worked out.

HANDEDNESS AND LANGUAGE ORGANIZATION

Handedness is known to be an important variable in brain organization for language. Clinical stroke data document that at least 97% of right-handers are left hemisphere dominant for language.¹⁴ There is considerably more variability among left-handers, although the details of language organization vary, depending on the assessment technique (Table 2). The anatomic markers of language organization, such as the planum temporale asymmetry, are more variable in left-handers.³⁷ Ratcliff et al.³⁸ documenting language representation by intracarotid amobarbital testing in a population of epileptics being considered for surgery, demonstrate an appropriate covariance among handedness, language representation, and morphologic asymmetries. The contributions of family handedness and strength of handedness as markers of language organization are receiving increasing attention.³⁹⁻⁴¹ These may affect language organization in both right- and left-handers.

The marked change in ratio of right- to left-handers from 10 to 1 in the normal population to 1 to 2 in patients with early left hemisphere damage highlights the importance of the concept of pathologic left-handedness when considering the epileptic population.¹³ But it is often forgotten that pathologic right-handedness also exists.⁴² This must be kept in mind when the commissurotomy patients are considered in the context of right hemisphere damage. Patients with pathologic right

Table 2. Speech Representation in Sinistrals

Speech Representation (%)			Technique	Study
Left	Bilateral	Right		
70	15	15	Amobarbital	Rasmussen and Milner ¹³
66		34	Electro-convulsive therapy	Warrington and Pratt ⁴³
40	40	20	Statistical correlation of lesion data	Satz ⁴⁴ Carter et al ⁴⁵

handedness obviously represent the most uncommon subset of persons with right hemisphere language.

ROLE OF SEX DIFFERENCES

Differences in language representation in males versus females have been suggested, although the data are controversial.⁴⁶ Greater bilateralization of language processes may be present in the female. Conceivably, the more rapid maturation of the female central nervous system^{47,48} is conducive to bilateralization.⁴⁹ Notable in this context is the greater frequency of febrile seizures (a reflection of central nervous system immaturity) among young males compared with females of the same age, and the preponderance of left hemisphere origin of febrile seizures in the first 2 years.⁵⁰ In view of the association between prolonged febrile seizures and mesial temporal sclerosis,²⁷ females with febrile convulsions at an early age should show a particularly high incidence of right hemisphere language, secondary to reorganization after left hemisphere injury.

The clinical lesion data supporting greater bilateralization of language in the adult female document deficits in verbal abilities measured by IQ tests and at intracarotid amobarbital testing, with lesions of either hemisphere,^{51,52} but epidemiologic studies of the incidence of aphasia with stroke have not corroborated the other clinical data, suggesting greater bilateralization of language in females.¹⁴

INDIVIDUAL VARIABILITY AND LANGUAGE ORGANIZATION

Individual variability cannot be discounted as a key factor in explaining the incidence of right hemisphere language in any population, including patients who have undergone commissurotomy. The planum temporale asymmetry to which we have previously alluded is present in only 65% of the normal right-handed population, 97% of whom are left hemisphere dominant for language. Thus, the planum temporale is not a sufficient marker of left hemisphere specialization, although it may reflect some subordinate aspect of lateralization.³⁷ Cortical stimulation studies demonstrate a wide range in localization of elicited language processes within the left hemisphere for epileptic individuals who oth-

erwise show relatively typical brain organization.^{53,54}

INTELLIGENCE AND HEMISPHERIC SPECIALIZATION

The effect of intelligence on hemispheric specialization is only beginning to be studied. Many investigators suggest that normal intelligence is a prerequisite to specialization of function.⁵⁵ Thus, the fact that a number of commissurotomy patients have only borderline intellectual abilities must be considered in the equation for anticipated right hemisphere language. Moreover, long-term studies of commissurotomy patients suggest that total callosotomy may selectively enhance the effect of preexisting pathologic processes in the right hemisphere,^{56,57} which may range from focal EEG abnormalities to grossly apparent hemispheric atrophy. The four patients in the study of Campbell et al.⁵⁷ with documented structural damage in the right hemisphere, showed both immediate and chronic depression of right hemisphere function on neuropsychologic tests. One might conclude that, even if minimal, damage to the right hemisphere would prevent the expression or acquisition of language in that hemisphere after commissurotomy. However, N.G., one of the five patients with demonstrable right hemisphere language skills, is one of the four patients with structural damage.

COMPENSATORY BEHAVIORAL STRATEGIES AND RIGHT HEMISPHERE LANGUAGE

Once the callosum is sectioned, the right hemisphere is theoretically freed from left hemisphere dominance. The most common ramification of this event on language is mutism. Lasting from days to months, mutism is a complication of both single-stage total commissurotomy and two-stage commissurotomy sparing the anterior commissure. The mutism is not a form of aphasia, both because the recovery pattern differs (no anomia, no paraphasias, and intact ability to write) and because there is no hemispheric damage to explain it. Bogen^{58,59} argues against the explanation that it is a mild form of akinetic mutism, since patients with comparable surgical manipulation in the area of the anterior third ventricle, but with sparing of the splenium, have not become mute. He favors a behavioral ex-

planation wherein the previously left-dominated right hemisphere is suddenly free to compete.⁵⁹ The competition results in mutism. On the other hand, L.B. awoke from surgery complaining vigorously and verbally of a headache.⁶⁰ The relatively short duration of mutism with two-stage procedures, be they anteroposterior or posteroanterior, suggests that a *modus vivendi* between the two partially separated hemispheres can be preemptively achieved. Bogen⁵⁸ reports at least one patient with right hemisphere atrophy from childhood who manifested no mutism. This suggests that when one hemisphere has carried the entire burden of cognition and consciousness, disconnecting the non-functioning hemisphere does not lead to mutism.

RIGHT HEMISPHERE LANGUAGE FOLLOWING COMMISSUROTOMY

The five patients who have shown evidence of right hemisphere language have different linguistic profiles. These have been discussed in detail elsewhere.⁵ Here we will examine their neurologic histories for cues that might possibly explain their special status.

SUMMARY OF NEUROLOGIC HISTORIES

PS,² at 20 months of age, a previously normal right-handed boy, developed a high fever and had many right-sided motor seizures, which became generalized. They were controlled by phenobarbital after 8 hours. He then exhibited hyperactivity and disruptive behavior, but was without seizures until age 4 years when he experienced "absences" about twice a month. At age 6, he had a normal EEG. At age 10, he began to have right-sided partial seizures characterized by adverse movement of the head, posturing of the right arm, and walking around in circles. These lasted for 2 minutes, and, following them, he slept for several hours. The EEG showed slow waves over the left hemisphere and spikes with spike-wave complexes in the left temporal leads, occasionally propagated to the right side. The only physical abnormalities were impaired ability to recognize objects placed in his right hand, and right-sided hearing loss. Diazepam and phenytoin were begun. Phenobarbital was later added, but the partial seizures increased in frequency. Phenytoin was increased to toxic levels.

By January 1974, when he was 14 years old, seizures were occurring 10 times monthly, and manipulation of various anticonvulsants, such as phenytoin, phenobarbital, diazepam, and primidone, was to no avail. The EEG showed spike-wave discharges in the left frontotemporal and posterior temporal regions, with dysrhythmia over the entire left hemisphere. Generalized akinetic seizures were added to his usual partial motor and complex attacks. The latter seizures were characterized by rocking back and forth, shouting, and bizarre automatism. Between seizures, his hyperactive, dis-

ruptive, and often violent behavior continued. Then partial motor seizures began to occur on his left side as well.

He was admitted for surgery in December of 1975 at age 15 years. Physical examination, skull x-ray, and brain scan were normal, as were pneumoencephalography and pancerebral angiography. EEGs showed diffuse seizure discharges over the left hemisphere, most marked in the temporal area, with slight secondary synchrony in the right temporal area. There was a discrepancy between verbal IQ (83) and performance IQ (99); the total IQ was 89, in the dull normal range. The neuropsychologic tests suggested "left hemisphere damage."

Complete extraventricular division of the corpus callosum was accomplished without incident. The patient recovered consciousness quickly and showed all the characteristics of an acute disconnection syndrome: mutism, immobility of the left side, left homonymous visual neglect, bilateral Babinski responses, and alternating periods of stupor and alertness. Mild hyponatremia and hypokalemia were corrected. Although the ventricular systems were not entered at all, the quadrigeminal cistern and the corpus callosum "cistern" were breeched as usual. Low-grade fever was associated with mild aseptic meningitis. Ampicillin had been started 2 days before surgery and was continued for 5 days after the operation. Dexamethasone was continued in diminishing doses for 10 days. Within a few days, he was afebrile, and the cerebrospinal fluid (CSF), although xanthochromic and under high pressure, contained no white cells.

By the second week after surgery, he was able to walk, eat, and dress himself. The acute disconnection syndrome subsided, but the most outstanding characteristics of the postoperative course was a regression to infantile behavior. He was querulous and demanded constant attention. He shouted constantly until someone sat with him, when he would talk rationally. In addition to phenytoin, he was placed on haloperidol. He complained of headaches. Lumbar punctures revealed normal fluid under high pressure. By the third postoperative week, CSF pressure was normal, but an infusion test was distinctly abnormal. CT scan showed small ventricles in the normal position.

The final lumbar puncture was followed by staphylococcal meningitis, which was quickly controlled. The patient was discharged on March 24, when his behavior was normal and he had no neurologic deficit. He was discharged on phenytoin, having experienced no seizures since operation. An EEG showed the same discharges in the left frontotemporal area. Activity in the right hemisphere was normal. In August 1976 he had a single generalized tonic-clonic seizure. His phenytoin level was only 3.2 $\mu\text{g/ml}$. Postoperative IQ was essentially unchanged. Within 1 month of his operation, he clearly had right hemisphere language, with the capacity to carry out commands that were strictly

lateralized to the right brain. Approximately 2 years after surgery, right hemisphere speech appeared.

VP,¹⁰ a 32-year-old right-handed woman, had a mixed seizure disorder that could not be managed with antiepileptic drugs. She was the product of a full-term pregnancy and uncomplicated delivery. She had no unusual illnesses in childhood and reached developmental landmarks normally. There was no family history of neurologic disorders. At age 9 years she experienced recurrent seizures following febrile illnesses that included measles and scarlet fever. The disorder was characterized by "minor spells" and rare generalized convulsions. Anticonvulsant medicine controlled the seizure disorder and she was graduated from high school. The EEG records from this period revealed spike-and-slow-wave activity diffusely. By 1976 she was experiencing episodes of blank staring lasting for seconds, occurring several times a day. EEG records during this period revealed bilateral 4 cps spike-and-slow-wave activity, and sharp activity with left temporal predominance. In 1979 she was experiencing generalized major motor, absence, and myoclonic seizures while on multiple anticonvulsants. She was referred for further management to the Medical College of Ohio. Evaluation included a normal skull film, CT scan, and angiogram. She underwent partial anterior callosal section in early April 1979, and the resection of her callosum was completed in a second operation 7 weeks later. Postoperative neurologic examination revealed no focal findings. Wechsler IQ scores were in the normal range. She gave evidence of right hemisphere language comprehension immediately after surgery. Within 1 year, right hemisphere speech appeared.

JW,¹⁰ a 30-year-old right-handed man, had intractable epilepsy since the age of 19 years. He was the product of a full-term pregnancy and uncomplicated delivery. Developmental milestones were appropriately achieved. There was no family history of neurologic disorder. At the age of 13 years, after concussive head trauma without skull fracture, he began to experience brief, infrequent absence spells. They were not treated. He was graduated from high school at age 18 years. One year after graduation, he experienced a major motor seizure. A complete neurologic evaluation, including lumbar puncture, brain scan, skull films, and blood chemistries, was normal. An EEG revealed irregular polyspike and high voltage repetitive 3 cps spike and wave bursts during sleep. These abnormalities had a right anterior temporal prominence. Over the next 7 years, there were many hospitalizations during which attempts were made to manage his epilepsy. In spite of adequate serum levels of antiepileptic medication, he had frequent generalized major motor seizures. The EEGs during this period revealed irregular polyspike and spike and wave activity bilaterally. A CT scan was normal. During 1977 to 1979, he continued to have

frequent generalized major motor seizures and many absence attacks each day while on adequate therapeutic doses of several different medications. He was referred to the Dartmouth-Hitchcock Medical Center. He underwent two-stage micro-neurosurgical section of his corpus callosum over the summer and fall of 1979. The posterior callosum was sectioned first. At neurologic examination 8 months after his second operation, he was oriented and alert and conversed easily about present and past events. His neurologic examination and his IQ were normal. He had the capacity to understand language in the right hemisphere immediately following his first operation as well as following his second. He has never developed right hemisphere speech.

NG,^{57,60,61} who was born after 6 months gestation, in 1933, required hospitalization for several weeks after birth. There was a family history of epilepsy. Her development was nonetheless normal until she began to have convulsions at the age of 18 years, during pregnancy. Her blood pressure was then 170/110 mmHg. She had a miscarriage, and her blood pressure returned to normal, but she continued to have seizures.

She was first admitted to the White Memorial Hospital in 1952, with a history of eight convulsions. Her husband reported that he was awakened at night by a strange sound and found his wife "stiffened out" for 2 or 3 minutes. Physical examination was normal. The patient was right-handed with no family history of sinistrality. Chest x-rays showed calcifications near the right hilum. The EEG showed left temporal slowing. The skull x-ray revealed a calcification, 1 cm in diameter, beneath the right central cortex. A right carotid angiogram was normal. Wechsler full-scale IQ was 76, with a verbal IQ of 79 and a performance IQ of 74.

Seizures of the tonic-clonic, absence, and partial complex types, with a left-sided sensory aura, increased in frequency. She had a complete cerebral commissurotomy in 1963 (aged 30). Postoperatively, there were bilateral neurologic signs transiently and the expected disconnection signs. Seizures (generalized, right focal, and absence) occurred in the hospital after operation when anticonvulsant medication was withdrawn, but none occurred subsequently for 8 years except for occasional episodes of transient left-sided numbness. During the ninth year, while medication was being reduced, the patient developed status epilepticus. In the hospital, the EEG, which had been essentially normal for 7 years, showed a left temporal focus. Anticonvulsant medication was reinstituted. The EEG again returned to normal (in less than 1 year), and the patient had no seizures of any kind for 2 years. Examination 9 years after operation revealed no focal neurologic abnormalities. Wechsler full-scale IQ at 4 and 13 years after operation was 71 and 74, respectively. This patient demonstrated right hemisphere language comprehension within her first postoperative year. She was the first

Table 3. Summary of Neurologic Histories of Five Commissurotomy Patients Without Right Hemisphere Language

Case	Age at Onset of Seizures (yr)	Seizure Type and Frequency	Preoperative EEG	Neurologic Examination
LR	11. Undetermined cause	Generalized: tonic-clonic, akinetic, absence Partial: complex	Bilateral synchronous and asynchronous spike-wave discharges	Normal left hemisphere dysfunction on neurologic exam
SW	6. Febrile seizures at 6 months	Generalized: tonic-clonic, akinetic, absence	Bilateral, symmetrical, and asymmetrical spike-wave discharges, frontal predominance	Normal
JK	4	Complex partial, partial motor, right, 5–10 day	Right frontotemporal spike and slow waves with diffuse bilateral spread	Normal
SF*	8	Tonic seizures more than 1 month later. Later akinetic seizure. Upper and lower extremity posturing (10–20 day) Generalized: several per year	Bifrontal (R>L) sharp wave, spike and wave polyspike and wave depth EEG—right temporal and frontal seizure onset plus sign. Left temporal discharges	Normal
EB	11	Partial generalized Lobectomy: Full	Paroxymal. Right occipital focus with 2° generalization. No change after occipital. Full scale IQ: 83; verbal IQ: 86; performance IQ: 81	Normal except left inferior quadrantanopia due to previous surgery

Table modified from Wilson et al, 1977; 1982

*Novelly, R. et al, in press.

patient in the California series to give evidence of right hemisphere language.

LB^{60,57} was born by cesarean section, in 1952, after a normal pregnancy. Birth weight was 5 pounds. He was cyanotic and remained in an Isolette for 8 days. Development was considered normal. He first had a convulsion at age 3½ years. Nonfocal seizures increased in frequency. No neurologic abnormality was found on preoperative amination. EEGs showed mild, diffuse abnormalities, most evident bitemporally. Pneumoencephalogram (in 1956) and bilateral carotid angiogram were normal. Preoperative Wechsler full scale IQ was 113.

The patient had a complete cerebral commissurotomy in April 1965. No search was made for the massa intermedia. His postoperative course was smooth. He spoke well the next day. He continued to do well, attending public school and doing passable work, except in the "new math." As an adult, he received on-the-job training as a shop assistant. Postoperative Wechsler full-scale IQs at 2 and 10 years were 109 and 110 with a verbal IQ of 113 and 109 and a performance IQ of 102 and 110, respectively.

Since the operation, he has had only seven major seizures and none for 4 years (compared with an average of one a week in the year preceding the operation). In the third year there were nine left-sided Jacksonian seizures. Neurologic examination 8 years after operation was nonfocal. He also gave

evidence of language comprehension in the right hemisphere during his first year after operation.

DISCUSSION OF THE CASES

As is apparent, the histories of these patients vary greatly. With regard to timing of injury, two patients had evidence of early abnormalities or seizures (PS, LB), one did not (JW), and in two this was uncertain (VP, NG). The location of possible structural lesions that might predispose to bilateral organization of language was apparent only in PS and LB. However, NG had a structural lesion in the right hemisphere, and JW and VP had no specific evidence of a localized structural lesion. Sex could be construed as a predisposing factor in two cases and handedness in none.

There are, of course, a host of patients with cerebral commissurotomy who have not given evidence of right hemisphere language at this writing. This would include approximately 13 cases in the California series⁵⁷ and over 35 cases in the Wilson series. We have also studied a few patients from other centers, including cases from the Yale and the Ohio series. Some of the patients had an unfavorable hemiplegia, which disallows them from full consideration of the presence or absence of bilateral language. Some transfer information visually, presumably through inadvertently spared splenial fibers or the remaining anterior commissure, but

<i>Other Pertinent Data</i>	<i>Age at Operation (yr)</i>	<i>Type of Operation</i>	<i>Complications</i>
IC: 84	25	Central commissurotomy 1. Posterior: Sept 14, 1979 2. Anterior: Dec. 4, 1979	1. None 2. None
IC: 83	20	Central commissurotomy 1. Posterior: Nov. 21, 1980 2. Anterior: May 1, 1981	1. Right-left dissociation 2. None
IC: 81	16	Complete commissurotomy: Feb. 6, 1973	None
Right lateral ventricle horn mildly dilated	20	Complete single-stage callosotomy	None
Normal, except for hemianopsia. Postoccipital corticectomy	23	Posterior callosotomy	None

most of these are still assessable because of the marked disconnection of tactile information. In such patients it is possible to examine tactile-auditory matching. Other patients are lost to follow-up. At present, a new effort is under way to identify all appropriate patients from several centers and to examine each case methodically.

Examination of the neurologic histories of five cases that have been tested for right hemisphere language in the past but have not exhibited such function yields no insights into any possible pattern in their early neurologic history (Table 3). Variability in age of onset and type of lesion was not illuminating. One case, SF, underwent a PET scan several years after split-brain surgery in an effort to determine the existence of right hemisphere damage that might explain his failure to demonstrate right hemisphere language. This patient, who did exhibit some signs of neglect when asked to draw pictures to command, could nonetheless carry out a picture-to-picture matching task within the right hemisphere. At the same time, picture-word matching was not possible, nor was auditory-object matching. The PET scan revealed no gross hemispheric differences and there was no evidence of a gross pathologic process.

SUMMARY AND CONCLUSIONS

The normal brain organization that supports both the comprehension and expression of human language is of great importance to the neurologist. Although the vast majority of neurologic reports would suggest the left hemisphere is predominantly responsible for language management, a number of reports suggest that some kind of right hemisphere involvement can occur. Adding sup-

port to this minority view over the years has been the series of studies on split-brain patients. What has not been generally realized is that these studies have been carried out on a select subset of the split-brain population, and therefore may have inadvertently created the impression that right hemisphere language occurs with a greater frequency following bisection of the brain. In addition, it has not been clear whether right hemisphere language, when present, is the product of normal or abnormal neurologic development.

In the foregoing, we have reviewed the factors involved in producing an increased probability of bilateral language. Our aim has been to identify factors that might explain the occurrence of documented right hemisphere language in split-brain patients. The data suggest that no coherent picture can yet be identified that predicts the presence or absence of right hemisphere language following cerebral commissurotomy. In some cases there are indications of predisposing damage and sometimes not. The same seems also to be true for patients who do not give evidence of right hemisphere language. It will remain for future studies, now in progress, on additional cases to identify such factors.

This study was aided by United States Public Health Service Grant 5 PO1 NS17778 to Dr. Gazzaniga and a Rockefeller Brothers Clinical Scholarship to Dr. Nass.

REFERENCES

1. Bogen JE, Vogel PJ: Cerebral commissurotomy in man. Preliminary case report. *Bull Los Angeles Neurol Soc* 27:169-172, 1962

2. Wilson DH, Reeves A, Gazzaniga M, Culver C: Cerebral commissurotomy for control of intractable seizures. *Neurology (Minneapolis)* 27:708-715, 1977
3. Wilson DH, Reeves A, Gazzaniga MS: "Central" commissurotomy for intractable generalized epilepsy: Series two. *Neurology (NY)* 32:687-697, 1982
4. Reeves A: *The Corpus Callosum and Epilepsy*. New York: Plenum Press, in press
5. Gazzaniga MS: Right hemisphere language following brain bisection: A 20-year perspective. *Am Psychol* 38:525-537, 1983
6. Gazzaniga MS, Sperry RW: Language after section of the cerebral commissures. *Brain* 90:131-148, 1967
7. Zaidel E: Auditory vocabulary of the right hemisphere following brain bisection or hemidecortication. *Cortex* 12:191-211, 1976
8. Gazzaniga MS, Wilson DH, LeDoux JE: Language, praxis and the right hemisphere: Clues to some mechanisms of consciousness. *Neurology (Minneapolis)* 27:1144-1147, 1977
9. Gazzaniga MS, LeDoux JE, Smylie CS, Volpe BT: Plasticity in speech organization following commissurotomy. *Brain* 102:805-815, 1979
10. Sidtis JJ, Volpe BT, Wilson DH, Ravport M, Gazzaniga MS: Variability in right hemisphere language function after callosal section: Evidence for a continuum of generative capacity. *J Neurosci* 1:323-331, 1981
11. Gazzaniga MS, Smylie CS, Baynes K, Hirst W, McCleary C: Profiles of right hemisphere language and speech following brain bisection. *Brain Lang*, 1984. In press
12. Moscovitch M: On the representation of language in the right hemisphere of right-handed people. *Brain Lang* 3:47-71, 1976
13. Rasmussen T, Milner B: The role of early left brain damage in determining the lateralization of cerebral speech functions. In Diamond S, Blizard D, Eds: *Evolution and Lateralization of the Brain*. New York: New York Academy of Sciences, 1977
14. Kertesz A: *Aphasia and associated disorders: Taxonomy, localization, and recovery*. New York: Grune & Stratton, 1979
15. Kennard MA: Reorganization of motor function in the cerebral cortex of monkeys deprived of motor and premotor areas in infancy. *J Neurophysiol* 1:477-496, 1938
16. Bassler LS: Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. *Brain* 85:427-460, 1962
17. Gott PS: Language after dominant hemispherectomy. *J Neurol Neurosurg Psychiatr* 36:1082-1088, 1973
18. St. James-Roberts I: A reinterpretation of hemispherectomy data without functional plasticity of the brain. I. Intellectual function. *Brain Lang* 13:31-53, 1981
19. Ojemann GA, Whitaker HA: Language localization and variability. *Brain Lang* 6:239-260, 1978
20. Goldman PS: An alternative to developmental plasticity: Heterology of CNS structures in infants and adults. In Stein DG, Rosen J, Butters N, Eds: *CNS Plasticity and Recovery of Function*. New York: Academic Press, 1974 pp 147-174
21. Brown J: Thalamic mechanisms in language. In Gazzaniga MS, Ed: *Handbook of Neurobiology*. New York: Plenum Press, 1979, pp 215-294
22. Jasper HH: Unspecific thalamocortical relations. In Fields J, Ed: *Handbook of Physiology*, Section 1, *Neurophysiology*, vol. 2. Baltimore: Waverly Press, 1960, pp 1307-1321
23. Yingling CD, Skinner JE: Selective regulation of thalamic sensory nuclei by nucleus reticularis thalami. *Electroenceph Clin Neurophysiol* 41:476-482, 1976
24. Ojemann GA, Fried I: Event related potential correlates of human language cortex measured during cortical resection for epilepsy. In Akimoto H, Kazamatsuri H, Scino M, Ward AA Jr, Eds: *Advances in Epileptology*. The XIIIth Epilepsy International Symposium. New York: Raven Press, 1982, pp 385-388
25. Bachman DS, Hodges FJ III, Freeman JM: Computerized axial tomography in chronic disorders of childhood. *Pediatrics* 58:828-832, 1976
26. Bauer G, Mayr U, Pallua A: Computerized axial tomography in chronic partial epilepsies. *Epilepsia* 21:227-233, 1980
27. Falconer MA, Taylor DC: Surgical treatment of drug resistant epilepsy due to mesial temporal sclerosis. *Arch Neurol* 29:353-361, 1968
28. Mathieson G: Pathologic aspects of epilepsy with special reference to the surgical pathology of focal cerebral seizures. In Purpura DP, Penry JK, Waler RD, Eds: *Advances in Neurology*, vol. 8. New York: Raven Press, 1975 pp 107-138
29. Gastaut H, Gastaut J-L: Computerized axial tomography in epilepsy. In Penry JK, Ed: *Epilepsy: The Eighth International Symposium*. New York: Raven Press, 1977, pp 5-15
30. Kuhl DE, Engel J Jr, Phelps ME, et al: Epileptic patterns of local cerebral metabolism and perfusion in man determined by emission computed tomography of ^{18}F FDG and $^{15}\text{NH}_3$. *Ann Neurol* 8:348-360, 1980
31. Gibbs EL, Gillen HW, Gibbs FA: Disappearance and migration of epileptic foci in children. *Am J Dis Child* 88:596-603, 1954
32. Lombroso CT: Sylvian seizures and midtemporal spike foci in children. *Arch Neurol* 17:52-59, 1967
33. Stewart LF, Dreifuss FE: "Centrencephalic" seizure discharges in focal hemispherical lesions. *Arch Neurol* 17:60-68, 1967
34. Mutani R, Durelli L: Mechanisms of interaction of asymmetrical bilateral epileptogenic foci in neocortex. *Epilepsia* 21:549-556, 1980
35. Gur RC, Sussman NM, Alavi A, Gur RE, Rosen AD, O'Connor M, Goldberg HI, Greenberg JH, Reivich M: Positron emission tomography in two cases of childhood epileptic encephalopathy (Lennox-Gastaut syndrome). *Neurology (NY)* 32:1191-1194, 1982
36. Stokes KA, McIntyre DC: Lateralized asymmetrical state-dependent learning produced by kindled convulsions from the rat hippocampus. *Physiol Behav* 26:163-169, 1981
37. Witelson SF: Neuroanatomical asymmetry in left-handers: A review and implications for functional asymmetry. In Herron J, Ed: *Neuropsychology of Left-Handedness*. New York: Academic Press, 1980
38. Ratcliff G, Dila C, Taylor L, Milner B: Morphological asymmetry of the hemispheres and cerebral dominance for speech: A possible relationship. *Brain Lang* 11:87-98, 1980
39. Johnstone J, Galin D, Herron J: Choice of handedness measures in studies of hemispheric specialization. *Int J Neurosci* 9:71-80, 1979
40. Hecaen H, de Agostini M, Monzon-Montes A: Cerebral organization in left-handers. *Brain Lang* 12:261-284, 1981
41. Andrews RJ: Aspects of language lateralization correlated with familial handedness. *Neuropsychologia* 15:769-778, 1977
42. Bishop DVM: Handedness, clumsiness and cognitive ability. *Dev Med Child Neurol* 22:569-579, 1980
43. Warrington EK, Pratt RTC: Language laterality in left-handers assessed by unilateral ECT. *Neuropsychologia* 11:223-238, 1973
44. Satz P: Incidence of aphasia in left-handers: A test of some hypothetical models of cerebral speech organization. In Herron J, Ed: *Neuropsychology of Left-Handedness*. New York: Academic Press, 1980
45. Carter RL, Hohenegger M, Satz P: Handedness and aphasia: An inferential method for determining the mode of cerebral speech specialization. *Neuropsychologia* 18:569-574, 1980
46. McGlone J: Sex differences in human brain asymmetries. *Behav Brain Sci* 3:215-263, 1980
47. Conel JR: *The Postnatal Development of the Human Cerebral Cortex*, 6 vols. Cambridge, MA: Harvard University Press, 1959-1968
48. Denckla, MB: Minimal brain dysfunction. In Chall JS, Mir-

- sky AF, Eds: Education and the Brain. Chicago: University of Chicago Press, 1977
49. Annett M: Laterality of childhood hemiplegia and the growth of speech and intelligence. *Cortex* 9:4-33, 1973
50. Taylor DC, Ounsted C: Biological mechanisms influencing the outcome of seizures in response to fever. *Epilepsia* 12:33-45, 1971
51. McGlone J: Sex differences in the cerebral organization of verbal functions in patients with unilateral brain lesions. *Brain* 100:775-793, 1977
52. McGlone J, Fox A: Evidence from sodium Amytal studies of greater asymmetry of verbal representation in men compared to women. In Akimoto H, Kazamatsuri H, Seino M, Ward AA Jr, Eds: *Advances in Epileptology. The XIIIth Epilepsy International Symposium*. New York: Raven Press, 1982, pp 389-391
53. Ojemann GA: Individual variability in cortical localization of language. *J Neurosurg* 50:164-169, 1979
54. Ojemann GA: Brain organization for language from the perspective of electrical stimulation mapping. *Behav Brain sci* 6:189-206, 1983
55. Berman A: The problem of assessing cerebral dominance and its relationship to intelligence. *Cortex* 7:372-386, 1971
56. Novelly RA, Lofrak M, Williamson PD, Spencer D: Total callosotomy selectively enhances effects of pre-existing minor hemisphere lesions: A test of the hypothesis. In Reeves A, Ed: *The Corpus Callosum and Epilepsy*. New York: Plenum Press. In press
57. Campbell AL Jr, Bogen JE, Smith A: Disorganization and reorganization of cognitive and sensorimotor functions in cerebral commissurotomy: Compensatory roles of the forebrain commissures and cerebral hemispheres in man. *Brain* 194:493-511, 1981
58. Bogen JE: Language functions in the short term following cerebral commissurotomy. In Avakian-Whitaker H, Whitaker HA, Eds: *Current Trends in Neurolinguistics*, vol. 2. New York: Academic Press, 1976
59. Bogen JE: The callosal syndrome. In Heilman K, Valenstein E, Eds: *Clinical Neuropsychology*. New York: Oxford Press, 1979, pp 308-359
60. Bogen JE, Vogel PJ: Neurologic status in the long term following complete cerebral commissurotomy. In Michael F, Schott B, Eds: *Les syndromes de disconnexion calleuse chez l'homme*. Lyon: Hôpital Neurologique Lyon, 1975, pp 227-253
61. Bogen JE, Fisher ED, Vogel PJ: Cerebral commissurotomy: A second case report. *JAMA* 194:1328-1329, 1965